A 46-year-old man comes to the emergency department due to intermittent severe right flank pain over the past few days. He has had decreased urination over the last week but has also noted occasional episodes of high urine output along with a feeling of generalized weakness. The patient has a history of chronic back pain for which he takes oxycodone daily, and he underwent a left total nephrectomy following a motor vehicle accident 25 years ago. He was recently started on low-dose lisinopril for a new diagnosis of hypertension. There is no family history of renal disease. On physical examination, blood pressure is 145/86 mm Hg and heart rate is 86/min. Laboratory results are as follows:

Serum chemistry	
Potassium	3.4 mEq/L
Creatinine	1.7 mg/dL
Urinalysis	
Protein	trace
White blood cells	4/hpf
Red blood cells	2/hpf
Casts	none

Which of the following is the most likely cause of this patient's symptoms?

A. Adrenal tumor
B. Glomerulonephritis
C. Inherited renal disease

Potassium	3.4 mEq/L
Creatinine	1.7 mg/dL

Urinalysis

Protein	trace
White blood cells	4/hpf
Red blood cells	2/hpf
Casts	none

Which of the following is the most likely cause of this patient's symptoms?

- A. Adrenal tumor
- B. Glomerulonephritis
- C. Inherited renal disease
- D. Interstitial nephritis
- E. Renal artery stenosis
- F. Urinary outflow obstruction

Submit

A 46-year-old man comes to the emergency department due to intermittent severe right flank pain over the past few days. He has had decreased urination over the last week but has also noted occasional episodes of high urine output along with a feeling of generalized weakness. The patient has a history of chronic back pain for which he takes oxycodone daily, and he underwent a left total nephrectomy following a motor vehicle accident 25 years ago. He was recently started on low-dose lisinopril for a new diagnosis of hypertension. There is no family history of renal disease. On physical examination, blood pressure is 145/86 mm Hg and heart rate is 86/min. Laboratory results are as follows:

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Urinalysis	
Protein	trace
White blood cells	4/hpf
Red blood cells	2/hpf
Casts	none

Which of the following is the most likely cause of this patient's symptoms?

A. Adrenal tumor [3%]
B. Glomerulonephritis [4%]
C. Inherited renal disease [1%]

Urinalysis

Protein	trace
White blood cells	4/hpf
Red blood cells	2/hpf
Casts	none

Which of the following is the most likely cause of this patient's symptoms?

- A. Adrenal tumor [3%]
- B. Glomerulonephritis [4%]
- C. Inherited renal disease [1%]
- D. Interstitial nephritis [20%]
- E. Renal artery stenosis [20%]
- ✔ F. Urinary outflow obstruction [48%]

Omitted
Correct answer
F

48%
Answered correctly

10 Seconds
Time Spent

10/31/2018
Last Updated

Explanation

This patient with a history of total nephrectomy now most likely has **unilateral obstructive uropathy** due to renal calculi. Symptoms that support this diagnosis include flank pain (renal capsular distension) and poor urine output (mechanical obstruction to urine outflow). **Intermittent** episodes of high-volume urination can occur when the obstruction is **overcome** by a large volume of retained urine (**post-obstructive diuresis**). Excessive diuresis may lead to **potassium wasting** and dehydration, both of which can cause **weakness**.

Post-obstructive diuresis may be seen in patients with bilateral functional kidneys as the affected kidney will produce a large volume of urine when the obstruction is relieved. However, patients with a **single kidney** are more likely to develop acute renal failure than are those with both kidneys intact.

(Choice A) Most adrenal tumors are benign and asymptomatic. Symptoms of hormone-producing adrenal tumors depend on the hormone being secreted. Nonfunctioning adrenal carcinomas may present with flank pain due to tumor growth. However, symptoms are less severe, and no change in urinary output would be expected.

(Choice B) Glomerulonephritis presents with hematuria, red blood cell casts, acute renal failure, hypertension, and edema.

(Choice C) The leading cause of inherited renal disease is autosomal dominant polycystic kidney disease. Patients typically present with persistent abdominal or flank pain and hematuria, which may be gross or microscopic (≥3 red blood cells/hpf). There is a positive family history in 75% of cases. Over half of patients have hypertension at presentation.

(Choice D) Interstitial nephritis most frequently occurs as a drug reaction. Patients present with fever, rash, acute kidney injury, and eosinophiluria with white blood cell casts.

(Choice E) Renal artery stenosis can cause hypertension with or without kidney injury. It does not cause flank pain or urinary symptoms.

Educational objective:
Obstructive uropathy presents with flank pain, low-volume voids with or without occasional high-volume voids, and, if bilateral, renal dysfunction.

A 45-year-old woman comes to the emergency department with vomiting and severe right flank pain that radiates to her groin. The patient has been to the emergency department twice in the past due to similar episodes of renal colic. Previous imaging showed radio-opaque stones. The stones passed spontaneously, and she did not seek further medical care. Imaging now shows another ureteral stone. A 24-hour urine collection shows urinary calcium excretion of 350 mg (normal is <250 mg in women). Laboratory results show a serum calcium concentration of 8.9 mg/dL; serum parathyroid hormone is normal. Further investigations fail to reveal a cause of the hypercalciuria. Apart from advising increased fluid intake, which of the following interventions will benefit this patient?

A. Aldosterone antagonist
B. High sodium intake
C. Low-dose furosemide
D. Restriction of dietary calcium
E. Thiazide diuretics

Submit

A 45-year-old woman comes to the emergency department with vomiting and severe right flank pain that radiates to her groin. The patient has been to the emergency department twice in the past due to similar episodes of renal colic. Previous imaging showed radio-opaque stones. The stones passed spontaneously, and she did not seek further medical care. Imaging now shows another ureteral stone. A 24-hour urine collection shows urinary calcium excretion of 350 mg (normal is <250 mg in women). Laboratory results show a serum calcium concentration of 8.9 mg/dL; serum parathyroid hormone is normal. Further investigations fail to reveal a cause of the hypercalciuria. Apart from advising increased fluid intake, which of the following interventions will benefit this patient?

- A. Aldosterone antagonist [2%]
- B. High sodium intake [1%]
- C. Low-dose furosemide [12%]
- D. Restriction of dietary calcium [7%]
- ✔ E. Thiazide diuretics [76%]

Omitted
Correct answer
E

76%
Answered correctly

3 Seconds
Time Spent

12/03/2018
Last Updated

Explanation

Prevention of recurrent nephrolithiasis

Explanation

Prevention of recurrent nephrolithiasis	
Dietary measures	• Increase fluids (produce >2L urine/day) • Reduce sodium (<100 mEq/day) • Reduce protein • Normal calcium intake (1200 mg/day) • Increase citrate (fruits & vegetables) • Reduced-oxalate diet for oxalate stones (dark roughage, vitamin C)
Drug therapy	• Thiazide diuretic • Urine alkalinization (potassium citrate/bicarbonate salt) • Allopurinol (for hyperuricosuria-related stones)

Renal stone formation involves the abnormal excretion of stone-forming salts and/or a deficiency of urinary inhibitors of crystal formation due to metabolic disorders or dietary factors. Calcium stones are the most common type of stone seen and usually present in the third or fourth decade of life. Most patients have hypercalciuria (24-hour urinary calcium excretion >4 mg/kg).

This patient most likely has recurrent calcium stones due to idiopathic hypercalciuria, which is best treated with increased fluid intake, dietary sodium and protein restriction, and thiazide/amiloride diuretics. The mild volume depletion caused by **thiazide diuretics** leads to a compensatory rise in reabsorption of sodium and water with resulting increased passive reabsorption of calcium. Thiazides are

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This patient most likely has recurrent calcium stones due to idiopathic hypercalciuria, which is best treated with increased fluid intake, dietary sodium and protein restriction, and thiazide/amiloride diuretics. The mild volume depletion caused by **thiazide diuretics** leads to a compensatory rise in reabsorption of sodium and water with resulting increased passive reabsorption of calcium. Thiazides are also thought to modulate calcium channels on the tubular membrane. Lowering the urinary concentration of calcium reduces its precipitation as insoluble calcium salts.

(Choices A and C) Aldosterone antagonists like spironolactone or eplerenone are potassium-sparing diuretics. Furosemide is a loop diuretic. They all increase urinary calcium excretion, potentially worsening stone formation.

(Choice B) Increased sodium load from high dietary sodium reduces reabsorption of sodium and thereby reduces the passive reabsorption of calcium. Typically <100 mEq/day of sodium is recommended.

(Choice D) Calcium binds with oxalate in the gut to form unabsorbable calcium oxalate. Reduced dietary calcium leads to an increase in oxalate absorption in the gut, which is then excreted into the urine and binds urinary calcium to form calcium oxalate stones. Excess calcium supplementation (ie, not from food sources) is also associated with increased stone formation.

Educational objective:
Measures to prevent urinary calcium stone formation include increasing fluid intake, following a low-sodium, low-protein diet, maintaining moderate calcium intake, and taking thiazide diuretics to lower urinary calcium excretion.

References

- Medical management to prevent recurrent nephrolithiasis in adults: a systematic review for an American College of Physicians Clinical Guideline.

A 14-year-old boy comes to the physician with two days of fever and nasal discharge. He has also had malaise, fatigue, and myalgia. He has no other medical problems. Family history is negative for any kidney disease. His temperature is 38.8° C (101.9° F), blood pressure is 122/74 mm Hg, pulse is 95/min, and respirations are 15/min. Examination shows no abnormalities. An incidental urine dipstick testing shows 2+ proteinuria but no hematuria, pyuria, or active urine sediment. Serum creatinine is within normal limits. Which of the following is the most appropriate next step in management?

- A. Repeat dipstick testing on two subsequent occasions
- B. Check serum protein and albumin levels
- C. Order 24-hour urinary collection for protein
- D. Order renal ultrasound
- E. Reassure, with no further workup
- F. Renal biopsy

Submit

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- ✔ A. Repeat dipstick testing on two subsequent occasions [35%]
- B. Check serum protein and albumin levels [9%]
- C. Order 24-hour urinary collection for protein [23%]
- D. Order renal ultrasound [2%]
- E. Reassure, with no further workup [26%]
- F. Renal biopsy [1%]

Omitted
Correct answer
A

35%
Answered correctly

2 Seconds
Time Spent

10/22/2018
Last Updated

Explanation

A urine dipstick can be positive in up to 10% of school-aged children. Proteinuria in children can be transient (intermittent), orthostatic, or persistent. Transient proteinuria is the most common cause of proteinuria and can be caused by fever, exercise,

Explanation

A urine dipstick can be positive in up to 10% of school-aged children. Proteinuria in children can be transient (intermittent), orthostatic, or persistent. Transient proteinuria is the most common cause of proteinuria and can be caused by fever, exercise, seizures, stress, or volume depletion. Orthostatic proteinuria is very common in adolescent boys and is defined as increased protein when the patient is in an upright position that returns to normal when the patient is recumbent. If the urinalysis shows no hematuria and is otherwise normal, the urine dipstick should be repeated on at least two additional specimens **(Choice A)**. If these subsequent tests are negative for protein, the diagnosis is transient proteinuria. Transient and orthostatic proteinurias are usually benign conditions that require no further evaluation.

If the proteinuria persists on the repeat sample or if any of the initial studies are abnormal, the patient should be referred to a pediatric nephrologist and evaluated for underlying renal disease. Further investigation may include 24-hour urinary collection for protein, renal ultrasound, and, possibly, renal biopsy **(Choices C, D, and F)**. This patient presents with proteinuria during a febrile illness and should be tested again in the future to rule out persistent proteinuria.

(Choice B) Checking serum protein (albumin) has little value in evaluating proteinuria since many patients with even nephrotic-range proteinuria have normal serum protein levels.

Educational objective:
Transient proteinuria is the most common cause of isolated proteinuria in children and should be reevaluated with a repeat urine dipstick testing on two separate occasions to rule out persistent proteinuria, which requires further evaluation for underlying renal disease.

References

- Isolated proteinuria: analysis of a school-age population

A 17-year-old man comes to the emergency department and complains of intense left flank pain that radiates to the groin. He refers to his symptom as "stone passage," which he has experienced "so many times since childhood." His uncle has the same problem. Urinalysis shows hexagonal crystals. The urinary cyanide nitroprusside test is positive. Which of the following is the most likely cause of this patient's condition?

A. Amino acid transport abnormality
B. Parathyroid adenoma
C. Abnormality of uric acid metabolism
D. Excessive intestinal reabsorption of oxalate
E. Infection

Submit

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- ✔ A. Amino acid transport abnormality [61%]
- B. Parathyroid adenoma [2%]
- C. Abnormality of uric acid metabolism [17%]
- D. Excessive intestinal reabsorption of oxalate [15%]
- E. Infection [2%]

Omitted
Correct answer
A

61%
Answered correctly

2 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

This patient's personal history of **recurrent stones since childhood**, **family history** of nephrolithiasis, and typical **hexagonal crystals** on urinalysis is suggestive of **cystinuria**.

Cystinuria is a group of disorders (with several modes of inheritance) characterized by **impaired transport** of **cystine** and the **dibasic amino acids** ornithine, lysine, and arginine by the brush borders of renal tubular and intestinal epithelial cells. This leads to

Explanation

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Cystinuria is a group of disorders (with several modes of inheritance) characterized by **impaired transport** of **cystine** and the **dibasic amino acids** ornithine, lysine, and arginine by the brush borders of renal tubular and intestinal epithelial cells. This leads to decreased reabsorption (ie, increased urinary excretion) of cystine, which—unlike ornithine, lysine, and arginine—is poorly soluble in water. As a result, patients form cystine stones, which are hard and can be radiolucent (or less radioopaque than calcium stones).

The urinary **cyanide-nitroprusside test** can detect elevated cystine levels and can help confirm the diagnosis; it is used as a qualitative screening test and is particularly helpful to detect individuals who are homozygous for the mutations causing impaired amino acid transport.

(Choices B, C, and D) Primary hyperparathyroidism due to parathyroid adenoma can lead to recurrent stones, but these are typically calcium oxalate stones. Excess uric acid excretion in the urine due to hyperuricemia (eg, gout) causes uric acid stones, and excess intestinal reabsorption of oxalate as a result of intestinal conditions (eg, Crohn disease, bariatric surgery) causes calcium oxalate stones. The constellation of recurrent stones since childhood, positive family history, hexagonal crystals, and positive cyanide-nitroprusside test is more consistent with cystinuria.

(Choice E) Infection with urease-producing microorganisms can result in struvite stone formation, but these stones rarely present acutely and are more likely to cause mild flank pain, urinary tract infection, hematuria.

Educational objective:
Cystinuria is an inherited disease causing recurrent renal stone formation. A personal history of recurrent kidney stones from childhood and a positive family history for nephrolithiasis should raise suspicion for the diagnosis. Urinalysis shows typical hexagonal crystals. The urinary cyanide-nitroprusside test is used as a qualitative screening procedure.

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Educational objective:
Cystinuria is an inherited disease causing recurrent renal stone formation. A personal history of recurrent kidney stones from childhood and a positive family history for nephrolithiasis should raise suspicion for the diagnosis. Urinalysis shows typical hexagonal crystals. The urinary cyanide-nitroprusside test is used as a qualitative screening procedure.

A 65-year-old woman is brought to the emergency department due to altered mental status. Her family notes that she awoke in the morning with a headache and became progressively incoherent over the course of the day. The day before, she had nausea and vomited several times during the day. Three weeks ago, the patient was diagnosed with depression and started on sertraline. Her only other medical problem is rheumatoid arthritis, which was diagnosed a year ago and is well controlled with adalimumab. She takes no other medications, supplements, or herbs. She is afebrile. Blood pressure is 110/70 mm Hg without an orthostatic drop, pulse is 75/min, and respirations are 15/min. She is disoriented and irritable. Neck is supple. Neurologic examination is otherwise unremarkable. There is no papilledema. Mucous membranes are moist. There is no jugular venous distension. Lungs are clear to auscultation. Abdomen is soft, nontender, and nondistended. There is no peripheral edema. Laboratory results are as follows:

Sodium	119 mEq/L
Potassium	4.0 mEq/L
Bicarbonate	24 mEq/L
Blood glucose	104 mg/dL
Blood urea nitrogen	20 mg/dL
Uric acid	1.5 mg/dL

Serum osmolality is 260 mOsm/kg H_2O, urine osmolality is 500 mOsm/kg H_2O, and urine sodium is 56 mmol/L. What is the most likely cause of this patient's hyponatremia?

A. Adverse effect of a drug
B. Decreased solute intake
C. Intracranial neoplasm

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- A. Adverse effect of a drug
- B. Decreased solute intake
- C. Intracranial neoplasm
- D. Intravascular volume depletion
- E. Mineralocorticoid deficiency
- F. Polydipsia
- G. Renal resistance to antidiuretic hormone

Submit

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- A. Adverse effect of a drug [52%]
- B. Decreased solute intake [3%]
- C. Intracranial neoplasm [13%]

Potassium	4.0 mEq/L
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- ✔ A. Adverse effect of a drug [52%]
- B. Decreased solute intake [3%]
- C. Intracranial neoplasm [13%]
- D. Intravascular volume depletion [7%]
- E. Mineralocorticoid deficiency [6%]
- F. Polydipsia [7%]
- G. Renal resistance to antidiuretic hormone [9%]

Omitted
Correct answer
A

52%
Answered correctly

4 Seconds
Time Spent

11/06/2018
Last Updated

Explanation

Syndrome of inappropriate antidiuretic hormone	
Etiologies	• CNS disturbance (eg, stroke, hemorrhage, trauma) • Medications (eg, carbamazepine, SSRIs, NSAIDs) • Lung disease (eg, pneumonia) • Ectopic ADH secretion (eg, small cell lung cancer) • Pain &/or nausea
Clinical features	• Mild/moderate hyponatremia - nausea, forgetfulness • Severe hyponatremia - seizures, coma • Euvolemia (eg, moist mucous membranes, no edema, no JVD)
Laboratory findings	• Hyponatremia • Serum osmolality <275 mOsm/kg H_2O (hypotonic) • Urine osmolality >100 mOsm/kg H_2O • Urine sodium >40 mEq/L
Management	• Fluid restriction ± salt tablets • Hypertonic (3%) saline for severe hyponatremia

ADH = antidiuretic hormone; **CNS** = central nervous system; **JVD** = jugular venous distension; **NSAIDs** = nonsteroidal anti-inflammatory drugs; **SSRIs** = selective serotonin reuptake inhibitors.

This patient has symptomatic **hypotonic** (serum osmolality <275 mOsm/kg H_2O) **hyponatremia** due to the **syndrome of**

This patient has symptomatic **hypotonic** (serum osmolality <275 mOsm/kg H_2O) **hyponatremia** due to the **syndrome of inappropriate antidiuretic hormone secretion (SIADH)**. Normally, the kidneys are able to excrete free water at a sufficient rate to prevent blood hypotonicity. However, in SIADH, inappropriately high levels of antidiuretic hormone (ADH) prevent the kidneys from excreting dilute urine (leading to **urine osmolality >100 mOsm/kg H_2O**), and hypotonicity and hyponatremia develop. Patients with SIADH are typically **euvolemic**; therefore, urine sodium concentration is typically elevated (>40 mEq/L), unlike in patients with hypovolemia. In addition, in SIADH, serum uric acid levels are characteristically low, serum potassium level is normal, and acid-base status is normal.

Selective serotonin reuptake inhibitors are commonly associated with SIADH and **elderly** patients are especially at risk. Nausea can further exacerbate SIADH. Symptoms of SIADH vary depending on the severity and rate of development of hyponatremia; patients can be asymptomatic, mildly symptomatic (eg, nausea, lethargy) or, in severe cases, present with profound confusion, seizures, or coma.

(Choices B and F) Both severely decreased solute intake (in the setting of ongoing free water intake) and polydipsia can lead to hyponatremia. However, in these situations, ADH levels are normal and patients are able to excrete dilute urine; therefore, urine osmolality would be appropriately low (<100 mOsm/kg H_2O).

(Choice C) Intracranial neoplasms may lead to hyponatremia due to SIADH or cerebral salt wasting (caused by increased intracranial pressure). However, the absence of papilledema and focal neurologic findings (eg, focal weakness) in this patient makes intracranial neoplasm less likely.

(Choice D) Intravascular volume depletion occurs in dehydration and can lead to hyponatremia due to decreased renal perfusion and excess free water retention. The urine sodium concentration should be low (<40 mEq/L) as the kidneys attempt to retain solute in an effort to increase blood volume.

(Choice E) Mineralocorticoid deficiency typically presents with hyperkalemia and metabolic acidosis, which are not present in this patient.

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(Choice E) Mineralocorticoid deficiency typically presents with hyperkalemia and metabolic acidosis, which are not present in this patient.

(Choice G) Renal resistance to ADH occurs in nephrogenic diabetes insipidus. The result is uncontrolled loss of free water by the kidneys and consequent hypernatremia.

Educational objective:
The syndrome of inappropriate antidiuretic hormone secretion is characterized by hypotonic hyponatremia and euvolemia. Low serum osmolality (<275 mOsm/kg), high urine osmolality (>100 mOsm/kg), and an elevated urine sodium concentration (>40 mEq/L) strongly suggest the diagnosis.

References

- Hyponatremia and the syndrome of inappropriate secretion of antidiuretic hormone associated with the use of selective serotonin reuptake inhibitors: a review of spontaneous reports.

A 16-year-old girl comes to the clinic with 2 days of a burning sensation with voiding. She had sexual intercourse last week and her partner used a condom. The patient says, "I always pee right after sex." She has no vaginal discharge and her vital signs are normal. Examination shows suprapubic tenderness. Urinalysis shows positive nitrites, positive leukocyte esterase, 50 white blood cells/high-power field, and many bacteria. Urine β-hCG is negative. Which of the following is the most likely cause of this patient's infection?

- A. Ascending infection
- B. Hematogenous spread of infection
- C. Lymphatic spread of infection
- D. Poor genital hygiene
- E. Sexually transmitted infection

Submit

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- ✔ A. Ascending infection [89%]
- B. Hematogenous spread of infection [0%]
- C. Lymphatic spread of infection [0%]
- D. Poor genital hygiene [8%]
- E. Sexually transmitted infection [1%]

Omitted
Correct answer
A

89%
Answered correctly

3 Seconds
Time Spent

11/01/2018
Last Updated

Explanation

Urinary tract infection	
Microbiology	*E coli* most common cause

Explanation

Urinary tract infection		
Microbiology	*E coli* most common cause	
Clinical features	**Cystitis**	Dysuria, frequency, urgency, hematuria, suprapubic pain
	Pyelonephritis	Fever >38 C (100.4 F), chills, flank pain, costovertebral angle tenderness & nausea/vomiting, +/− cystitis symptoms
Diagnosis	Urinalysis & urine culture	
Treatment	Antibiotics	

This patient's clinical features (suprapubic pain, dysuria) and abnormal urinalysis (pyuria, bacteriuria) are highly suggestive of a urinary tract infection (UTI). The urethra and bladder are normally sterile; UTI is typically acquired when bacteria around the vaginal introitus **ascend the urethra** to the bladder. Compared to men, women are at increased risk for UTI due to a shorter urethra. Recent **sexual activity** (eg, honeymoon cystitis) is a common predisposing factor, as bacteria can be introduced to the introitus during intercourse. Enteric organisms, particularly coliforms such as ***Escherichia coli***, are the most common causes of UTI. Bacterial infection of the bladder results in cystitis. Untreated cystitis can spread up the ureters to the kidneys, leading to pyelonephritis.

This patient's clinical features (suprapubic pain, dysuria) and abnormal urinalysis (pyuria, bacteriuria) are highly suggestive of a urinary tract infection (UTI). The urethra and bladder are normally sterile; UTI is typically acquired when bacteria around the vaginal introitus **ascend the urethra** to the bladder. Compared to men, women are at increased risk for UTI due to a shorter urethra. Recent **sexual activity** (eg, honeymoon cystitis) is a common predisposing factor, as bacteria can be introduced to the introitus during intercourse. Enteric organisms, particularly coliforms such as ***Escherichia coli***, are the most common causes of UTI. Bacterial infection of the bladder results in cystitis. Untreated cystitis can spread up the ureters to the kidneys, leading to pyelonephritis.

(Choices B and C) Bacteria can disseminate hematogenously to the kidneys via the renal artery. This mechanism is common in young infants but rare in older children and adults.

(Choice D) Poor vaginal hygiene (eg, wiping back to front, failure to change menstrual pads or tampons regularly, remaining in wet or sweaty undergarments) can increase the concentration of bacteria in the vaginal area and has been associated with increased UTI risk. However, UTI is common even in women with post-coital voiding and no evidence of poor vaginal hygiene, such as this patient.

(Choice E) All sexually active patients should be screened annually for sexually transmitted infection (STI). However, this patient's history of barrier protection, normal genital examination, and evidence of bacteriuria on urinalysis make a STI less likely as the cause of her symptoms.

Educational objective:
Urinary tract infection (UTI) most commonly arises by bacteria ascending into the bladder from the vaginal introitus. Sexual intercourse is an important risk factor for UTIs in women due to introduction of uropathogens into the urethra.

References

- Urinary tract infection pathogenesis: host factors.
- Diagnosis and treatment of acute uncomplicated cystitis.

A 22-year-old man comes to the urgent care clinic complaining of dark urine he noticed earlier this morning. He is recovering from an upper respiratory tract infection that started 4 days ago. The patient's temperature is 37.1 C (98.9 F), blood pressure is 145/92 mm Hg, pulse is 80/min, and respirations are 14/min. Physical examination shows no skin rash and no joint abnormalities. Laboratory results are as follows:

Urinalysis

Glucose	Negative
Protein	1+
Ketones	Negative
Leukocyte esterase	Negative
Nitrites	Negative
White blood cells	3-6/hpf
Red blood cells	30-50/hpf
Casts	Red blood cells

Serum chemistry

Serum sodium	138 mEq/L
Serum potassium	4.5 mEq/L
Bicarbonate	22 mEq/L
Blood urea nitrogen	18 mg/dL

Bicarbonate	22 mEq/L
Blood urea nitrogen	18 mg/dL
Serum creatinine	1.4 mg/dL

Serum complement levels (C3 and C4) are within normal limits, and other serological workup is pending. Which of the following is the most likely diagnosis?

- A. Acute interstitial nephritis
- B. Acute postinfectious glomerulonephritis
- C. Alport syndrome
- D. Anti-glomerular basement membrane disease
- E. Benign recurrent hematuria
- F. Goodpasture's syndrome
- G. Henoch-Schönlein purpura
- H. IgA nephropathy
- I. Lupus nephritis

Submit

A 22-year-old man comes to the urgent care clinic complaining of dark urine he noticed earlier this morning. He is recovering from an upper respiratory tract infection that started 4 days ago. The patient's temperature is 37.1 C (98.9 F), blood pressure is 145/92 mm Hg, pulse is 80/min, and respirations are 14/min. Physical examination shows no skin rash and no joint abnormalities. Laboratory results are as follows:

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Blood urea nitrogen	18 mg/dL

Blood urea nitrogen	18 mg/dL
Serum creatinine	1.4 mg/dL

Serum complement levels (C3 and C4) are within normal limits, and other serological workup is pending. Which of the following is the most likely diagnosis?

- A. Acute interstitial nephritis [3%]
- B. Acute postinfectious glomerulonephritis [31%]
- C. Alport syndrome [0%]
- D. Anti-glomerular basement membrane disease [1%]
- E. Benign recurrent hematuria [1%]
- F. Goodpasture's syndrome [2%]
- G. Henoch-Schönlein purpura [0%]
- ✔ H. IgA nephropathy [59%]
- I. Lupus nephritis [0%]

Omitted
Correct answer
H

59%
Answered correctly

8 Seconds
Time Spent

12/03/2018
Last Updated

	IgA nephropathy	Postinfectious glomerulonephritis
Clinical presentation	• Usually within 5 days of upper respiratory tract infection (**synpharyngitic**) • More common in young adult men (age 20-30) • Recurrent gross hematuria	• Usually 10-21 days after upper respiratory tract infection (post-pharyngitic) • More common in children (age 6-10), but can occur in adults • Gross hematuria • Adults can be asymptomatic or develop acute nephritic syndrome
Diagnosis	• Normal serum complements • Mesangial IgA deposits seen in kidney biopsy	• Low C3 complement • Elevated anti-streptolysin O &/or anti-DNAse B • Kidney biopsy with subepithelial humps consisting of C3 complement
Prognosis	• Usually benign • Possible rapidly progressive glomerulonephritis or nephrotic syndrome with worse prognosis	• Children have good prognosis • Possible chronic kidney disease in adults

This patient's gross hematuria is associated with an upper respiratory tract infection (URI), hypertension, proteinuria, red blood cell casts, and acute kidney injury. This suggests glomerulonephritis (GN). After a URI, GN can occur due to IgA nephropathy and postinfectious GN. Postinfectious GN typically occurs 10-21 days after a URI (post-pharyngitic) and is more common in children. Adults can be asymptomatic or develop acute nephritic syndrome. Laboratory studies usually show low C3 complement and elevated anti-streptolysin O and/or anti-DNAse B. This patient's normal serum complements and hematuria time course (4 days after URI) are not consistent with postinfectious GN **(Choice B)**.

IgA nephropathy is the most common cause of GN in adults. Patients can have recurrent episodes of hematuria usually within 5 days of a URI (synpharyngitic). IgA nephropathy is more common in young adult men. Serum complement levels tend to be normal with mesangial IgA deposits seen in kidney biopsy. Patients can have a benign course or develop rapidly progressive GN or nephrotic syndrome.

(Choice A) Acute interstitial nephritis is an acute inflammatory process involving the renal tubules and interstitium following exposure to a drug (eg, antibiotics, nonsteroidal anti-inflammatory drugs, proton-pump inhibitors). Other findings can include fever, skin rash, eosinophilia, eosinophiluria, or white blood cell casts. However, this patient lacks other findings (eg, fever, skin rash) and has no history of drug exposure; this makes acute interstitial nephritis less likely.

(Choice C) Alport syndrome is an X-linked defect in collagen-IV formation and presents with hearing loss, ocular abnormalities, hematuria, and progressive renal insufficiency. Kidney biopsy usually shows thinning of the glomerular basement membrane. However, post-URI gross hematuria is not typically seen in Alport syndrome.

(Choices D and F) Anti-glomerular basement membrane (anti-GBM) disease is due to anti-GBM antibodies against collagen IV (alpha-5 chain) damaging the glomeruli and alveolar lining. Anti-GBM disease manifests as either a renal limited process (rapidly progressive GN) or alveolar hemorrhage (pulmonary renal syndrome). Goodpasture's syndrome refers to a pulmonary-renal syndrome that is a manifestation of anti-GBM disease. This patient's absence of significantly elevated creatinine makes this less likely.

(Choice E) Benign recurrent hematuria, also known as thin basement membrane nephropathy, is a benign familial condition that

progressive GN) or alveolar hemorrhage (pulmonary renal syndrome). Goodpasture's syndrome refers to a pulmonary-renal syndrome that is a manifestation of anti-GBM disease. This patient's absence of significantly elevated creatinine makes this less likely.

(Choice E) Benign recurrent hematuria, also known as thin basement membrane nephropathy, is a benign familial condition that presents as isolated microscopic hematuria. However, it does not worsen kidney function or present as gross hematuria after a URI.

(Choice G) Henoch-Schönlein purpura is a systemic form of IgA involvement of the glomeruli, skin, joints, and intestines. Henoch-Schönlein purpura is more common in children and presents as an erythematous and papular skin rash involving the dorsal aspect of lower extremities, abdominal pain, arthralgia/arthritis, and microscopic hematuria/proteinuria.

(Choice I) Lupus nephritis can present as nephritic syndrome, nephrotic syndrome, rapidly progressive GN, or pulmonary-renal syndrome. Patients usually have low complement levels (C3 and C4) and positive lupus antibodies (anti-nuclear antibodies, anti-dsDNA, anti-Smith antibodies). This patient's normal serum complement levels make this unlikely.

Educational objective:
IgA nephropathy is the most common cause of glomerulonephritis in adults. Patients have recurrent episodes of gross hematuria, usually within 5 days after an upper respiratory tract infection (synpharyngitic presentation). IgA nephropathy is differentiated from postinfectious glomerulonephritis based on earlier onset of upper respiratory tract infection-related glomerulonephritis and normal serum complement levels. Kidney biopsy can also help differentiate these 2 processes.

References

- The diagnosis of glomerular diseases: acute glomerulonephritis and the nephrotic syndrome.
- Immunoglobulin A nephropathy: a review of current literature on emerging pathophysiology

A 24-year-old woman comes to the physician because of a 24-hour history of right flank pain, burning micturition and high-grade fever with chills. Her temperature is 38.9° C (102° F), blood pressure is 90/60 mm Hg, pulse is 130/min, and respirations are 20/min. Physical examination shows costovertebral angle tenderness. Which of the following is the most likely urine dipstick finding in this patient?

A. Positive for nitrites and esterase
B. Positive for nitrites only
C. Positive for esterase only
D. Negative for both esterase and nitrites

Submit

A 24-year-old woman comes to the physician because of a 24-hour history of right flank pain, burning micturition and high-grade fever with chills. Her temperature is 38.9° C (102° F), blood pressure is 90/60 mm Hg, pulse is 130/min, and respirations are 20/min. Physical examination shows costovertebral angle tenderness. Which of the following is the most likely urine dipstick finding in this patient?

- ✔ A. Positive for nitrites and esterase [86%]
- B. Positive for nitrites only [6%]
- C. Positive for esterase only [4%]
- D. Negative for both esterase and nitrites [2%]

Omitted
Correct answer
A

86%
Answered correctly

2 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

Dipsticks are commercially available kits that detect the presence of leukocyte esterase and nitrites in the urine of patients with suspected UTI. Leukocyte esterase in the urine indicates significant pyuria, whereas nitrites signify the presence of Enterobacteriaceae, which converts urinary nitrates to nitrites. (Remember that the most likely responsible organism of UTI is E.coli.) The advent of dipstick testing has significantly reduced the cost associated with urine culture; however, dipsticks are associated with a high false positive rate and high false negative rate. For this reason, a negative dipstick test in a patient with symptoms of UTI should still have urine cultures done.

D. Negative for both esterase and nitrites [2%]

Omitted
Correct answer
A

86%
Answered correctly

2 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

Dipsticks are commercially available kits that detect the presence of leukocyte esterase and nitrites in the urine of patients with suspected UTI. Leukocyte esterase in the urine indicates significant pyuria, whereas nitrites signify the presence of Enterobacteriaceae, which converts urinary nitrates to nitrites. (Remember that the most likely responsible organism of UTI is E.coli.) The advent of dipstick testing has significantly reduced the cost associated with urine culture; however, dipsticks are associated with a high false positive rate and high false negative rate. For this reason, a negative dipstick test in a patient with symptoms of UTI should still have urine cultures done.

In this case, the patient's clinical presentation (i.e., pyuria, significant bacteriuria) is highly suggestive of acute pyelonephritis. The expected dipstick finding is positive for both nitrites and esterase.

Educational objective:
Dipsticks are commercially available kits that detect the presence of leukocyte esterase and nitrite in the urine of patients with suspected UTI. Positive leukocyte esterase signifies significant pyuria and positive nitrites indicate the presence of Enterobacteriaceae.

A 3-year-old boy is brought to the physician for evaluation of an abdominal "swelling" discovered by his mother during bathing. He has no pain or changes in appetite. Review of systems is negative. He was recently treated with antibiotics for streptococcal pharyngitis but has no other medical problems. Weight and height have been tracking along the 75th percentile. Vital signs are normal. Examination shows a well-appearing, well-nourished boy. A firm, nontender mass is palpable in the left abdomen. Urinalysis results are as follows:

Color	Yellow
Blood	2+
Glucose	Negative
Protein	Negative
Nitrite	Negative
Leukocyte esterase	Negative
Bacteria	None
Red blood cells	34/hpf
White blood cells	1/hpf

What is the most likely diagnosis?

A. Nephrolithiasis
B. Neuroblastoma
C. Posterior urethral valves

Protein	Negative
Nitrite	Negative
Leukocyte esterase	Negative
Bacteria	None
Red blood cells	34/hpf
White blood cells	1/hpf

What is the most likely diagnosis?

- A. Nephrolithiasis
- B. Neuroblastoma
- C. Posterior urethral valves
- D. Poststreptococcal glomerulonephritis
- E. Pyelonephritis
- F. Renal cell carcinoma
- G. Wilms tumor (nephroblastoma)

Submit

A 3-year-old boy is brought to the physician for evaluation of an abdominal "swelling" discovered by his mother during bathing. He has no pain or changes in appetite. Review of systems is negative. He was recently treated with antibiotics for streptococcal pharyngitis but has no other medical problems. Weight and height have been tracking along the 75th percentile. Vital signs are normal. Examination shows a well-appearing, well-nourished boy. A firm, nontender mass is palpable in the left abdomen. Urinalysis results are as follows:

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Glucose	Negative
Protein	Negative
Nitrite	Negative
Leukocyte esterase	Negative
Bacteria	None
Red blood cells	34/hpf
White blood cells	1/hpf

What is the most likely diagnosis?

A. Nephrolithiasis [0%]
B. Neuroblastoma [5%]
C. Posterior urethral valves [0%]

Nitrite	Negative
Leukocyte esterase	Negative
Bacteria	None
Red blood cells	34/hpf
White blood cells	1/hpf

What is the most likely diagnosis?

- A. Nephrolithiasis [0%]
- B. Neuroblastoma [5%]
- C. Posterior urethral valves [0%]
- D. Poststreptococcal glomerulonephritis [12%]
- E. Pyelonephritis [0%]
- F. Renal cell carcinoma [0%]
- ✔ G. Wilms tumor (nephroblastoma) [80%]

Omitted
Correct answer
G

80%
Answered correctly

4 Seconds
Time Spent

09/29/2018
Last Updated

Explanation

Wilms tumor (nephroblastoma)	

Epidemiology	• Most common renal malignancy in childhood
	• Fourth most common childhood cancer
	• Peak age 2-5 years
	• Usually sporadic
	• Associated syndromes:
	  • WAGR (**W**ilms tumor, **A**niridia, **G**enitourinary anomalies, intellectual disability [mental **R**etardation])
	  • Beckwith-Wiedemann syndrome
	  • Denys-Drash syndrome

Clinical presentation	• Asymptomatic, firm, smooth, abdominal mass that does not cross midline
Treatment	• Tumor excision or nephrectomy • Chemotherapy • +/- Radiation therapy
Prognosis	• 5-year survival rate with treatment: 90%

Wilms tumor (nephroblastoma) is the **most common primary renal neoplasm of childhood**. It is usually diagnosed at **age 2-5**

Wilms tumor (nephroblastoma) is the **most common primary renal neoplasm of childhood**. It is usually diagnosed at **age 2-5 years** and affects a single kidney. The most common presentation is an **asymptomatic abdominal mass** that is found incidentally by a caretaker or physician. Some patients have abdominal pain, hypertension, hematuria, and fever. Less than 10% of patients have bilateral renal involvement (stage V disease). Although the lungs are the most common site of metastatic spread, children rarely present with pulmonary symptoms.

Abdominal ultrasonography should be the first step in imaging to differentiate Wilms tumor from other causes of abdominal masses. It should be followed by **contrast-enhanced computed tomography of the abdomen** to evaluate the nature and extent of the mass and of the chest to identify any **pulmonary metastases**. Treatment includes surgery and chemotherapy with the addition of radiation therapy for high-stage disease. Survival rates are excellent especially if treated in the early stages.

(Choice A) Kidney stones can cause hematuria but are usually very painful. Even large staghorn calculi would present as urinary tract infection and pain rather than abdominal mass.

(Choice B) **Neuroblastoma** should be considered when examining a child with an abdominal mass. It is the third most common pediatric cancer after leukemia and brain tumors but the most common cancer in the **first year of life**. Neuroblastoma can arise anywhere in the sympathetic nervous system but typically involves the adrenal glands and presents as an abdominal mass that **crosses the midline** with systemic symptoms. This patient's age and asymptomatic abdominal mass are more characteristic of Wilms tumor than neuroblastoma.

(Choice C) Posterior urethral valves can cause bilateral obstructive uropathy and are usually diagnosed prenatally. Boys who present later in life typically have frequent urinary tract infections and signs of renal failure. The associated hydronephrosis is not palpable, making this diagnosis unlikely.

(Choice D) Glomerulonephritis can occur as a complication of streptococcal pharyngitis (even if treated). In addition to hematuria, patients typically have proteinuria, hypertension, and edema. Glomerulonephritis would not cause a firm abdominal mass.

(Choice E) Pyelonephritis typically presents with fever, chills, flank pain, pyuria (white blood cells >5-10/hpf), and bacteriuria. This patient has no symptoms, pyuria, or bacteriuria, making this diagnosis unlikely.

tract infection and pain rather than abdominal mass.

(Choice B) **Neuroblastoma** should be considered when examining a child with an abdominal mass. It is the third most common pediatric cancer after leukemia and brain tumors but the most common cancer in the **first year of life**. Neuroblastoma can arise anywhere in the sympathetic nervous system but typically involves the adrenal glands and presents as an abdominal mass that **crosses the midline** with systemic symptoms. This patient's age and asymptomatic abdominal mass are more characteristic of Wilms tumor than neuroblastoma.

(Choice C) Posterior urethral valves can cause bilateral obstructive uropathy and are usually diagnosed prenatally. Boys who present later in life typically have frequent urinary tract infections and signs of renal failure. The associated hydronephrosis is not palpable, making this diagnosis unlikely.

(Choice D) Glomerulonephritis can occur as a complication of streptococcal pharyngitis (even if treated). In addition to hematuria, patients typically have proteinuria, hypertension, and edema. Glomerulonephritis would not cause a firm abdominal mass.

(Choice E) Pyelonephritis typically presents with fever, chills, flank pain, pyuria (white blood cells >5-10/hpf), and bacteriuria. This patient has no symptoms, pyuria, or bacteriuria, making this diagnosis unlikely.

(Choice F) Renal cell carcinoma is the most common primary renal neoplasm. However, it occurs predominantly in men age 50-80 years and is rare in children.

Educational objective:
Wilms tumor is the most common pediatric renal malignancy. It should be suspected in a toddler with a firm, smooth, unilateral abdominal mass and hematuria.

References

- Management of Wilms' tumour: current practice and future goals.

- +/- Radiation therapy

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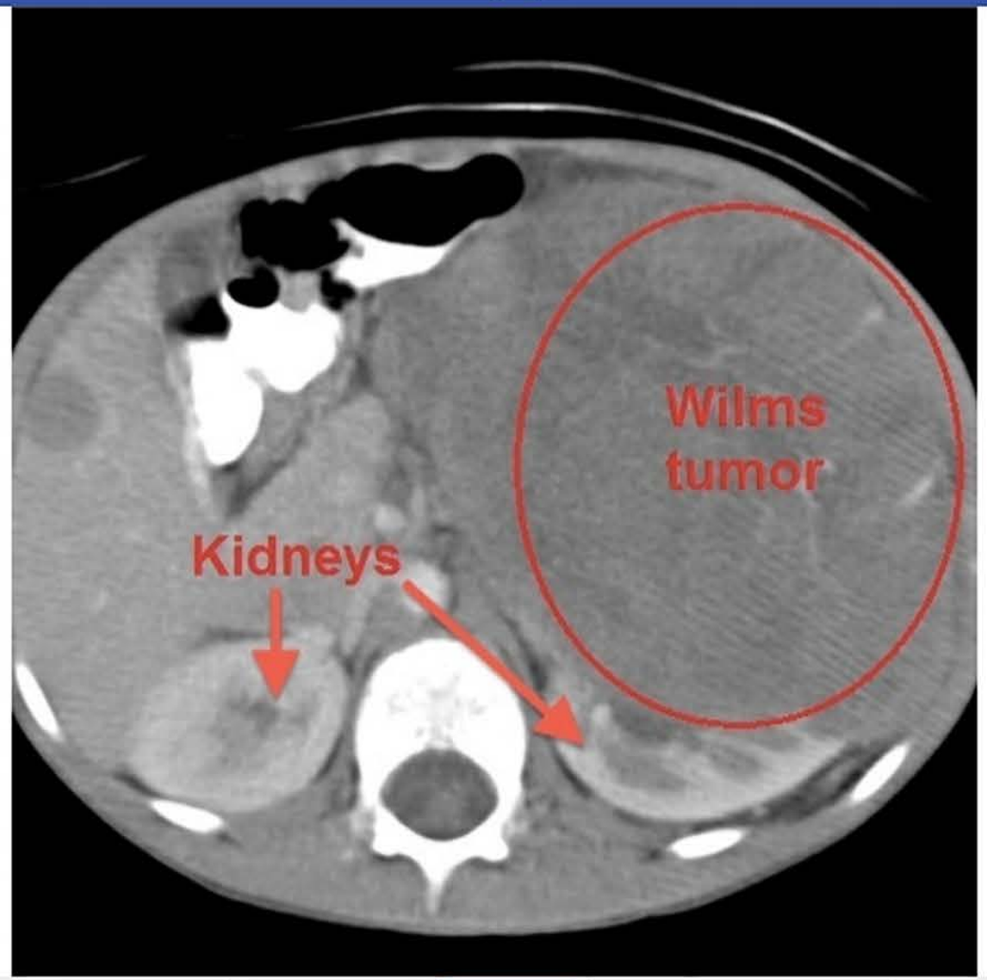

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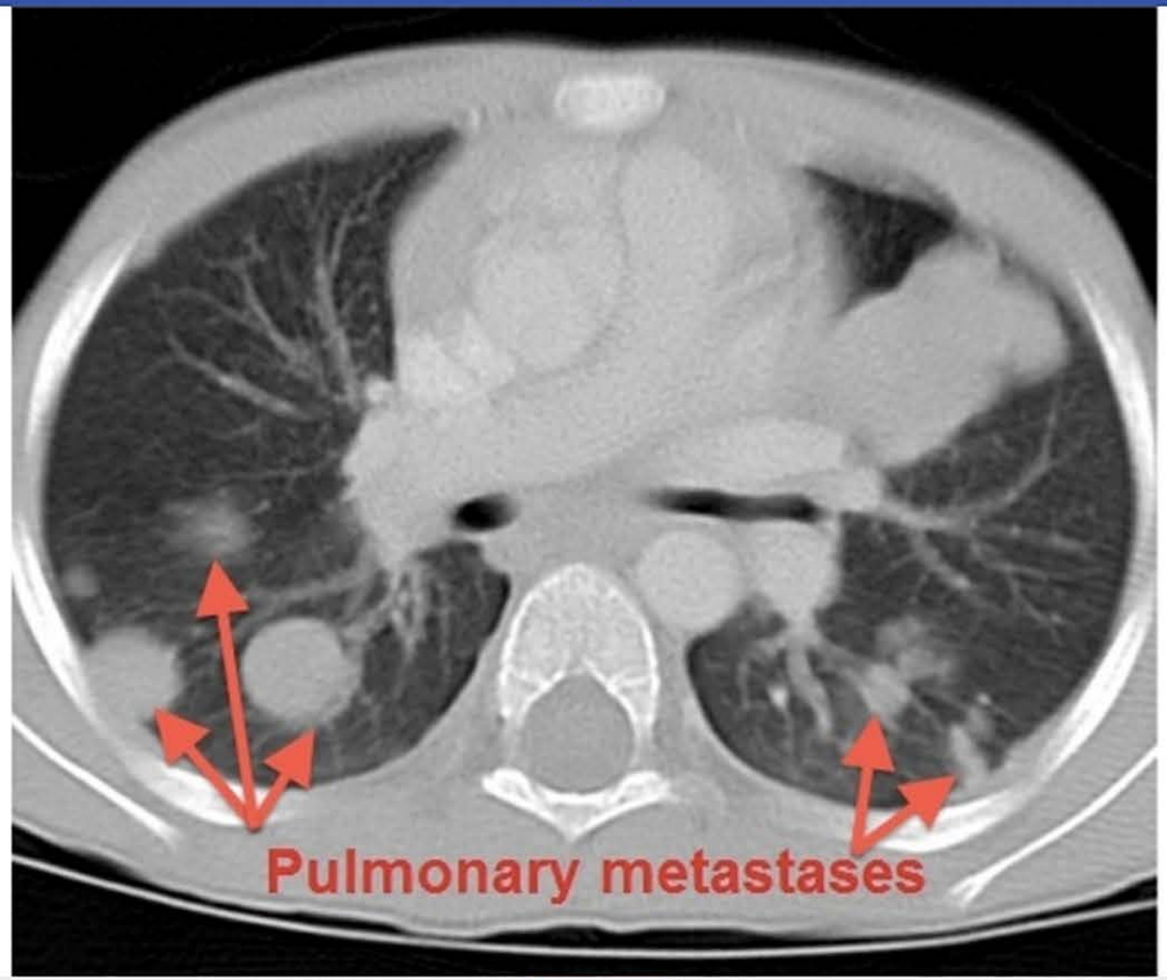

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A 6-year-old girl is brought to the office by her mother for evaluation of abdominal pain. The child has vomited a few times since the pain started this morning. The pain is diffuse, constant, and moderate in intensity. The patient has had no fevers, diarrhea, or bloody stools. Her mother says, "I was wondering if the pain could be due to a bug that's been going around at school." She first noticed an erythematous, macular rash over her daughter's legs and back 2 days ago that has become darker and more confluent today. The patient has a history of moderate persistent asthma for which she uses an inhaled fluticasone inhaler daily and albuterol as needed for wheezing. Her maternal aunt has systemic lupus erythematosus, and her paternal cousin has acute lymphoblastic leukemia that is in remission. Blood pressure is 95/60 mm Hg. Physical examination shows a mildly uncomfortable-appearing girl lying on the table. The abdomen is soft and has diffuse mild tenderness without rebound, guarding, or appreciable masses. A palpable, nonblanching rash is noted over the legs and back. The right knee is mildly swollen and painful with passive range of motion. Laboratory results are as follows:

Complete blood count	
Hemoglobin	13.5 g/dL
Platelets	350,000/mm^3
Leukocytes	9,100/mm^3
Serum chemistry	
Sodium	140 mEq/L
Potassium	3.6 mEq/L
Chloride	105 mEq/L
Bicarbonate	25 mEq/L

Chloride	105 mEq/L
Bicarbonate	25 mEq/L
Blood urea nitrogen	8 mg/dL
Creatinine	0.4 mg/dL
Calcium	9.2 mg/dL
Glucose	118 mg/dL

Urinalysis

Specific gravity	1.022
Protein	+1
Blood	large
Glucose	negative
Ketones	negative
Leukocyte esterase	negative
Nitrites	negative

Which of the following glomerular abnormalities is most likely present in this patient?

A. Glomerular basement membrane thickening

B. Glomerular basement membrane thinning

Urinalysis

Specific gravity	1.022
Protein	+1
Blood	large
Glucose	negative
Ketones	negative
Leukocyte esterase	negative
Nitrites	negative

Which of the following glomerular abnormalities is most likely present in this patient?

A. Glomerular basement membrane thickening
B. Glomerular basement membrane thinning
C. Linear deposition of IgG on the basement membrane
D. Localized areas of mesangial sclerosis and collapse
E. Mesangial deposition of IgA
F. Podocyte fusion

Submit

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Specific gravity	1.022
Protein	+1
Blood	large
Glucose	negative
Ketones	negative
Leukocyte esterase	negative
Nitrites	negative

Which of the following glomerular abnormalities is most likely present in this patient?

A. Glomerular basement membrane thickening [4%]

B. Glomerular basement membrane thinning [2%]

Specific gravity	1.022
Protein	+1
Blood	large
Glucose	negative
Ketones	negative
Leukocyte esterase	negative
Nitrites	negative

Which of the following glomerular abnormalities is most likely present in this patient?

- A. Glomerular basement membrane thickening [4%]
- B. Glomerular basement membrane thinning [2%]
- C. Linear deposition of IgG on the basement membrane [9%]
- D. Localized areas of mesangial sclerosis and collapse [4%]
- ✔ E. Mesangial deposition of IgA [75%]
- F. Podocyte fusion [3%]

Omitted
Correct answer
E

75%
Answered correctly

9 Seconds
Time Spent

07/17/2018
Last Updated

Explanation

Henoch-Schönlein purpura	
Pathogenesis	• IgA-mediated leukocytoclastic vasculitis
Clinical manifestations	• Palpable purpura • Arthritis/arthralgia • Abdominal pain, intussusception • Renal disease similar to IgA nephropathy
Laboratory findings	• Normal platelet count & coagulation studies • Normal to ↑ creatinine • Hematuria ± RBC casts ± proteinuria
Treatment	• Supportive (hydration & NSAIDs) for most patients • Hospitalization & systemic glucocorticoids in patients with severe symptoms

NSAIDs = nonsteroidal anti-inflammatory drugs; **RBC** = red blood cell.

This child's abdominal pain, lower extremity purpura (shown here in an adult), arthritis, and hematuria are consistent with **Henoch-Schönlein purpura** (HSP), an **IgA-mediated vasculitis** of the small vessels. The rash consists of nonblanching **palpable purpura** symmetrically distributed over the lower legs, back, and buttocks. **Arthralgia/arthritis** most commonly affects the hips, knees, and ankles and is typically transient, causing no permanent joint damage. Colicky **abdominal pain** (due to local vasculitis) is present in the majority of patients.

symmetrically distributed over the lower legs, back, and buttocks. **Arthralgia/arthritis** most commonly affects the hips, knees, and ankles and is typically transient, causing no permanent joint damage. Colicky **abdominal pain** (due to local vasculitis) is present in the majority of patients.

Renal involvement occurs in over one third of children and may present months after the onset of illness. Most patients have relatively mild disease characterized by **hematuria**, red cell casts, non–nephrotic-range **proteinuria**, and a normal or only slightly elevated serum creatinine. However, more severe complications (including nephrotic syndrome, hypertension, and acute renal failure) may occur. The diagnosis of HSP is usually made clinically; in children with atypical presentations, a renal biopsy may be helpful to confirm the diagnosis and demonstrates **mesangial deposition of IgA**.

(Choice A) Membranous nephropathy is characterized by thickening of the glomerular basement membrane. It is far more common in adults than children and presents with edema and proteinuria, not a rash.

(Choice B) Alport syndrome is a hereditary nephritis caused by abnormalities of type IV collagen. In addition to renal disease, patients develop sensorineural hearing loss and ocular abnormalities. Thinning and splitting of the glomerular basement membrane are seen on microscopy.

(Choice C) Goodpasture syndrome is caused by antibodies directed against the basement membrane and typically presents with pulmonary hemorrhage and glomerulonephritis. It is seen in adolescents and adults, and linear deposition of IgG on the basement membrane is characteristic.

(Choice D) Focal segmental glomerulosclerosis is the most common glomerular cause of end-stage renal disease in adults in the United States. It typically presents with edema and proteinuria and is characterized by localized regions of mesangial sclerosis and basement membrane collapse on light microscopy.

(Choice F) Minimal change disease is the most common form of nephrotic syndrome in children and typically presents with edema and proteinuria. Electron microscopy findings include fusion or flattening of the podocytes ("foot processes").

Educational objective:

Henoch-Schönlein purpura is an IgA-mediated small vessel vasculitis that manifests with palpable purpura on the lower extremities

in adults than children and presents with edema and proteinuria, not a rash.

(Choice B) Alport syndrome is a hereditary nephritis caused by abnormalities of type IV collagen. In addition to renal disease, patients develop sensorineural hearing loss and ocular abnormalities. Thinning and splitting of the glomerular basement membrane are seen on microscopy.

(Choice C) Goodpasture syndrome is caused by antibodies directed against the basement membrane and typically presents with pulmonary hemorrhage and glomerulonephritis. It is seen in adolescents and adults, and linear deposition of IgG on the basement membrane is characteristic.

(Choice D) Focal segmental glomerulosclerosis is the most common glomerular cause of end-stage renal disease in adults in the United States. It typically presents with edema and proteinuria and is characterized by localized regions of mesangial sclerosis and basement membrane collapse on light microscopy.

(Choice F) Minimal change disease is the most common form of nephrotic syndrome in children and typically presents with edema and proteinuria. Electron microscopy findings include fusion or flattening of the podocytes ("foot processes").

Educational objective:
Henoch-Schönlein purpura is an IgA-mediated small vessel vasculitis that manifests with palpable purpura on the lower extremities, arthralgia/arthritis, abdominal pain, and renal disease (hematuria ± proteinuria). Renal biopsy shows IgA deposition in the mesangium.

References

- Renal manifestations of Henoch-Schönlein purpura in a 6-month prospective study of 223 children.
- Interventions for preventing and treating kidney disease in Henoch-Schönlein purpura (HSP).

A 27-year-old man comes to the physician because of a 1-day history of fever and joint pains. He is being treated with cephalexin for a skin infection. His urine has turned darker. His temperature is 38.5° C (101.3° F), blood pressure is 125/70 mm Hg, pulse is 90/min, and respirations are 15/min. Examination shows a skin rash; examination otherwise shows no abnormalities. Urinalysis shows: 8 RBCs/HPF, 12 WBCs/HPF with white cell casts, eosinophiluria, and a mild degree of proteinuria. Laboratory studies show a BUN of 40 mg/dl and serum creatinine of 2.2 mg/dl. Which of the following is the most appropriate next step in management?

A. Discontinue cephalexin
B. Start ampicillin and gentamicin
C. Start oral ciprofloxacin
D. Start intravenous steroids
E. Start oral steroids

Submit

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- A. Discontinue cephalexin [80%] ✔
- B. Start ampicillin and gentamicin [3%]
- C. Start oral ciprofloxacin [2%]
- D. Start intravenous steroids [8%]
- E. Start oral steroids [4%]

Omitted
Correct answer
A

80%
Answered correctly

2 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The patient presented in this clinical vignette is most likely suffering from drug-induced interstitial nephritis. Drug-induced interstitial nephritis occurs with many drugs such as penicillins, cephalosporins and sulfonamides. Clinical features include fever, rash and arthralgias. Other features are peripheral eosinophilia, hematuria, sterile pyuria and eosinophiluria. WBC casts may be present in the

D. Start intravenous steroids [8%]

E. Start oral steroids [4%]

Omitted
Correct answer
A

80%
Answered correctly

2 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The patient presented in this clinical vignette is most likely suffering from drug-induced interstitial nephritis. Drug-induced interstitial nephritis occurs with many drugs such as penicillins, cephalosporins and sulfonamides. Clinical features include fever, rash and arthralgias. Other features are peripheral eosinophilia, hematuria, sterile pyuria and eosinophiluria. WBC casts may be present in the urine, but red cell casts are rare. Discontinuing the offending agent is the treatment of drug-induced interstitial nephritis.

(Choice D) Steroids may hasten recovery in cases of drug-induced interstitial nephritis, but they may aggravate the underlying infection.

(Choice C) Oral ciprofloxacin or IV ampicillin and gentamicin are used to treat acute pyelonephritis.

Educational Objective:
70% of cases with interstitial nephritis are caused by drugs such as cephalosporins, penicillins, sulfonamides, sulfonamide containing diuretics, NSAIDs, rifampin, phenytoin, and allopurinol. Discontinuing the offending agent is the treatment of drug-induced interstitial nephritis.

A 45-year-old man comes to the physician for a routine check-up. He has no symptoms and says he feels perfectly healthy. He was diagnosed with hypertension and hypercholesterolemia a year ago. The patient is currently taking hydrochlorothiazide, amiloride, and simvastatin daily. He does not use tobacco, alcohol, or illicit drugs. His blood pressure is 135/85 mm Hg. Physical examination is unremarkable. Laboratory results are as follows:

Hemoglobin	14.2 g/dL
Mean corpuscular volume	86 fL
Platelet count	260,000/μL
Leukocyte count	8,500/μL
Neutrophils	70%
Eosinophils	1%
Lymphocytes	24%
Monocytes	5%
Sodium	138 mEq/L
Potassium	5.7 mEq/L
Chloride	100 mEq/L
Bicarbonate	24 mEq/L
Blood urea nitrogen	10 mg/dL
Creatinine	1.1 mg/dL

Bicarbonate	24 mEq/L
Blood urea nitrogen	10 mg/dL
Creatinine	1.1 mg/dL
Calcium	9.0 mg/dL
Blood Glucose	118 mg/dL
Total cholesterol	170 mg/dL
LDL cholesterol	90 mg/dL

The blood sample is checked and is not hemolysed. Electrocardiogram shows normal sinus rhythm without other abnormalities. Which of the following is the most appropriate next step in the management of this patient?

A. Change amiloride to amlodipine and recheck laboratory results in 1 week
B. Change hydrochlorothiazide to amlodipine and recheck laboratory results in 1 week
C. Intravenous calcium gluconate
D. Intravenous dextrose and insulin
E. Start patient on a low-potassium diet

Submit

A 45-year-old man comes to the physician for a routine check-up. He has no symptoms and says he feels perfectly healthy. He was diagnosed with hypertension and hypercholesterolemia a year ago. The patient is currently taking hydrochlorothiazide, amiloride, and simvastatin daily. He does not use tobacco, alcohol, or illicit drugs. His blood pressure is 135/85 mm Hg. Physical examination is unremarkable. Laboratory results are as follows:

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The blood sample is checked and is not hemolysed. Electrocardiogram shows normal sinus rhythm without other abnormalities. Which of the following is the most appropriate next step in the management of this patient?

- ✔ A. Change amiloride to amlodipine and recheck laboratory results in 1 week [58%]
- B. Change hydrochlorothiazide to amlodipine and recheck laboratory results in 1 week [13%]
- C. Intravenous calcium gluconate [7%]
- D. Intravenous dextrose and insulin [6%]
- E. Start patient on a low-potassium diet [13%]

Omitted
Correct answer
A

58%
Answered correctly

4 Seconds
Time Spent

07/24/2018
Last Updated

Explanation

Medications that can cause hyperkalemia	
Medication	**Mechanism**
Nonselective beta-adrenergic blockers	Inhibit beta-2–mediated intracellular potassium uptake
ACE inhibitors	Inhibit angiotensin II formation, leading to decreased aldosterone secretion
ARBs	Inhibit AT_1 receptor, leading to decreased aldosterone secretion
K^+-sparing diuretics	Inhibit ENaC or aldosterone receptor
Cardiac glycosides (eg, digoxin)	Inhibit the Na^+/K^+-ATPase pump
NSAIDs	Inhibit local prostaglandin synthesis, leading to decreased renin & aldosterone secretion

ARBs = angiotensin II receptor blockers; **AT_1** = angiotensin II type 1; **ENaC** = epithelial sodium channel; **Na^+/K^+-ATPase** = sodium/potassium adenosine triphosphatase; **NSAIDs** = nonsteroidal anti-inflammatory drugs.

This patient has hyperkalemia (K^+ >5.0 mEq/L), which is most often due to decreased urinary potassium excretion. The most common etiologies of hyperkalemia are acute or chronic kidney disease and medications or disorders that impair the renin-angiotensin axis. Other etiologies include increased potassium movement out of cells (eg, uncontrolled hyperglycemia, metabolic acidosis), increased tissue catabolism (eg, trauma, tumor lysis syndrome), or pseudohyperkalemia (eg, hemolyzed blood sample). Patients with chronic

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Initial evaluation of hyperkalemia includes ECG to evaluate for conduction abnormalities. Acute therapy (eg, calcium gluconate, insulin with glucose) is typically reserved for patients with characteristic ECG changes, potassium ≥7.0 mEq/L (with or without ECG changes), or rapidly rising potassium due to tissue breakdown. Dialysis should be reserved for patients with renal failure and those with severe life-threatening hyperkalemia unresponsive to initial therapy. This patient has a normal ECG and does not require acute therapy **(Choices C and D)**.

The next step is to exclude acute treatable secondary causes (eg, uncontrolled hyperglycemia, tumor lysis syndrome). Patients should then have a review of recent/current medications as these can frequently cause hyperkalemia. Common offending medications include nonselective beta-adrenergic blockers, potassium-sparing diuretics (eg, triamterene, amiloride), angiotensin-converting-enzyme inhibitors, angiotensin II receptor blockers, and nonsteroidal anti-inflammatory drugs. This patient takes amiloride, which should be changed to a different antihypertensive (eg, amlodipine) with follow-up testing in one week.

(Choice B) Hydrochlorothiazide helps increase renal potassium excretion and can cause hypokalemia instead of hyperkalemia.

(Choice E) Increased dietary potassium intake can cause hyperkalemia in patients with hypoaldosteronism or acute or chronic kidney disease. However, it is not a significant cause of hyperkalemia in patients with normal kidney function. As a result, a low-potassium diet is not likely to have a significant effect in this patient.

Educational objective:

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Educational objective:
The most common causes of hyperkalemia include acute or chronic kidney disease, medications, or disorders impairing the renin-angiotensin axis. Common offending medications include nonselective beta-adrenergic blockers, potassium-sparing diuretics (eg, amiloride), angiotensin-converting-enzyme inhibitors, angiotensin II receptor blockers, and nonsteroidal anti-inflammatory drugs.

References

- Management of hyperkalemia.
- Hyperkalemia.

A 56-year-old man comes to the emergency department complaining of 3 days of progressively worsening shortness of breath. He recently had a cold, and his symptoms have gradually exacerbated since then. He now has a mild productive cough but no fever or chills. The patient has a 25-pack-year smoking history and has been diagnosed with mild emphysema, type 2 diabetes mellitus, hypertension, hyperlipidemia, and hypothyroidism. He takes glipizide, lisinopril, furosemide, aspirin, atorvastatin, levothyroxine, and uses an albuterol inhaler as needed. Physical examination shows decreased breath sounds with diffuse wheezes bilaterally. There is trace bilateral lower extremity edema.

Arterial blood gas results are as follows:

pH	7.23
PaO_2	88 mm Hg
$PaCO_2$	40 mm Hg
Serum HCO_3^-	16 mEq/L

Which of the following best describes the acid-base status of this patient?

A. Mixed metabolic acidosis and respiratory alkalosis
B. Mixed metabolic and respiratory acidosis
C. Normal acid-base balance
D. Primary metabolic acidosis
E. Primary respiratory acidosis

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$PaCO_2$	40 mm Hg
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Which of the following best describes the acid-base status of this patient?

A. Mixed metabolic acidosis and respiratory alkalosis [8%]
✔ B. Mixed metabolic and respiratory acidosis [48%]
C. Normal acid-base balance [0%]
D. Primary metabolic acidosis [37%]
E. Primary respiratory acidosis [4%]

Explanation

Acid-base disorders	
Primary disorder	**Appropriate compensation**
Metabolic acidosis	$PaCO_2$ = 1.5 (serum HCO_3) + 8 ± 2
Metabolic alkalosis	↑ $PaCO_2$ by 0.7 mm Hg for every 1 mEq/L rise in serum HCO_3
Acute respiratory acidosis	↑ Serum HCO_3 by 1 mEq/L for every 10 mm Hg rise in $PaCO_2$
Acute respiratory alkalosis	↓ Serum HCO_3 by 2 mEq/L for every 10 mm Hg decrease in $PaCO_2$

The first step in assessing a patient's acid-base status is to analyze the arterial pH, which indicates the overall acid-base state. Arterial pH ≤7.35 indicates acidemia while pH ≥7.45 indicates alkalemia. This patient's arterial pH of 7.23 is consistent with acidemia. The next step is to check pCO_2 and serum HCO_3^- to determine if the cause is respiratory or metabolic. The serum HCO_3^- is low, indicating that the primary disorder is metabolic acidosis. The lungs try to compensate for the acidosis by increasing respirations to lower pCO_2 in the **same** direction as the serum HCO_3^-.

Winter's formula (arterial pCO_2 = 1.5 [HCO_3^-] + 8 ± 2) can help determine appropriate respiratory compensation. This patient's expected arterial pCO_2 would be 32 mm Hg ± 2 ([1.5 * 16 mm Hg + 8] ± 2). As a result, a pCO_2 of 30-34 mm Hg in this patient would be interpreted as primary metabolic acidosis with respiratory compensation. However, this patient's higher than expected pCO_2 (40 mm Hg) suggests that an independent primary respiratory acidosis is causing a mixed (≥2 primary disturbances) acid-base disorder. He likely has lactic acidosis (due to pneumonia). This causes metabolic acidosis and chronic obstructive pulmonary disease (COPD) exacerbation, which leads to respiratory acidosis. This patient's COPD at baseline causes chronic respiratory acidosis (pCO2 >40 mm Hg), and his body is unable to mount the required respiratory response to combat metabolic acidosis and lower pCO_2 to the expected range.

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Educational objective:
Mixed acid-base disorders refer to ≥2 primary acid-base disturbances in a patient. After identifying a primary acid-base disorder, the calculated expected pCO_2 or HCO_3^- should be compared to measured values to distinguish between appropriate compensation and a mixed disorder.

References

- A stepwise approach to acid-base disorders. Practical patient evaluation for metabolic acidosis and other conditions.
- A practical approach to acid-base disorders

A 53-year-old man comes to the office due to occasional red urine for the last 3 months. He states that his urine stream appears normal initially but turns red by the end of voiding. He has also noticed small clots in his urine. The patient has not had any fever, edema, flank pain, or weight loss. Medical history is significant for chronic back pain. He currently smokes a pack of cigarettes daily but does not use alcohol. His temperature is 37.5 C (99.5 F) and blood pressure is 160/90 mm Hg. Physical examination is within normal limits. Urinalysis is positive only for blood. Which of the following is the most likely cause of this patient's symptoms?

- A. Bladder disease
- B. Glomerular disease
- C. Nephrolithiasis
- D. Polycystic kidney disease
- E. Urethral injury
- F. Urinary tract infection

Submit

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- ✔ A. Bladder disease [79%]
- B. Glomerular disease [8%]
- C. Nephrolithiasis [3%]
- D. Polycystic kidney disease [4%]
- E. Urethral injury [4%]
- F. Urinary tract infection [0%]

Omitted
Correct answer
A

79%
Answered correctly

3 Seconds
Time Spent

07/23/2018
Last Updated

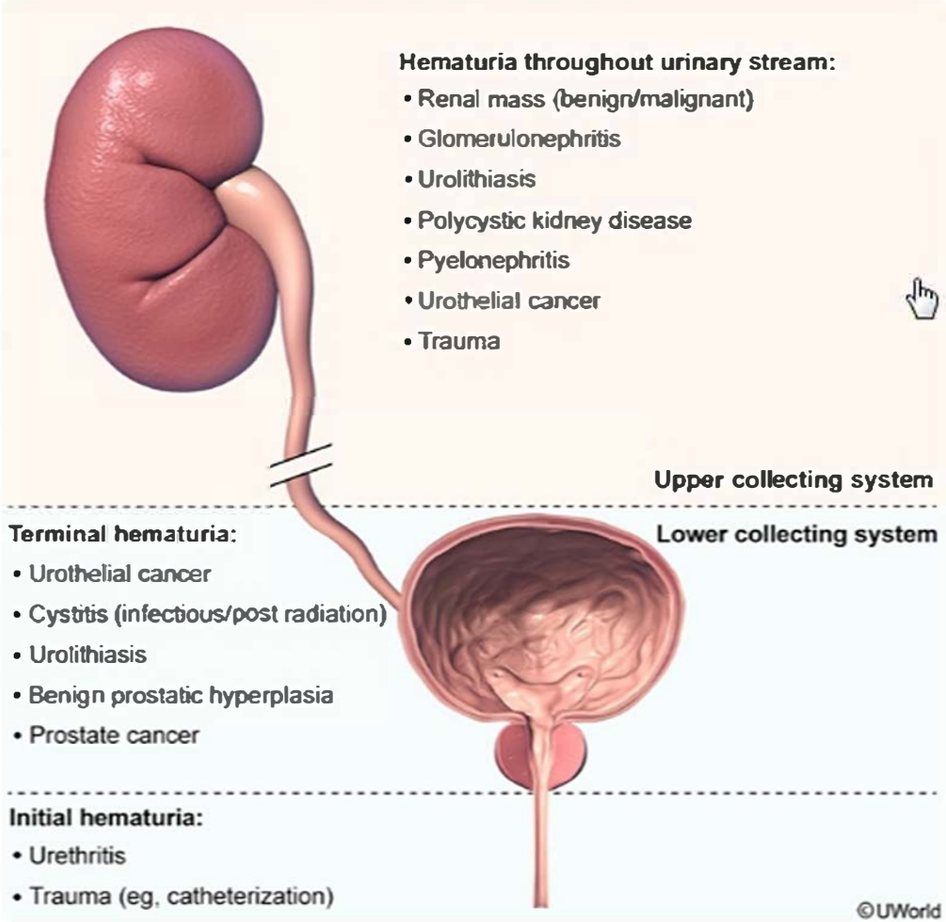
Explanation

Causes of hematuria

Explanation

Exhibit Display

Causes of hematuria



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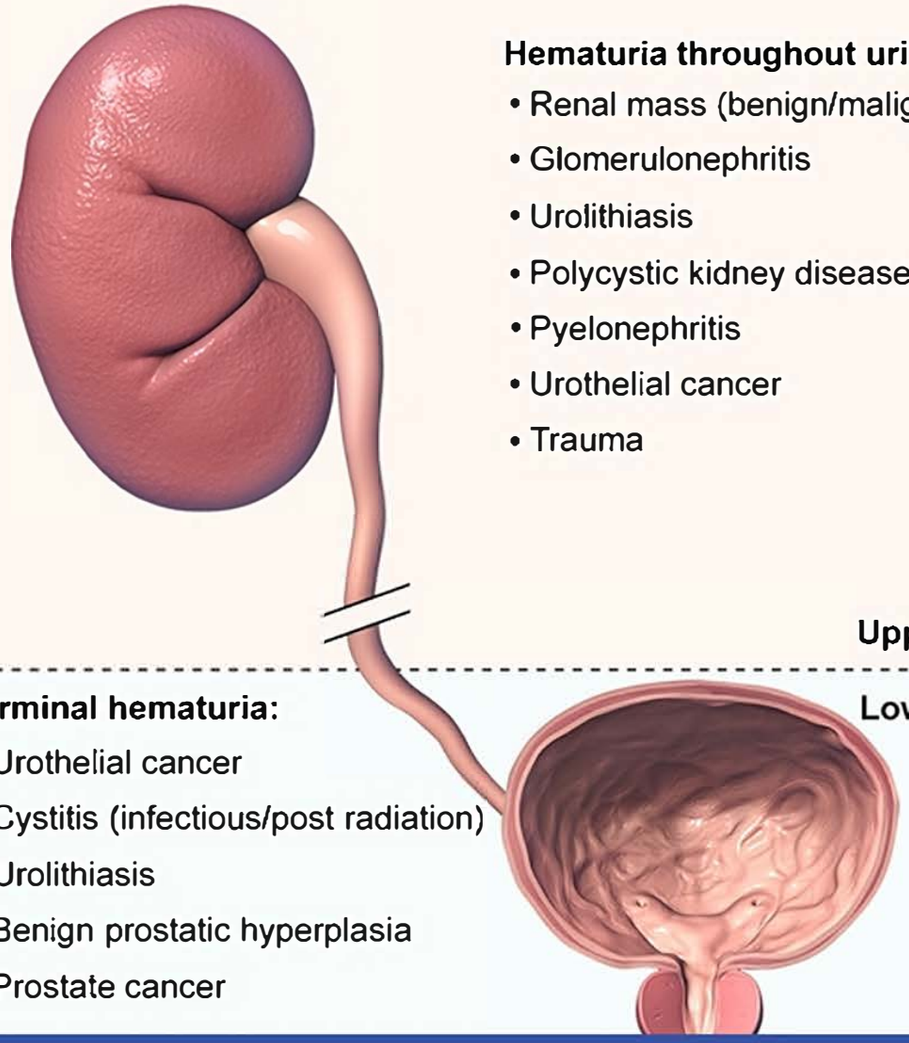
Causes of hematuria



Hematuria throughout urinary stream:

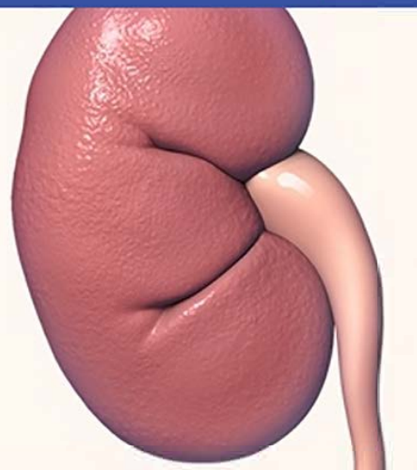
- Renal mass (benign/malignant)
- Glomerulonephritis
- Urolithiasis
- Polycystic kidney disease
- Pyelonephritis
- Urothelial cancer
- Trauma

Upper collecting system

Lower collecting system

Terminal hematuria:

- Urothelial cancer
- Cystitis (infectious/post radiation)
- Urolithiasis
- Benign prostatic hyperplasia
- Prostate cancer



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Upper collecting system

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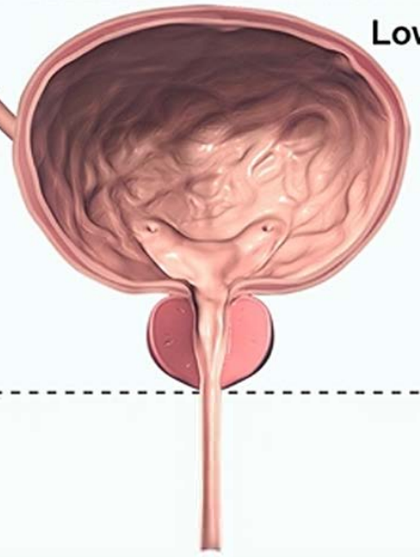
Lower collecting system



Initial hematuria:

- Urethritis
- Trauma (eg, catheterization)

Gross (ie, visible or macroscopic) **hematuria** can be classified based on the stage of voiding at which bleeding predominates:

- Initial hematuria is characterized by blood at the beginning of the voiding cycle.
- Total hematuria is characterized by blood during the entire voiding cycle.
- Terminal hematuria is characterized by blood at the end of voiding cycle.

Terminal hematuria often suggests bleeding from the prostate, bladder neck or trigone, or posterior urethra. In this patient, the presence of terminal hematuria with **clots** suggests bleeding within the bladder or ureters and is concerning for **urothelial cancer**, particularly given his risk factors of age (>40), sex, and smoking. He should be evaluated for bladder cancer by **cystoscopy**.

(Choice B) Glomerular diseases can cause nephritic syndrome with microscopic or gross hematuria. Patients can also present with total hematuria. However, clots would be unusual, and urinalysis frequently shows red blood cell casts and may show proteinuria.

(Choice C) Nephrolithiasis can cause hematuria. However, stones usually present with flank or groin pain, depending on the location of the stone.

(Choice D) Polycystic kidney disease is the leading heritable cause of renal disease in adults. However, it usually presents as abdominal or flank pain with microscopic or gross total hematuria and, occasionally, a bulky mass on abdominal examination.

(Choice E) Urethritis or urethral injury (eg, Foley catheterization) typically manifests as initial hematuria.

(Choice F) All urinary tract infections (pyelonephritis, cystitis, urethritis) may present with microscopic or gross hematuria. However, pyelonephritis usually presents with flank pain and systemic illness (eg, fever, nausea, vomiting); cystitis and urethritis present with irritative voiding symptoms (eg, dysuria, urinary frequency, hesitancy).

Educational objective:
Initial hematuria suggests urethral damage, terminal hematuria indicates bladder or prostatic damage, and total hematuria reflects damage in the kidney or ureters. Clots are not usually seen with renal causes of hematuria (eg, glomerular diseases).

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- Total hem
- Terminal

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(Choice B) G
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(Choice D) Pc
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(Choice E) Ur

(Choice F) All
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Educational o
Initial hematuri
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Exhibit Display

Indications for cystoscopy

- Gross hematuria with no evidence of glomerular disease or infection
- Microscopic hematuria with no evidence of glomerular disease or infection but increased risk for malignancy
- Recurrent urinary tract infections
- Obstructive symptoms with suspicion for stricture, stone
- Irritative symptoms without urinary infection
- Abnormal bladder imaging or urine cytology

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A 20-year-old man is found to have an elevated calcium level on pre-employment screening blood tests. He feels well and has no polyuria, polydipsia, or constipation. His medical history is unremarkable. He takes no prescription medications, although he has been consuming "protein shakes and vitamin supplements" in preparation for running a marathon. The patient does not use tobacco, alcohol, or illicit drugs. Temperature is 37 C (98.6 F), blood pressure is 110/70 mm Hg, and pulse is 82/min. Physical examination is unremarkable. Laboratory results are as follows:

Sodium	140 mEq/L
Potassium	4.0 mEq/L
Chloride	103 mEq/L
Bicarbonate	24 mEq/L
Blood urea nitrogen	18 mg/dL
Serum creatinine	0.8 mg/dL
Calcium	11.2 mg/dL
Glucose	98 mg/dL
Albumin	4.2 g/L
Serum parathyroid hormone level	65 pg/mL (normal 10-65 pg/mL)
Urine calcium/creatinine clearance ratio	<0.01

Which of the following conditions is most consistent with this patient's findings?

A. Familial hypocalciuric hypercalcemia

Blood urea nitrogen	18 mg/dL
Serum creatinine	0.8 mg/dL
Calcium	11.2 mg/dL
Glucose	98 mg/dL
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Which of the following conditions is most consistent with this patient's findings?

- A. Familial hypocalciuric hypercalcemia
- B. Multiple myeloma
- C. Primary hyperparathyroidism
- D. Renal cell carcinoma
- E. Sarcoidosis
- F. Vitamin D toxicity

Submit

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Which of the following conditions is most consistent with this patient's findings?

A. Familial hypocalciuric hypercalcemia [67%]

Serum creatinine	0.8 mg/dL
Calcium	11.2 mg/dL
Glucose	98 mg/dL
Albumin	4.2 g/L
Serum parathyroid hormone level	65 pg/mL (normal 10-65 pg/mL)
Urine calcium/creatinine clearance ratio	<0.01

Which of the following conditions is most consistent with this patient's findings?

- ✔ A. Familial hypocalciuric hypercalcemia [67%]
- B. Multiple myeloma [0%]
- C. Primary hyperparathyroidism [14%]
- D. Renal cell carcinoma [0%]
- E. Sarcoidosis [0%]
- F. Vitamin D toxicity [17%]

Omitted
Correct answer
A

67%
Answered correctly

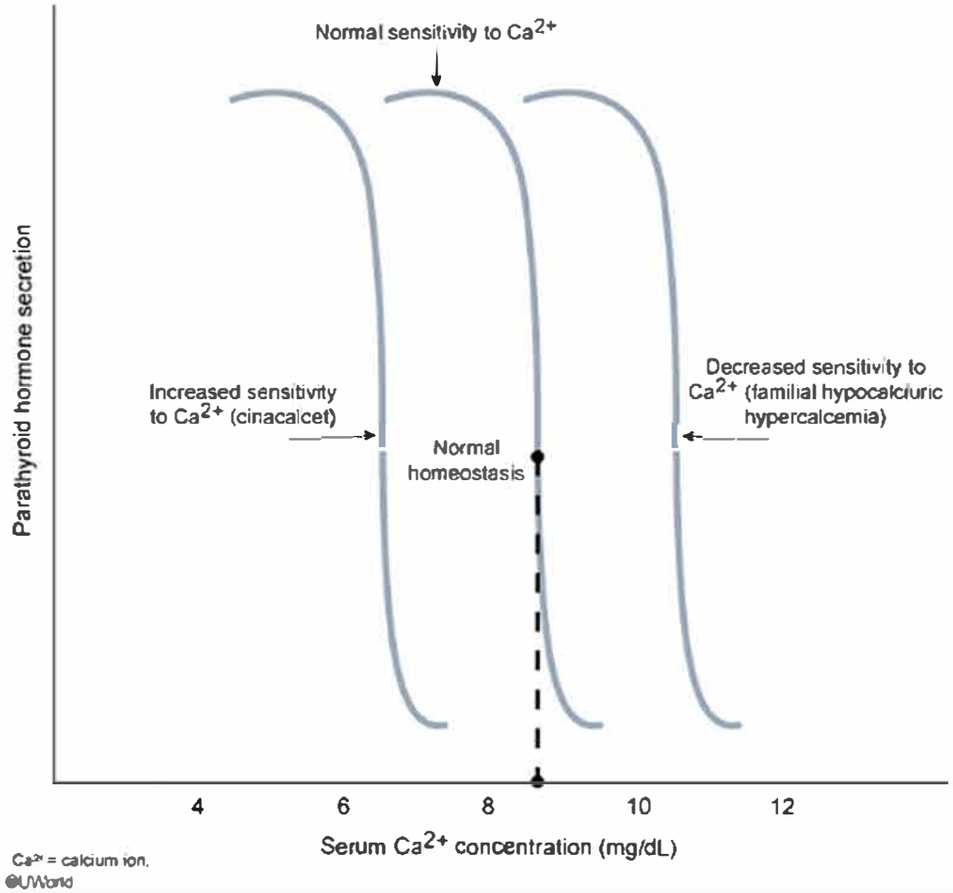
5 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

Exhibit Display

Calcium homeostasis



Ca^{2+} = calcium ion.

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This patient has mild, **asymptomatic hypercalcemia** and normal renal function. In light of a high-normal parathyroid hormone (PTH) level and low urinary calcium excretion, this presentation is consistent with **familial hypocalciuric hypercalcemia** (FHH). FHH is a benign autosomal dominant disorder caused by a mutation of the **calcium-sensing receptor (CaSR)**. Normally, high-normal calcium levels suppress PTH secretion by the parathyroid glands, but in FHH, higher calcium concentrations are required to suppress PTH release. Concurrently, the defective CaSR leads to increased reabsorption of calcium in renal tubules.

In the evaluation of hypercalcemia, an elevated (or inappropriately normal) PTH level suggests either primary hyperparathyroidism or FHH. However, patients with primary hyperparathyroidism have increased urinary calcium excretion due to excessive mobilization of calcium from bones, whereas patients with FHH usually have very low urinary calcium levels (typically <100 mg/24 hr). Urine calcium excretion can be more precisely assessed using the **urine calcium/creatinine clearance ratio** (UCCR):

$$UCCR = (Ca_{urine}/Ca_{serum})/(Creat_{urine}/Creat_{serum})$$

UCCR is usually <0.01 in FHH compared to >0.02 in primary hyperparathyroidism **(Choice C)**.

Most patients with FHH are asymptomatic, although potential complications include pancreatitis and chondrocalcinosis. In the absence of complications, no specific treatment is required.

(Choices B, D, E, and F) Patients with multiple myeloma, renal cell carcinoma, sarcoidosis, and vitamin D toxicity may also present with hypercalcemia, but PTH levels are typically suppressed due to feedback inhibition.

Educational objective:
Familial hypocalciuric hypercalcemia is caused by a mutation in the calcium-sensing receptor. It is a benign disorder characterized by asymptomatic hypercalcemia, elevated or inappropriately normal parathyroid hormone levels, and low urinary calcium excretion. It can be differentiated from primary hyperparathyroidism, which has increased urinary calcium excretion, by the urine calcium/creatinine clearance ratio.

A 5-month-old girl is brought to the physician for a weight check. The patient has been evaluated several times for poor weight gain. She takes 6 ounces of regular formula every 4 hours. Increasing the caloric density of her formula has not improved her growth. The patient has no diarrhea or vomiting. There is a family history of nephrolithiasis. She was born full term without complications. The patient's birth weight was 3.6 kg (8 lb, 50th percentile). Current weight is <5th percentile; length and head circumference have been tracking along the 25th percentile. The infant appears thin, but the remainder of the physical examination is unremarkable. Newborn screening results were normal. Laboratory results are as follows:

Serum chemistry

Sodium	140 mEq/L
Potassium	3 mEq/L
Chloride	121 mEq/L
Blood urea nitrogen	10 mg/dL
Creatinine	0.5 mg/dL
Calcium	9 mg/dL
Glucose	98 mg/dL

Arterial blood gases

pH	7.21
$PaCO_2$	31 mm Hg
Bicarbonate	14 mEq/L

Omit question?

You have not answered this question.

Do you wish to continue without answering?

YES
No

Arterial blood gases

pH	7.24
$PaCO_2$	31 mm Hg
Bicarbonate	14 mEq/L

Urinalysis

pH	7.9
Potassium	Normal
Sodium	Normal

Which of the following is the most likely cause of this patient's failure to thrive?

A. Cystic fibrosis
B. Gastroesophageal reflux
C. Insufficient caloric intake
D. Lactic acidosis
E. Renal tubular acidosis

Submit

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pH	7.21
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$PaCO_2$	31 mm Hg
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Urinalysis

pH	7.9
Potassium	Normal
Sodium	Normal

Which of the following is the most likely cause of this patient's failure to thrive?

- A. Cystic fibrosis [11%]
- B. Gastroesophageal reflux [0%]
- C. Insufficient caloric intake [3%]
- D. Lactic acidosis [10%]
- ✔ E. Renal tubular acidosis [73%]

Omitted
Correct answer
E

73%
Answered correctly

3 Seconds
Time Spent

09/26/2018
Last Updated

Explanation

Explanation

Renal tubular acidosis			
Type	**1 (Distal)**	**2 (Proximal)**	**4**
Primary defect	Poor hydrogen secretion into urine	Poor bicarbonate resorption	Aldosterone resistance
Urine pH	≥5.5	<5.5	<5.5
Serum potassium	Low-normal	Low-normal	High
Causes	• Genetic disorders • Medication toxicity • Autoimmune disorders (eg, Sjögren syndrome, rheumatoid arthritis)	Fanconi syndrome (glucosuria, phosphaturia, aminoaciduria)	• Obstructive uropathy • Congenital adrenal hyperplasia

This infant has evidence of a **normal anion gap acidosis** and **failure to thrive**. In the absence of an anion gap, a renal or gastrointestinal etiology of the acidosis is most likely. This child has no diarrhea but does have markedly alkalotic urine. These findings are suggestive of **renal tubular acidosis (RTA)**. RTA is caused by a defect in the ability of the renal tubules to reabsorb bicarbonate (type 2 RTA) or excrete hydrogen (type 1 RTA). Type 1 RTA is often a genetic disorder and is commonly associated with

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All types of RTA can present as **growth failure** (due to poor cellular growth and division in acidic conditions). Screening laboratory results will show a **low serum bicarbonate** level and hyperchloremia, which lead to a normal anion gap metabolic acidosis. Evaluation of urine pH and urine electrolytes can help distinguish between the types of RTA. Given the markedly alkalotic urine in this patient, type 1 RTA is the most likely diagnosis. Treatment consists of oral sodium bicarbonate to normalize the serum bicarbonate levels.

(Choice A) Cystic fibrosis can present with failure to thrive due to malabsorption, chronic diarrhea, and frequent sinopulmonary infections. This diagnosis is unlikely given the lack of gastrointestinal and pulmonary symptoms.

(Choice B) Gastroesophageal reflux is common in infants and can cause failure to thrive if severe. However, this infant does not have a history of spitting up or vomiting.

(Choice C) This infant is taking 36 ounces of formula a day, which is above the normal caloric intake for an infant (up to 32 ounces a day).

(Choice D) Lactic acidosis causes a high anion gap metabolic acidosis.

Educational objective:
Renal tubular acidosis is caused by a defect in either hydrogen excretion or bicarbonate resorption in the kidney. In infancy, it most commonly presents with failure to thrive due to a chronic, normal anion gap metabolic acidosis. Treatment consists of oral bicarbonate replacement.

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References

- Bicarbonate therapy improves growth in children with incomplete distal renal tubular acidosis.
- Clinical profile and outcome of renal tubular disorders in children: A single center experience.

A 5-year-old girl is brought to the office due to "wetting the bed." Since age 3, she had been urinating and stooling while on the toilet during the day and night, with no accidents. However, for the past 2 weeks, the patient has wet the bed nightly. She is also more thirsty than usual, urinates more often during the day, and has had accidents at school. Review of systems is positive for fatigue and negative for dysuria and hesitancy. The patient has had no major illnesses and has met all developmental milestones. A month ago, the family moved to a new house after her brother was born. Vital signs are normal. Weight and height are at the 20th and 40th percentile, respectively, but both were at the 40th percentile at her well-child visit 3 months ago. Physical examination shows a tired-appearing girl. Mucous membranes are dry. The rest of the examination is unremarkable. Which of the following is the most likely explanation for this patient's symptoms?

- A. Autoimmune destruction of pancreatic beta cells
- B. Bacterial infection of the bladder
- C. Behavior regression from changes in the home
- D. Functional fecal retention
- E. Impaired renal tubule response to antidiuretic hormone

Submit

A 5-year-old girl is brought to the office due to "wetting the bed." Since age 3, she had been urinating and stooling while on the toilet during the day and night, with no accidents. However, for the past 2 weeks, the patient has wet the bed nightly. She is also more thirsty than usual, urinates more often during the day, and has had accidents at school. Review of systems is positive for fatigue and negative for dysuria and hesitancy. The patient has had no major illnesses and has met all developmental milestones. A month ago, the family moved to a new house after her brother was born. Vital signs are normal. Weight and height are at the 20th and 40th percentile, respectively, but both were at the 40th percentile at her well-child visit 3 months ago. Physical examination shows a tired-appearing girl. Mucous membranes are dry. The rest of the examination is unremarkable. Which of the following is the most likely explanation for this patient's symptoms?

- ✔ A. Autoimmune destruction of pancreatic beta cells [75%]
- B. Bacterial infection of the bladder [0%]
- C. Behavior regression from changes in the home [14%]
- D. Functional fecal retention [0%]
- E. Impaired renal tubule response to antidiuretic hormone [7%]

Omitted
Correct answer
A

75%
Answered correctly

3 Seconds
Time Spent

09/25/2018
Last Updated

Explanation

Explanation

Type 1 diabetes mellitus	

Onset	• Bimodal distribution
	  ◦ Age 4-6
	  ◦ Age 10-14

Pathophysiology	• Destruction of pancreatic beta islet cells
Clinical manifestations	• Polydipsia • Polyuria, nocturia, enuresis • Weight loss • Fatigue • Blurred vision
Treatment	• Insulin replacement

Enuresis is urinary incontinence in children age ≥5. Children who have never achieved dryness have primary enuresis. **Secondary enuresis** refers to incontinence after ≥6 months of dryness. As shown in this case, secondary enuresis is often pathologic and requires evaluation.

In addition to day and night incontinence, this patient has **polyuria**, **polydipsia**, and poor weight gain, findings suggestive of **type 1 diabetes mellitus (DM)**. Type 1 DM, which results in insulin deficiency from autoimmune destruction of pancreatic beta cells, has a bimodal onset: Patients typically start having symptoms at age 4-6 or at early puberty. When hyperglycemia exceeds the renal threshold for glucose, the resultant glucosuria leads to osmotic diuresis and dehydration (eg, dry mucous membranes). **Weight loss**

In addition to day and night incontinence, this patient has **polyuria**, **polydipsia**, and poor weight gain, findings suggestive of **type 1 diabetes mellitus (DM)**. Type 1 DM, which results in insulin deficiency from autoimmune destruction of pancreatic beta cells, has a bimodal onset: Patients typically start having symptoms at age 4-6 or at early puberty. When hyperglycemia exceeds the renal threshold for glucose, the resultant glucosuria leads to osmotic diuresis and dehydration (eg, dry mucous membranes). **Weight loss** and **fatigue** are additional common presenting symptoms.

Diagnosis is confirmed by elevated serum glucose or the presence of glucose in the urine. When euglycemia is achieved with insulin therapy, glucosuria and, in turn, polyuria and enuresis, resolve.

(Choice B) Urinary tract infections can cause enuresis. However, most patients also have dysuria, hesitancy, and urgency, making this diagnosis less likely.

(Choices C and D) Psychologic stress (eg, new home, birth of a sibling, parental divorce) can cause behavioral regression, including secondary enuresis. In addition, functional constipation causing stool retention in the distal bowel can lead to decreased bladder capacity and enuresis. Both causes should be investigated in a child with enuresis. However, fatigue, polydypsia, signs of dehydration, and weight loss would not be expected, so type 1 DM is more likely in this case.

(Choice E) Nephrogenic diabetes insipidus, an impaired renal response to antidiuretic hormone, presents with polydipsia and excessive excretion of dilute urine. In contrast to type 1 DM, diabetes insipidus is rare in children and far less likely to cause this patient's symptoms.

Educational objective:
Enuresis in the setting of polyuria, polydipsia, and weight loss is suggestive of new-onset type 1 diabetes mellitus.

References

- Diabetes update: primary care of patients with type 1 diabetes.
- Enuresis in children: a case based approach.

A 31-year-old motorcyclist is involved in a motor vehicle collision in which he suffered a direct blow to his lower abdomen and pelvis. He reports diffuse abdominal pain and a dull pain in the left shoulder. The patient has no prior medical problems and has been healthy. Blood pressure is 110/80 mm Hg, pulse is 92/min, and respirations are 16/min. No deformity of the left shoulder is noted and complete range of motion is preserved. Cardiopulmonary examination shows no abnormalities. Diffuse abdominal tenderness with guarding is present. No other injuries are seen. Which one of the following injuries is most likely to be seen on CT scan of the abdomen in this patient?

- A. Anterior bladder wall rupture
- B. Bladder dome rupture
- C. Bladder neck rupture
- D. Renal laceration
- E. Transection of anterior urethra
- F. Transection of membranous urethra

Submit

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- A. Anterior bladder wall rupture [14%]
- ✔ B. Bladder dome rupture [46%]
- C. Bladder neck rupture [8%]
- D. Renal laceration [16%]
- E. Transection of anterior urethra [3%]
- F. Transection of membranous urethra [10%]

Omitted
Correct answer
B

46%
Answered correctly

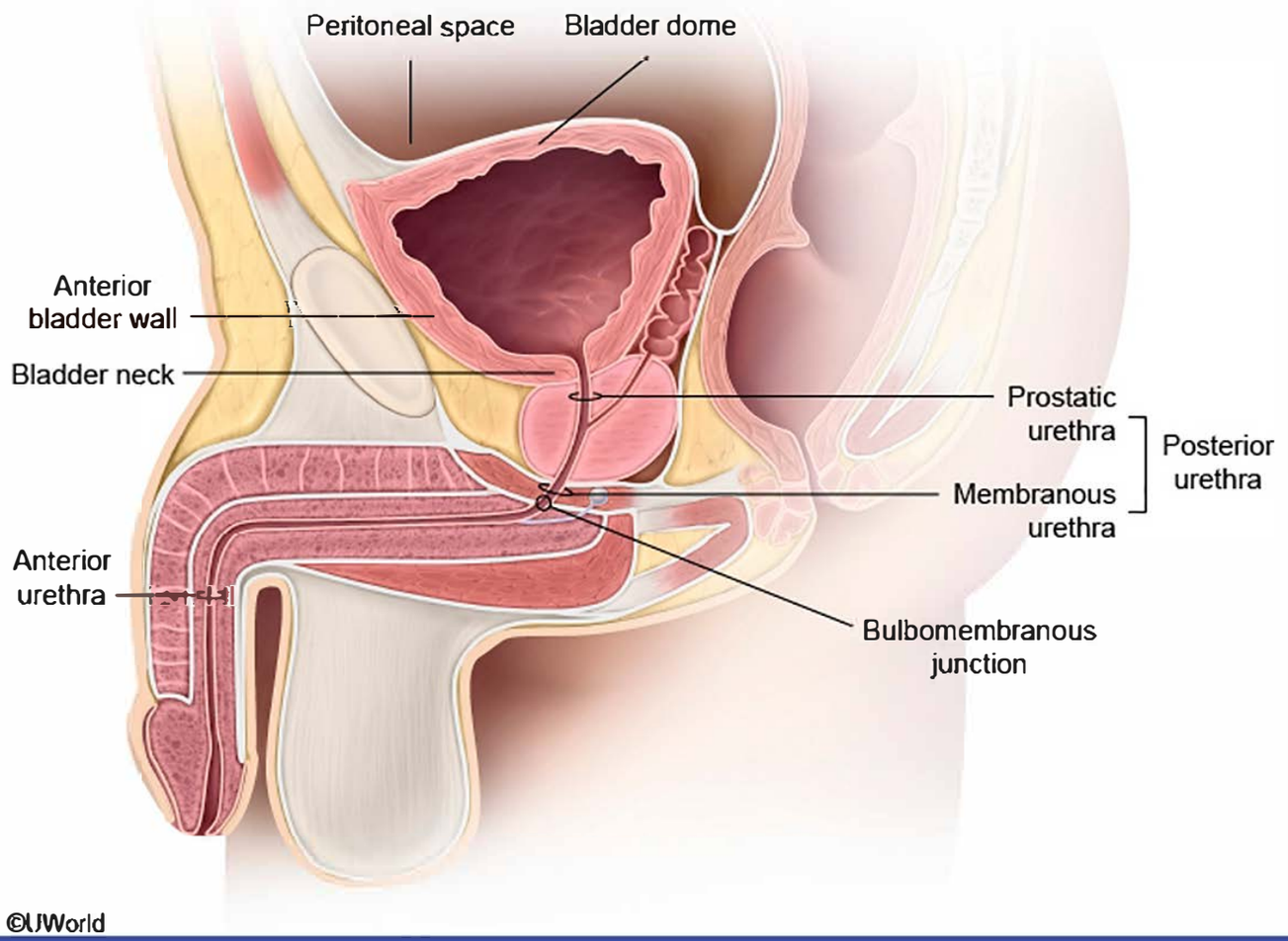
3 Seconds
Time Spent

09/23/2018
Last Updated

Explanation

Explanation

Male urogenital anatomy



In the setting of blunt abdominal trauma, spillage of blood, bowel contents, bile, pancreatic secretions, or urine into the **peritoneal cavity** can cause **acute chemical peritonitis**, which is evidenced by **diffuse abdominal pain** and guarding. The superior and lateral surfaces of the bladder compose the dome of the bladder and are bordered by the peritoneal cavity. Therefore, **rupture of the dome of the bladder** causes urine to spill into the peritoneum, leading to peritonitis. Bladder rupture after blunt trauma is due to a sudden increase in intravesical pressure and most likely occurs following a blow to the lower abdomen when the bladder is full and distended.

In addition, irritation of the peritoneal lining of the right or left hemidiaphragm may cause **referred pain** to the ipsilateral shoulder (Kehr sign) as sensory innervation to the shoulder originates from the **C3 to C5 spinal roots**; these roots are also the origin of the **phrenic nerve** innervating the **diaphragm**.

(Choices A and C) The anterior bladder wall and the bladder neck are extraperitoneal structures. A tear in these locations is almost always accompanied by pelvic fracture and causes extraperitoneal leakage of urine, leading to localized lower abdominal pain. Signs of peritonitis should not be present.

(Choice D) The kidney is a retroperitoneal structure and laceration would cause bleeding into the retroperitoneal space. Patients with this injury typically have flank pain and hematuria.

(Choices E and F) The bulbomembranous junction (junction of the anterior and posterior urethra) is the most common site of urethral injury. However, the urethra is an extraperitoneal structure, so its injury does not lead to peritonitis. In posterior urethral injury (eg, membranous urethral injury) and bulbomembranous transection, digital rectal examination may reveal a high-riding prostate. In the case of anterior urethral injury, penile trauma (eg, laceration, contusion) is often visible.

Educational objective:
Rupture of the dome of the bladder causes urine to leak into the peritoneal cavity and can lead to chemical peritonitis. Intraabdominal pathology causing pain in one or both shoulders suggests referred pain due to subdiaphragmatic peritonitis.

References

A 68-year-old man with a history of type 2 diabetes mellitus and coronary artery disease (status post triple-vessel coronary artery bypass surgery 5 years ago) undergoes a left total knee replacement for significant degenerative joint disease. He has an uncomplicated perioperative course and is undergoing physical therapy starting on postoperative day 3. On postoperative day 5, the patient complains of new-onset abdominal discomfort. Over the past 12 hours, he has spontaneously voided 200 mL of urine. Review of the medical chart shows infrequent recording of fluid input/output over the previous 4 days. His temperature is 37.0° C (98.6° F), blood pressure is 110/70 mm Hg, pulse is 80/min, and respirations are 14/min. His body mass index is 40 kg/m². Abdominal examination shows diffuse discomfort and difficulty in appreciating masses due to obesity. Portable bladder scan is inconclusive. Laboratory results are as follows:

Hemoglobin	12.5 g/dL
Platelets	170,000/μL
Leukocytes	9,700/μL
Serum sodium	130 mEq/L
Serum potassium	5.0 mEq/L
Chloride	101 mEq/L
Bicarbonate	21 mEq/L
Blood urea nitrogen	70 mg/dL
Serum creatinine	3.5 mg/dL
Calcium	9.6 mg/dL

His renal function was normal prior to the surgery. Which of the following is the most appropriate next step in management of this

Serum potassium	5.0 mEq/L
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His renal function was normal prior to the surgery. Which of the following is the most appropriate next step in management of this patient?

- A. Bladder catheterization
- B. Computed tomography angiography of the abdomen
- C. Immediate hemodialysis
- D. Intravenous fluid bolus
- E. Intravenous loop diuretic
- F. Urine culture
- G. Urine sediment analysis

Submit

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Calcium	9.6 mg/dL

His renal function was normal prior to the surgery. Which of the following is the most appropriate next step in management of this patient?

- A. Bladder catheterization [54%]
- B. Computed tomography angiography of the abdomen [5%]
- C. Immediate hemodialysis [5%]
- D. Intravenous fluid bolus [27%]
- E. Intravenous loop diuretic [1%]
- F. Urine culture [1%]
- G. Urine sediment analysis [4%]

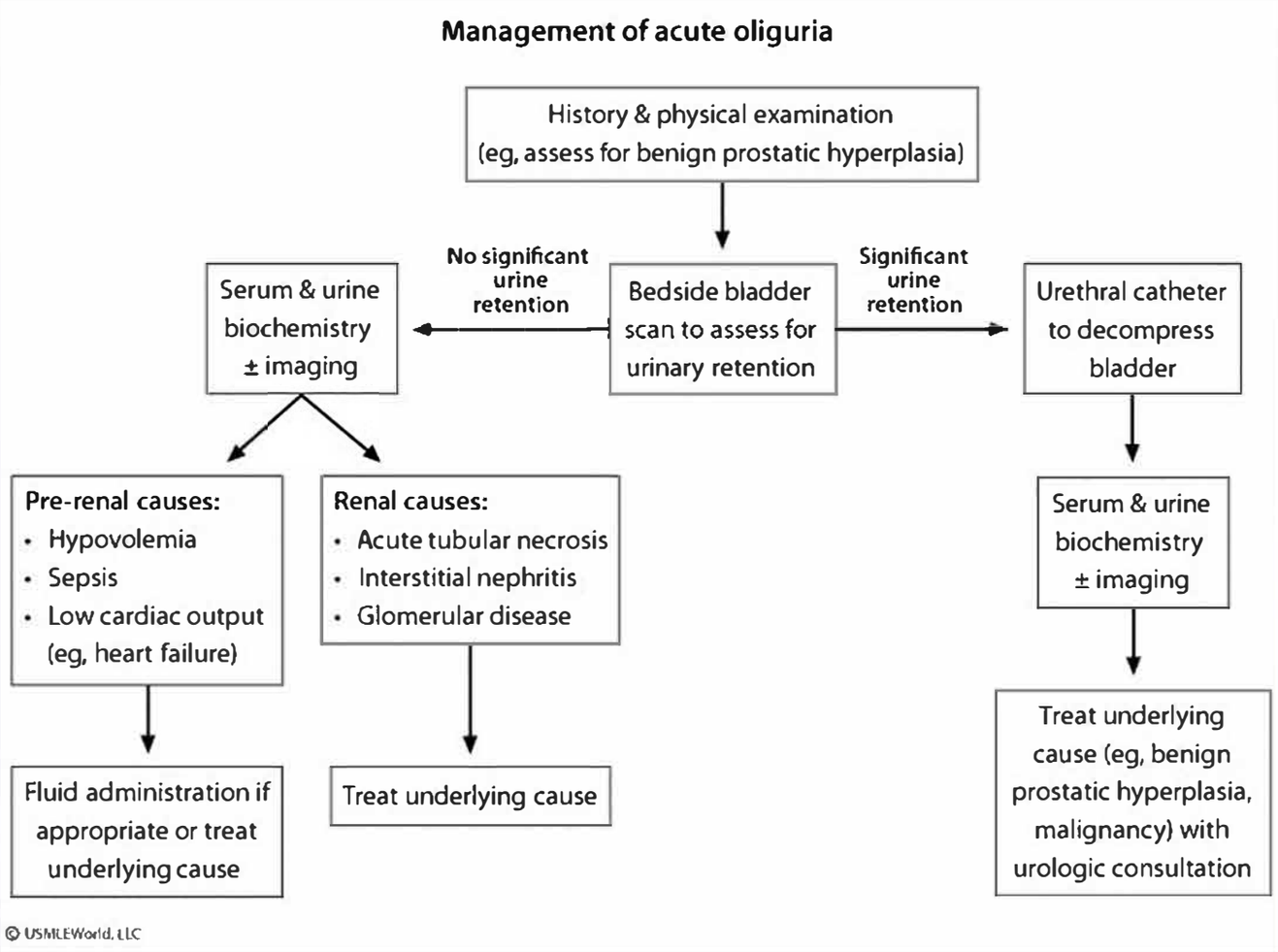
Omitted
Correct answer
A

54%
Answered correctly

5 Seconds
Time Spent

10/14/2018
Last Updated

Explanation



This patient presents with acute abdominal pain, oliguria (<250 mL urine in 12 hours), increased blood urea nitrogen, and increased

This patient presents with acute abdominal pain, oliguria (<250 mL urine in 12 hours), increased blood urea nitrogen, and increased serum creatinine in the postoperative setting. Acute kidney injury (AKI) with oliguria can be due to pre-renal, intrinsic renal disease, or post-renal causes. Postoperative urinary retention (PUR) is a common complication of surgery and anesthesia. A precipitating event (eg, bladder distention during general anesthesia, epidural anesthesia use) can lead to inefficient detrusor muscle activity and acute urinary retention (the most likely cause in this patient). The risk of PUR also increases with advancing age, high fluid intake during surgery, and concomitant use of other medications (eg, opiates, anticholinergics).

Postoperative oliguria (≤ 0.5 mL/kg/hr) requires immediate assessment with initial portable bladder scan (if available) to assess bladder volume. Patients with significant urinary retention and likely distal obstruction require Foley catheterization to restore normal urine output and resolve or prevent hydronephrosis, tubular atrophy, and renal injury. If catheterization does not relieve the patient's oliguria or if there is no significant urinary retention, the patient's AKI may be due to other etiologies (ie, intrinsic, pre-renal). This patient recently had surgery, and the oliguria and abdominal discomfort suggest urinary retention. Because the portable bladder scan is inconclusive (due to obesity), bladder catheterization is the appropriate next step.

(Choice B) Computed tomography (CT) angiography of the abdomen is useful for diagnosing renovascular disease due to atherosclerosis (unilateral or bilateral renal artery stenosis). Progressive renal vascular disease can cause chronic kidney disease but is an unlikely cause of acute renal failure. Moreover, the use of intravenous contrast for CT angiography is contraindicated in acute renal failure.

(Choice C) Urgent hemodialysis is indicated in patients with severe acidemia, hyperkalemia, volume overload, certain drug toxicities and overdoses, or uremic encephalopathy.

(Choice D) Intravenous fluids are critical for treating pre-renal AKI due to hypovolemia. However, administering fluids without first placing a bladder catheter in patients with suspected post-renal obstruction would further increase the strain on the kidneys and bladder and potentially worsen symptoms.

(Choice E) Patients with heart failure can have volume overload but low cardiac output (ie, cardiorenal syndrome). Renal perfusion

is an unlikely cause of acute renal failure. Moreover, the use of intravenous contrast for CT angiography is contraindicated in acute renal failure.

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(Choice E) Patients with heart failure can have volume overload but low cardiac output (ie, cardiorenal syndrome). Renal perfusion in these patients can be improved with intravenous loop diuretics. They should not be used in those with oliguria due to suspected bladder outlet obstruction.

(Choices F and G) Urine sediment analysis is helpful for differentiating between pre-renal and intrinsic renal disease. Muddy brown casts are present in acute tubular necrosis; urine eosinophils suggest acute interstitial nephritis. Urinary tract infections and/or pyelonephritis would be suggested by a positive urine culture. However, this patient's presentation is more concerning for urinary retention and requires bladder catheterization before other etiologies are considered.

Educational objective:
Urgent bladder scan and catheterization should be performed in all patients with oliguria and acute renal failure due to suspected bladder outlet obstruction in the postoperative setting. Placement of bladder catheter in a timely fashion can rapidly improve symptoms, reverse acute renal failure, and prevent long-term renal damage.

References

- Postoperative urinary retention.

A 36-year-old woman is hospitalized following a vaginal hysterectomy for symptomatic uterine fibroids. Medical history is otherwise unremarkable, and preoperative laboratory results were normal. The patient tolerated the procedure well but was transferred to an observation unit for additional monitoring overnight due to postoperative pain and mild residual sedation. Medications at the time of transfer included morphine sulfate by patient-controlled analgesia and an intravenous infusion of 5% dextrose with 0.45% saline. The next morning the patient is confused and reports headache, nausea, and vomiting. Temperature is 36.7 C (98.1 F), blood pressure is 110/70 mm Hg, pulse is 88/min, and respirations are 14/min. Neurologic examination shows moderate lethargy but no focal abnormalities. Laboratory results are as follows:

Serum chemistry

Sodium	119 mEq/L
Potassium	3.7 mEq/L
Chloride	95 mEq/L
Urea nitrogen	8 mg/dL
Creatinine	0.5 mg/dL
Glucose	78 mg/dL

Which of the following is the most appropriate treatment for this patient's acute condition?

A. Administer intravenous dexamethasone
B. Administer intravenous sodium bicarbonate
C. Change fluids to hypertonic (3%) saline
D. Change fluids to isotonic (0.9%) saline

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E. Change fluids to lactated Ringer solution

Submit

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Which of the following is the most appropriate treatment for this patient's acute condition?

A. Administer intravenous dexamethasone [2%]
B. Administer intravenous sodium bicarbonate [1%]
✔ C. Change fluids to hypertonic (3%) saline [48%]
D. Change fluids to isotonic (0.9%) saline [44%]

Serum chemistry

Sodium	119 mEq/L
Potassium	3.7 mEq/L
Chloride	95 mEq/L
Urea nitrogen	8 mg/dL
Creatinine	0.5 mg/dL
Glucose	78 mg/dL

Which of the following is the most appropriate treatment for this patient's acute condition?

A. Administer intravenous dexamethasone [2%]
B. Administer intravenous sodium bicarbonate [1%]
✔ C. Change fluids to hypertonic (3%) saline [48%]
D. Change fluids to isotonic (0.9%) saline [44%]
E. Change fluids to lactated Ringer solution [3%]

Omitted
Correct answer
C

48%
Answered correctly

5 Seconds
Time Spent

10/18/2018
Last Updated

Explanation

Iatrogenic hyponatremia	
Risk factors	• Hypotonic fluid hydration • Children, premenopausal women, elderly • Hypoxia • Central nervous system disorders
Clinical presentation	• Headache • Nausea/vomiting • Encephalopathy (eg, mental status changes, seizure)
Management	• Hypertonic (3%) saline • Serial measurement of electrolytes • Increase serum sodium 6-8 mEq/L in first 24 hrs

This patient has acute symptomatic hyponatremia with headache and lethargy, likely due to **hyponatremic encephalopathy**. Iatrogenic hyponatremia is a common complication of hypotonic fluids such as 0.45% normal saline (and in this patient, pain and morphine, both of which are associated with the syndrome of inappropriate antidiuretic hormone secretion, likely exacerbated the hyponatremia). The risk of iatrogenic hyponatremia is increased in premenopausal women, the elderly, children, and patients with hypoxia or central nervous system disorders. Characteristic symptoms include headache, nausea/vomiting, weakness, seizures, and mental status changes. Untreated patients may develop cerebral edema and brain herniation. Acute hyponatremic encephalopathy is a medical emergency and should be treated with **hypertonic (3%) saline** with close monitoring of electrolytes.

To reduce the risk of hyponatremia, patients who do not have specific indications for hypotonic fluids should routinely be given isotonic

mental status changes. Untreated patients may develop cerebral edema and brain herniation. Acute hyponatremic encephalopathy is a medical emergency and should be treated with **hypertonic (3%) saline** with close monitoring of electrolytes.

To reduce the risk of hyponatremia, patients who do not have specific indications for hypotonic fluids should routinely be given isotonic (0.9%) saline for maintenance hydration. Dextrose (5%) may be added; although it increases the osmolarity of the solution, it does not significantly affect tonicity as the sugar is rapidly taken up by cells.

(Choice A) Acute adrenal insufficiency (adrenal crisis) is often seen in a postoperative setting and requires rapid administration of hydrocortisone or dexamethasone. It can cause nausea, weakness, confusion, and hyponatremia, but most patients have severe, refractory hypotension; this patient is normotensive.

(Choice B) Sodium bicarbonate is indicated for treatment of salicylate toxicity, tricyclic antidepressant overdose, severe metabolic acidosis (eg, renal failure), and hyperkalemia. This patient's clinical features are attributable to hyponatremia; therefore, bicarbonate is not needed.

(Choices D and E) Patients with acute severe/symptomatic hyponatremia require rapid correction of sodium to prevent further deterioration in mental status; this cannot be accomplished with isotonic solutions (eg, 0.9% saline). Balanced salt solutions (eg, Ringer solution) are isotonic and are not appropriate for treatment of symptomatic hyponatremia; they can be used for maintenance hydration but are not superior to saline for most patients.

Educational objective:
Iatrogenic hyponatremia is a common complication of hypotonic fluids. Symptoms include headache, nausea/vomiting, weakness, seizures, and mental status changes. Acute hyponatremic encephalopathy should be treated with hypertonic (3%) saline with close monitoring of electrolytes.

References

- Maintenance intravenous fluids in acutely ill patients.

A 57-year-old man comes to the physician for 2 episodes of blood in his urine. He also complains of fatigue and fever for the last 4 weeks. He has no other medical problems and takes no medications. The patient has a 50-pack-year smoking history but does not use alcohol or illicit drugs. His father died from a blood disorder, but the patient is unsure of the specific name. Vital signs are within normal limits. Examination shows a left-sided varicocele that fails to empty when the patient is recumbent. The remainder of the examination shows no abnormalities.

Laboratory results are as follows:

Hemoglobin	18.0 g/dL
WBCs	7,400/μL
Platelets	580,000/μL
Urinalysis	>10 RBCs/hpf

Which of the following is the most appropriate diagnostic procedure?

A. Chest x-ray
B. Abdominal CT scan
C. Urine cytology
D. Serum alpha-fetoprotein levels
E. Ultrasound of the testicles
F. Bone marrow biopsy

A 57-year-old man comes to the physician for 2 episodes of blood in his urine. He also complains of fatigue and fever for the last 4 weeks. He has no other medical problems and takes no medications. The patient has a 50-pack-year smoking history but does not use alcohol or illicit drugs. His father died from a blood disorder, but the patient is unsure of the specific name. Vital signs are within normal limits. Examination shows a left-sided varicocele that fails to empty when the patient is recumbent. The remainder of the examination shows no abnormalities.

Laboratory results are as follows:

Hemoglobin	18.0 g/dL
WBCs	7,400/μL
Platelets	580,000/μL
Urinalysis	>10 RBCs/hpf

Which of the following is the most appropriate diagnostic procedure?

- A. Chest x-ray [1%]
- ✔ B. Abdominal CT scan [48%]
- C. Urine cytology [13%]
- D. Serum alpha-fetoprotein levels [3%]
- E. Ultrasound of the testicles [13%]
- F. Bone marrow biopsy [19%]

Omitted
Correct answer
B

48%
Answered correctly

6 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Renal cell carcinoma

- Flank pain, hematuria & a palpable abdominal renal mass
- Scrotal varicoceles (left-sided)
- Paraneoplastic symptoms
  - Anemia or erythrocytosis
  - Thrombocytosis
  - Fever
  - Hypercalcemia
  - Cachexia

This patient most likely has renal cell carcinoma (RCC). Most RCC patients are asymptomatic until the disease is advanced. The classic triad of RCC (flank pain, hematuria, and a palpable abdominal renal mass) is found in only 10% of patients; when present, it strongly suggests advanced/metastatic disease. Hematuria is seen in about 40% of patients and signifies tumor invasion of the collecting system. Scrotal varicoceles (most are left-sided) are observed in about 10% of patients. Varicoceles typically fail to empty when the patient is recumbent due to tumor obstruction of the gonadal vein where it enters the renal vein. Presence of this finding should always raise suspicion for mass obstruction to venous flow, as is seen in RCC.

This patient most likely has renal cell carcinoma (RCC). Most RCC patients are asymptomatic until the disease is advanced. The classic triad of RCC (flank pain, hematuria, and a palpable abdominal renal mass) is found in only 10% of patients; when present, it strongly suggests advanced/metastatic disease. Hematuria is seen in about 40% of patients and signifies tumor invasion of the collecting system. Scrotal varicoceles (most are left-sided) are observed in about 10% of patients. Varicoceles typically fail to empty when the patient is recumbent due to tumor obstruction of the gonadal vein where it enters the renal vein. Presence of this finding should always raise suspicion for mass obstruction to venous flow, as is seen in RCC.

Twenty percent of patients may also have constitutional symptoms such as fever, night sweats, anorexia, weight loss, or easy fatigability. Ectopic production of erythropoietin by the tumor can produce polycythemia, although most advanced tumors are associated with anemia. CT scan of the abdomen is the most sensitive and specific test for diagnosing RCC and should be obtained when the index of suspicion is high.

(Choice A) Chest x-ray is important to check for metastasis but will not indicate the diagnosis in this patient.

(Choice C) The combination of fever, scrotal varicocele, and polycythemia would virtually exclude bladder cancer, although this patient is at risk due to his smoking history and presence of hematuria. Urine cytology is not helpful in diagnosing RCC.

(Choices D & E) This patient's findings are unlikely to indicate testicular carcinoma; therefore, an ultrasound of the testicles or serum alpha-fetoprotein measurements would not be helpful.

(Choice F) Erythrocytosis and thrombocytosis are seen as paraneoplastic manifestations of RCC. Bone marrow biopsy is not indicated at this time.

Educational objective:
Unilateral varicoceles that fail to empty when a patient is recumbent raise suspicion for an underlying mass pathology, such as renal cell carcinoma (RCC), that obstructs venous flow. CT scan of the abdomen is the most sensitive and specific test for diagnosing RCC.

References

A 72-year-old man presents to the clinic for routine health maintenance. He has been feeling well recently, and has no specific complaints. His medical history is significant for hypertension and hyperlipidemia. His current medications are hydrochlorothiazide, felodipine, and simvastatin. He has a 40-pack-year smoking history, but quit 5 years ago. He drinks alcohol occasionally. His brother was recently diagnosed with bladder cancer. Physical examination reveals normal findings. The patient wants to know whether he should be screened for bladder cancer. Which of the following is the most appropriate statement for his question?

A. You do not require any screening for bladder cancer
B. You need screening because of your smoking history
C. You need screening because of your family history
D. You need screening because of your family and smoking histories
E. You need screening because of your age

Submit

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- ✔ A. You do not require any screening for bladder cancer [58%]
- B. You need screening because of your smoking history [14%]
- C. You need screening because of your family history [1%]
- D. You need screening because of your family and smoking histories [23%]
- E. You need screening because of your age [1%]

Omitted
Correct answer
A

58%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Bladder cancer is the second most common urologic cancer with approximately 56,000 new cases per year. It is more common in men than women. The average age of diagnosis is 65 years. Cigarette smoking and exposure to industrial chemicals account for 50% and 15% of new cases per year, respectively.

D. You need screening because of your family and smoking histories [23%]

E. You need screening because of your age [1%]

Omitted
Correct answer
A

58%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Bladder cancer is the second most common urologic cancer with approximately 56,000 new cases per year. It is more common in men than women. The average age of diagnosis is 65 years. Cigarette smoking and exposure to industrial chemicals account for 60% and 15% of new cases per year, respectively.

The United States Preventive Services Task Force (USPSTF) recommends against screening for bladder cancer due to its relatively low incidence and poor positive predictive value of the current screening tests (e.g., urine analysis and cytology, bladder tumor antigen, nuclear matrix protein). The USPSTF did not issue a statement regarding screening of people with occupational exposures.

(Choices B, C, D and E) There are no current recommendations for bladder cancer screening despite advanced age or significant smoking and family histories.

Educational objective:
Screening for bladder cancer is not recommended, even in patients who are at risk of developing the disease.

A 42-year-old man is brought to the emergency department immediately after having a seizure. His family describes a medical history of generalized tonic-clonic seizures. The patient has been on valproic acid for the past 10 years but stopped taking the drug 6 months ago as he had no seizures in the last 9 years. He is otherwise healthy and had been doing well until this seizure episode. He does not use tobacco, alcohol, or illicit drugs. The patient is afebrile. Blood pressure is 105/68 mm Hg, pulse is 96/min, and respirations are 18/min. Pulse oximetry shows 99% on room air. The patient appears confused and lethargic. Chest auscultation is unremarkable, and the abdomen is soft and nontender. A limited neurologic examination is nonfocal. Laboratory results are as follows:

Serum chemistry	
Sodium	140 mEq/L
Potassium	4.0 mEq/L
Chloride	103 mEq/L
Bicarbonate	17 mEq/L
Blood urea nitrogen	20 mg/dL
Creatinine	0.8 mg/dL
Glucose	98 mg/dL

Arterial blood gas shows pH 7.24. Chest x-ray and urinalysis are within normal limits, and a CT scan of the head is unremarkable. Which of the following is the most appropriate next step in management of this patient's metabolic acidosis?

A. Administer intravenous bicarbonate

B. Check serum amylase

Sodium	140 mEq/L
Potassium	4.0 mEq/L
Chloride	103 mEq/L
Bicarbonate	17 mEq/L
Blood urea nitrogen	20 mg/dL
Creatinine	0.8 mg/dL
Glucose	98 mg/dL

Arterial blood gas shows pH 7.24. Chest x-ray and urinalysis are within normal limits, and a CT scan of the head is unremarkable.
Which of the following is the most appropriate next step in management of this patient's metabolic acidosis?

- A. Administer intravenous bicarbonate
- B. Check serum amylase
- C. Check serum ketones
- D. Observe and repeat the laboratory tests after 2 hours
- E. Obtain a serum ammonia level
- F. Start a norepinephrine infusion

Submit

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Serum chemistry

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Arterial blood gas shows pH 7.24. Chest x-ray and urinalysis are within normal limits, and a CT scan of the head is unremarkable. Which of the following is the most appropriate next step in management of this patient's metabolic acidosis?

- A. Administer intravenous bicarbonate [33%]
- B. Check serum amylase [0%]
- C. Check serum ketones [10%]
- ✔ D. Observe and repeat the laboratory tests after 2 hours [42%]
- E. Obtain a serum ammonia level [11%]

Chloride	103 mEq/L
Bicarbonate	17 mEq/L
Blood urea nitrogen	20 mg/dL
Creatinine	0.8 mg/dL
Glucose	98 mg/dL

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- ✔ D. Observe and repeat the laboratory tests after 2 hours [42%]
- E. Obtain a serum ammonia level [11%]
- F. Start a norepinephrine infusion [0%]

Omitted
Correct answer
D

42%
Answered correctly

5 Seconds
Time Spent

11/26/2018
Last Updated

Explanation

This patient has **anion gap metabolic acidosis** that is likely due to **postictal lactic acidosis**. Seizure activity, especially a **tonic-clonic seizure**, can significantly raise serum lactic acid levels due to **skeletal muscle hypoxia** and impaired hepatic lactic acid uptake. The postictal lactic acidosis is typically transient and self-limited and typically resolves within 90 minutes. Therefore, the most appropriate management of this patient is observation and a repeat chemistry panel after approximately 2 hours. If the metabolic acidosis has not resolved, other potential causes of anion gap metabolic acidosis (eg, intoxication, sepsis) should be investigated.

(Choice A) The risks and benefits of treating acute metabolic acidosis with sodium bicarbonate are not entirely clear; however, it is generally recommended in patients with severe acute metabolic acidosis with pH <7.1. Administration of sodium bicarbonate may cause myocardial depression and increased lactic acid production; therefore, in patients with pH ≥7.1, the relatively small benefits of sodium bicarbonate do not typically outweigh the risks.

(Choice B) An elevation in the serum amylase level is nonspecific as it can occur in a variety of disease processes (eg, acute pancreatitis, HIV, lymphoma, rheumatologic disease). Serum amylase levels do not play a role in the management of metabolic acidosis.

(Choice C) The common causes of metabolic acidosis due to ketosis include diabetes, alcoholism, and starvation. Ketosis due to seizure activity is not typical.

(Choice E) Serum ammonia levels have minimal clinical use and do not play a role in the management of metabolic acidosis.

(Choice F) Norepinephrine acts as a positive inotrope and vasoconstrictor and is often used in patients with lactic acidosis due to hypotension and poor organ perfusion (eg, sepsis) that persists following fluid resuscitation. Although this patient's metabolic acidosis is likely due to lactic acidosis, the inadequate oxygen delivery to skeletal muscle due to seizure activity was transient and is now resolved. This patient's confusion and lethargy are consistent with a typical postictal state rather than cerebral hypoperfusion.

(Choice A) The risks and benefits of treating acute metabolic acidosis with sodium bicarbonate are not entirely clear; however, it is generally recommended in patients with severe acute metabolic acidosis with pH <7.1. Administration of sodium bicarbonate may cause myocardial depression and increased lactic acid production; therefore, in patients with pH ≥7.1, the relatively small benefits of sodium bicarbonate do not typically outweigh the risks.

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(Choice E) Serum ammonia levels have minimal clinical use and do not play a role in the management of metabolic acidosis.

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Educational objective:
Postictal lactic acidosis commonly occurs following a tonic-clonic seizure. It is a transient anion gap metabolic acidosis that resolves without treatment within 90 minutes following resolution of seizure activity.

References

- Lactic acidosis following convulsions.

B. Check serum amylase [0%]
C. Ch
D. Ob
E. Ob
F. Sta

Omitted
Correct answer
D

Explanation

This patient ha
clonic seizure
uptake. The p
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(Choice A) Th
generally recor
cause myocard
sodium bicarbonate do not typically outweigh the risks.

Exhibit Display

Anion gap metabolic acidosis	
Calculation	Anion gap = Sodium – (Chloride + Bicarbonate) (Normal = 10-14)
Common causes Mnemonic: MUDPILES	• Methanol • Uremia • Diabetic ketoacidosis • Propylene glycol/paraldehyde • Isoniazid/iron • Lactic acidosis • Ethylene glycol (antifreeze) • Salicylates (aspirin)

Add To Flash Card

A 33-year-old woman is brought to the emergency department after an episode of generalized tonic-clonic seizures. She is confused and unable to provide further history. Review of the medical record indicates that she has schizophrenia but stopped taking all her psychiatric medications 3 weeks ago. Temperature is 36.7 C (98 F), blood pressure is 130/76 mm Hg, pulse is 80/min, and respirations are 14/min. Examination shows normal pupils. The patient's lungs are clear to auscultation and her heart sounds are normal. Her abdomen is soft. Extremities examination shows no edema. Laboratory results are as follows:

Serum sodium	118 mEq/L
Serum potassium	3.4 mEq/L
Serum creatinine	0.8 mg/dL
Serum calcium	8.4 mg/dL
Serum glucose	98 mg/dL
Serum osmolality	252 mOsm/kg
Urine osmolality	78 mOsm/kg
Urine specific gravity	1.002

Which of the following is the most likely cause of this patient's electrolyte abnormalities?

A. Adrenal insufficiency
B. Fluoxetine-mediated hypothalamic antidiuretic hormone production
C. Idiopathic central diabetes insipidus
D. Idiopathic nephrogenic diabetes insipidus

Serum potassium	3.4 mEq/L
Serum creatinine	0.8 mg/dL
Serum calcium	8.4 mg/dL
Serum glucose	98 mg/dL
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- D. Idiopathic nephrogenic diabetes insipidus
- E. Primary polydipsia
- F. Syndrome of inappropriate antidiuretic hormone secretion due to lung cancer

Submit

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Serum calcium	8.4 mg/dL
Serum glucose	98 mg/dL
Serum osmolality	252 mOsm/kg
Urine osmolality	78 mOsm/kg
Urine specific gravity	1.002

Which of the following is the most likely cause of this patient's electrolyte abnormalities?

A. Adrenal insufficiency [3%]
B. Fluoxetine-mediated hypothalamic antidiuretic hormone production [8%]
C. Idiopathic central diabetes insipidus [6%]
D. Idiopathic nephrogenic diabetes insipidus [11%]

Serum creatinine	0.8 mg/dL
Serum calcium	8.4 mg/dL
Serum glucose	98 mg/dL
Serum osmolality	252 mOsm/kg
Urine osmolality	78 mOsm/kg
Urine specific gravity	1.002

Which of the following is the most likely cause of this patient's electrolyte abnormalities?

- A. Adrenal insufficiency [3%]
- B. Fluoxetine-mediated hypothalamic antidiuretic hormone production [8%]
- C. Idiopathic central diabetes insipidus [6%]
- D. Idiopathic nephrogenic diabetes insipidus [11%]
- ✔ E. Primary polydipsia [67%]
- F. Syndrome of inappropriate antidiuretic hormone secretion due to lung cancer [2%]

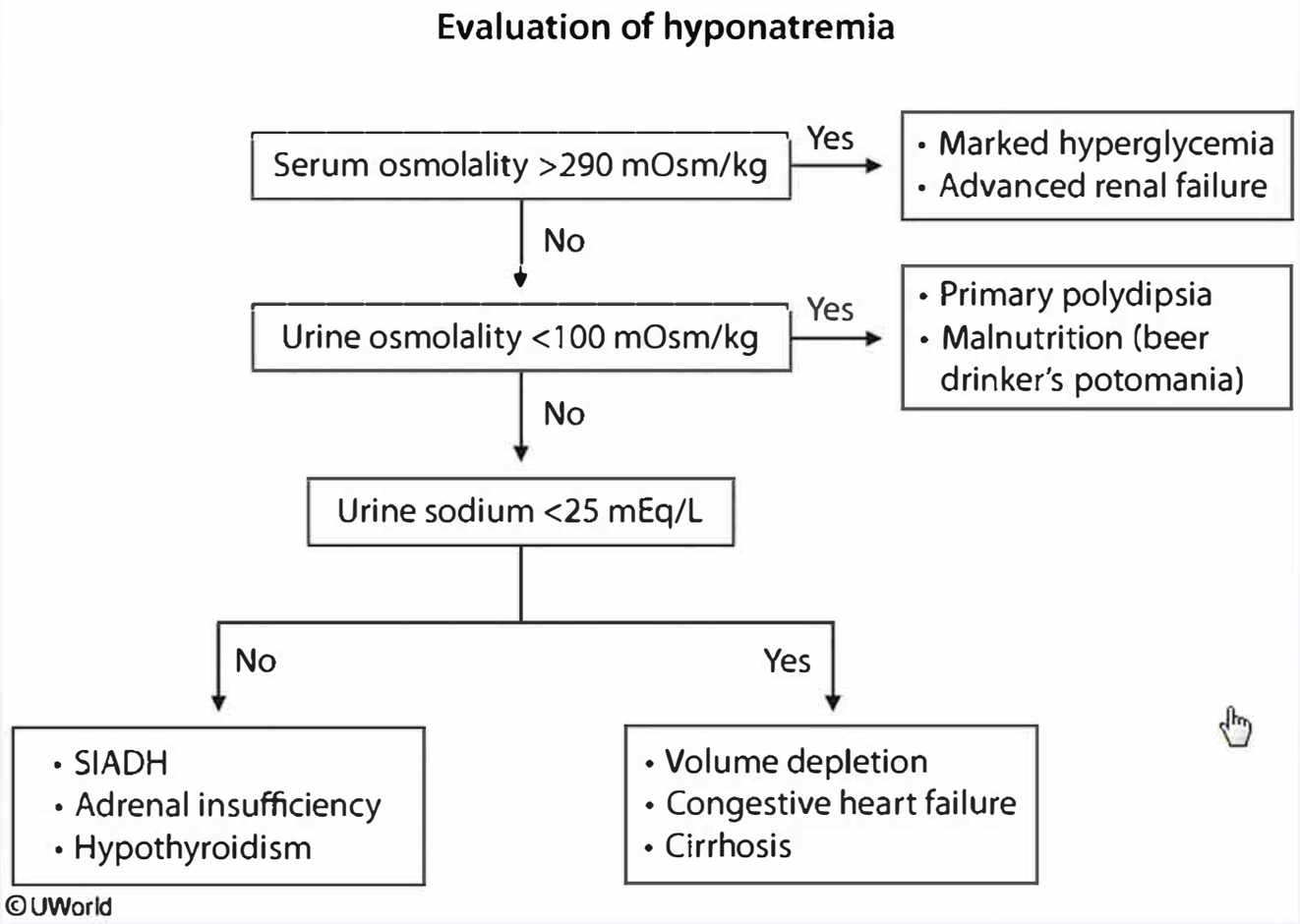
Omitted
Correct answer
E

67%
Answered correctly

6 Seconds
Time Spent

11/18/2018
Last Updated

Explanation



This patient has had seizures due to severe **hyponatremia**. Hyponatremia is often due to excess antidiuretic hormone (ADH) secretion leading to impaired renal water excretion. However, hyponatremia can also be due to significantly increased water intake

serum osmolality >290 mOsm/kg suggests either marked hyperglycemia or advanced renal failure as likely causes of the hyponatremia. The next step in evaluating patients with serum osmolality <290 mOsm/kg is to measure the urine osmolality. A **urine osmolality <100** mOsm/kg, as in this patient, suggests either primary polydipsia or malnutrition (eg, beer potomania).

Primary polydipsia is more common in patients with psychiatric conditions (eg, schizophrenia), possibly due to a central defect in thirst regulation. These patients continue to drink water despite a decreased serum osmolality that should normally inhibit the thirst reflex. The kidney increases water excretion, which **dilutes the urine** maximally to an osmolality <100 mOsm/kg. However, hyponatremia can develop if the water intake is higher than the kidney's ability to excrete water. Patients with significant hyponatremia can develop confusion, lethargy, psychosis, and seizures.

(Choice A) Adrenal insufficiency typically presents with hypovolemia, which leads to increased ADH and hyponatremia. However, the increased ADH would lead to concentrated urine with urine osmolality greater than the serum osmolality.

(Choices C and D) Diabetes insipidus can be central (decreased ADH release from the pituitary) or nephrogenic (normal ADH levels with renal ADH resistance). The decreased ADH action in both types leads to decreased renal water reabsorption, water loss with polyuria, and dilute urine. However, patients typically develop hypernatremia.

(Choices B and F) Many drugs (eg, carbamazepine, cyclophosphamide, selective serotonin reuptake inhibitors such as fluoxetine) can stimulate hypothalamic ADH production and cause syndrome of inappropriate antidiuretic hormone secretion (SIADH). Lung cancer (eg, small cell cancer) can also cause SIADH due to ectopic ADH production by the tumor cells. The excess ADH in both cases leads to water retention in the kidney and hyponatremia. However, the urine is inappropriately concentrated (instead of dilute). As a result, the urine osmolality is higher than the serum osmolality.

Educational objective:
Causes of hyponatremia include syndrome of inappropriate antidiuretic hormone secretion and primary polydipsia. Primary polydipsia is more common in patients with psychiatric conditions (eg, schizophrenia), possibly due to a central defect in thirst regulation. Patients typically develop hyponatremia and dilute urine with urine osmolality <100 mOsm/kg. Those with significant hyponatremia can develop confusion, lethargy, psychosis, and seizures.

A 60-year-old man who emigrated from Russia comes to the physician complaining of dizziness, fatigue, and weight loss. He has experienced daily fevers and cough for the past 2 months. The patient does not use tobacco, alcohol, or illicit drugs and takes no medications. His blood pressure is 98/54 mm Hg while standing, pulse is 105/min and regular, and respirations are 14/min and unlabored. Oxygen saturation is 98% on room air. Laboratory results are as follows:

Chemistry panel	
Sodium	132 mEq/L
Potassium	5.9 mEq/L
Chloride	102 mEq/L
Creatinine	0.8 mg/dL
Glucose	55 mg/dL
Complete blood count	
Hemoglobin	10.0 g/dL
Platelets	430,000/mm^3
Leukocytes	4500/mm^3
Neutrophils	46%
Lymphocytes	45%
Eosinophils	9%

Glucose	55 mg/dL
Complete blood count	
Hemoglobin	10.0 g/dL
Platelets	430,000/mm^3
Leukocytes	4500/mm^3
Neutrophils	46%
Lymphocytes	45%
Eosinophils	9%

Chest x-ray shows a right upper-lobe cavitary lesion. Which of the following acid-base disturbances is expected in this patient?

- A. Elevated anion gap metabolic acidosis
- B. Metabolic alkalosis
- C. Normal anion gap metabolic acidosis
- D. Respiratory acidosis
- E. Respiratory alkalosis

Submit

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Leukocytes	4500/mm^3
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Eosinophils	9%

Complete blood count

Hemoglobin	10.0 g/dL
Platelets	430,000/mm^3
Leukocytes	4500/mm^3
Neutrophils	46%
Lymphocytes	45%
Eosinophils	9%

Chest x-ray shows a right upper-lobe cavitary lesion. Which of the following acid-base disturbances is expected in this patient?

- A. Elevated anion gap metabolic acidosis [20%]
- B. Metabolic alkalosis [11%]
- ✔ C. Normal anion gap metabolic acidosis [40%]
- D. Respiratory acidosis [17%]
- E. Respiratory alkalosis [10%]

Omitted
Correct answer
C

40%
Answered correctly

7 Seconds
Time Spent

12/30/2018
Last Updated

Explanation

Clinical features of primary adrenal insufficiency	
Etiology	• Autoimmune • Infections (eg, tuberculosis, HIV, disseminated fungal) • Hemorrhagic infarction (eg, meningococcemia, anticoagulants) • Metastatic cancer (eg, lung)
Clinical presentation	**Acute** • Most commonly with shock • Abdominal tenderness with deep palpation (unclear etiology) • Unexplained fever • Nausea, vomiting, weight loss & anorexia • Hyponatremia, hyperkalemia, hypercalcemia & eosinophilia **Chronic** • Fatigue, weakness & anorexia • Gastrointestinal (eg, nausea, vomiting, abdominal pain) • Weight loss • Hyperpigmentation or vitiligo • Hypotension, hyponatremia, hyperkalemia & hypercalcemia • Anemia & eosinophilia

Etiology	• Infections (eg, tuberculosis, HIV, disseminated fungal) • Hemorrhagic infarction (eg, meningococcemia, anticoagulants) • Metastatic cancer (eg, lung)
Clinical presentation	**Acute** • Most commonly with shock • Abdominal tenderness with deep palpation (unclear etiology) • Unexplained fever • Nausea, vomiting, weight loss & anorexia • Hyponatremia, hyperkalemia, hypercalcemia & eosinophilia **Chronic** • Fatigue, weakness & anorexia • Gastrointestinal (eg, nausea, vomiting, abdominal pain) • Weight loss • Hyperpigmentation or vitiligo • Hypotension, hyponatremia, hyperkalemia & hypercalcemia • Anemia & eosinophilia
Diagnosis	• Measure ACTH and serum cortisol with high-dose (250 µg) ACTH stimulation test • **Primary adrenal insufficiency:** Low cortisol, high ACTH • **Secondary/tertiary adrenal insufficiency:** Low cortisol, low ACTH

This patient's presentation is consistent with likely tuberculosis (TB). He comes from a TB-endemic area and presents with fever, cough, and a right upper-lobe cavitary lesion on chest x-ray. Common extrapulmonary sites for TB include liver, spleen, kidney, bone, and adrenal gland. TB is a common cause of chronic primary adrenal insufficiency in endemic areas. Patients typically develop gradual fatigue, weakness, borderline hypotension, and electrolyte abnormalities. This patient also has hyperkalemia, hypoglycemia, and eosinophilia, findings suggestive of adrenal insufficiency. Other granulomatous diseases (eg, histoplasmosis, coccidioidomycosis, cryptococcosis, and sarcoidosis) may also cause adrenal insufficiency.

Primary adrenal insufficiency (Addison's disease) is characterized by decreased cortisol, adrenal sex hormone, and aldosterone secretion. Aldosterone normally acts on the distal renal tubules to increase sodium reabsorption (saves sodium) and secrete potassium and hydrogen ions. If aldosterone is deficient, the kidney inappropriately loses sodium while retaining excessive potassium and hydrogen ions. This results in a normal anion gap and hyperkalemic and hyponatremic metabolic acidosis.

(Choice A) Elevated anion gap metabolic acidosis is characteristically seen in the following conditions: ketoacidosis (eg, alcoholic, diabetic, starvation), intoxications (eg, methanol, salicylate, ethylene glycol, isoniazide, metformin), tissue hypoxia (eg, ischemia, carbon monoxide, cyanide), and renal failure.

(Choice B) Metabolic alkalosis occurs with vomiting (most common), hyperaldosteronism, and excessive volume contraction (eg, thiazides, loop diuretics). This patient's absence of vomiting, medication use, and hypokalemia makes these unlikely.

(Choice D) Respiratory acidosis is caused by conditions that impair proper ventilation. This patient is not hypoventilating and has unlabored respirations, making respiratory acidosis less likely.

(Choice E) Respiratory alkalosis is usually due to conditions that cause hyperventilation. Pulmonary parenchymal disease leading to respiratory failure can also cause respiratory alkalosis, but this patient has no findings of respiratory failure (eg, tachypnea, dyspnea).

Educational objective:
Tuberculosis is a common cause of chronic primary adrenal insufficiency (Addison's disease) in endemic areas. Addison's disease

cryptococcosis, and sarcoidosis) may also cause adrenal insufficiency.

Primary adrenal insufficiency (Addison's disease) is characterized by decreased cortisol, adrenal sex hormone, and aldosterone secretion. Aldosterone normally acts on the distal renal tubules to increase sodium reabsorption (saves sodium) and secrete potassium and hydrogen ions. If aldosterone is deficient, the kidney inappropriately loses sodium while retaining excessive potassium and hydrogen ions. This results in a normal anion gap and hyperkalemic and hyponatremic metabolic acidosis.

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Educational objective:
Tuberculosis is a common cause of chronic primary adrenal insufficiency (Addison's disease) in endemic areas. Addison's disease causes aldosterone deficiency and presents with a non-anion gap and hyperkalemic and hyponatremic metabolic acidosis.

References

- A case of disseminated tuberculosis with adrenal insufficiency.

A 36-year-old man is brought to the emergency department due to confusion, nausea, and decreased arousal. He is unable to answer questions, and no other history is available. Temperature is 36.7 C (98.2 F), pulse is 86/min, and respirations are 22/min. His arterial blood gas and serum electrolyte results are shown below:

pH	7.21
PaO2	96 mm Hg
PaCO2	28 mm Hg
Sodium	136 mEq/L
Potassium	3.6 mEq/L
Chloride	90 mEq/L
Bicarbonate	12 mEq/L
Blood urea nitrogen	30 mg/dL
Creatinine	1.2 mg/dL

What is the most likely primary acid-base disorder in this patient?

A. Non-anion gap metabolic acidosis [3%]
B. Anion gap metabolic acidosis [93%]
C. Metabolic alkalosis [0%]
D. Respiratory alkalosis [0%]

PaO2	96 mm Hg
PaCO2	28 mm Hg
Sodium	136 mEq/L
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Submit

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- C. Metabolic alkalosis [0%]
- D. Respiratory alkalosis [0%]
- E. Respiratory acidosis [1%]

Omitted
Correct answer
B

93%
Answered correctly

4 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

This patient's primary acid-base disorder is **anion gap metabolic acidosis**. This is evidenced by his acidic pH (< 7.35) and his severe primary decrease in HCO3 (< 24 mEq/L). Normally, the anion gap (AG) is made up of negatively charged molecules (eg, protein, citrate, phosphate, sulfate) that are normally present in serum. These molecules account for the normal anion gap value of 10-14 mEq/L. The AG can be calculated using the following formula:

AG = Measured cations (positive charge) − Measured anions (negative charge)

Because the major cation in the serum is Na^+, and the major anions in the serum are Cl^- and HCO_3^-, the formula for plasma AG can be simplified as follows:

$$AG = Na^+ - (HCO_3^- + Cl^-)$$

This patient's calculated AG = 136 − (90 + 12) = 136 − 102 = 34 mEq/L, an abnormally elevated value. An increase in the AG indicates the presence of nonchloride acids that contain inorganic (phosphate, sulfate), organic (ketoacids, lactate, uremic organic anions), exogenous (salicylate or ingested toxins with organic acid production), or unidentified anions.

The most common causes of anionic gap metabolic acidosis and their corresponding unmeasured anions that compose the anion gap are:

- Lactic acidosis (lactate)
- Ketoacidosis (beta-hydroxy butyrate, acetoacetic acid)
- Methanol / formaldehyde ingestion (formic acid)
- Ethylene glycol ingestion (glycolic acid, oxalic acid)
- Salicylate poisoning (salicylic, lactic, sulfuric and phosphoric acids)
- Uremia (eg, endstage renal disease) (impaired excretion of H^+)

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Educational objective:
The anion gap (AG) represents the concentration of unmeasured serum anions. In anion gap metabolic acidosis, the AG is increased by the abnormal presence of non-chlorinated acids in the serum.

A 67-year-old man with mild chronic obstructive pulmonary disease is brought to the emergency department by his daughter with the chief complaint of seizure. The patient has had episodes of confusion and lethargy over the past week. He complains of some exertional shortness of breath and nonproductive cough. His daughter believes that he is losing weight and has decreased appetite. His temperature is 37.2 C (99 F), blood pressure is 134/88 mm Hg, and pulse is 104/min and irregular. The mucous membranes are moist and there is no peripheral edema. Neurologic examination is unremarkable. Laboratory results are as follows:

Complete blood count	
Hematocrit	34%
Serum chemistry	
Sodium	117 mEq/L
Potassium	5.4 mEq/L
Bicarbonate	22 mEq/L
Creatinine	1.3 mg/dL

Rapid correction of this patient's metabolic abnormalities puts him at highest risk of which of the following?

A. Cerebral edema

B. Embolic stroke

Serum chemistry

Sodium	117 mEq/L
Potassium	5.4 mEq/L
Bicarbonate	22 mEq/L
Creatinine	1.3 mg/dL

Rapid correction of this patient's metabolic abnormalities puts him at highest risk of which of the following?

A. Cerebral edema
B. Embolic stroke
C. Hydrocephalus
D. Osmotic demyelination
E. Ventricular arrhythmias

Submit

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Creatinine	1.3 mg/dL

Rapid correction of this patient's metabolic abnormalities puts him at highest risk of which of the following?

A. Cerebral edema [10%]

B. Embolic stroke [0%]

Sodium	117 mEq/L
Potassium	5.4 mEq/L
Bicarbonate	22 mEq/L
Creatinine	1.3 mg/dL

Rapid correction of this patient's metabolic abnormalities puts him at highest risk of which of the following?

- A. Cerebral edema [10%]
- B. Embolic stroke [0%]
- C. Hydrocephalus [0%]
- ✔ D. Osmotic demyelination [87%]
- E. Ventricular arrhythmias [1%]

Omitted
Correct answer
D

87%
Answered correctly

7 Seconds
Time Spent

10/02/2018
Last Updated

Explanation

This patient presents with a provoked seizure in the setting of severe hyponatremia. This is considered a medical emergency and requires prompt correction of the serum sodium concentration with 3% saline solution. However, correction of the serum sodium should not exceed 0.5 mEq/L/hr to avoid causing irreversible brain damage from osmotic demyelination or central pontine myelinolysis. Rapid correction of serum sodium in the setting of hyponatremia results in excess water being moved by osmosis from the intracellular compartment (neurons and glia) into the extracellular compartment. This in turn leads to disruption of cellular metabolic activity and subsequent cell damage. The opposite is true when rapidly correcting a patient with hypernatremia, when cerebral edema can occur **(Choice A)**.

This patient's hyponatremia could be from lung cancer-associated syndrome of inappropriate antidiuretic hormone given his euvolemic status, recent weight loss, and smoking history.

(Choice B) This patient has an irregular heart rhythm, which may be secondary to atrial fibrillation, and therefore is at increased risk of an embolic stroke. However, rapid correction of his hyponatremia will not contribute to that risk.

(Choice C) Rapid correction of hyponatremia results in increased water movement out of brain tissue and so would not contribute to the formation of hydrocephalus. Hydrocephalus can be either obstructive (noncommunicating) or nonobstructive (communicating), resulting from excess cerebrospinal fluid production or impaired cerebrospinal fluid absorption.

(Choice E) Electrolyte abnormalities can result in cardiac arrhythmias. However, rapid correction of hyponatremia is more likely to result in osmotic demyelination in the central nervous system than to cause a ventricular arrhythmia.

Educational objective:
Acute, symptomatic hyponatremia (impaired mental status/seizures) is a medical emergency. It requires a prompt increase in the serum sodium concentration with 3% or hypertonic saline at a rate of no more than 0.5 mEq/L/hr to avoid causing central nervous system osmotic demyelination syndrome.

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A 45-year-old man is brought to the emergency department due to altered mental status. The patient appears agitated and disoriented. He was recently treated by his physician for cold symptoms. He also takes aspirin for chronic arthritis and ranitidine for acid reflux. He works as an auto mechanic. He has smoked for the last 25 years and drinks heavily during the weekend. His temperature is 37.2 C (99 F), blood pressure is 110/70 mm Hg, pulse is 90/min, and respirations are 22/min. His laboratory findings are as follows:

Serum chemistry	
Sodium	141 mEq/L
Potassium	4.6 mEq/L
Chloride	100 mEq/L
Bicarbonate	13 mEq/L
Blood urea nitrogen	28 mg/dL
Creatinine	2.5 mg/dL
Glucose	90 mg/dL
Osmolality, plasma	350 mOsm/kg H_2O
Arterial blood gases	
pH	7.21
PaO_2	100 mm Hg
$PaCO_2$	30 mm Hg

Creatinine	2.5 mg/dL
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Arterial blood gases

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PaO_2	100 mm Hg
$PaCO_2$	30 mm Hg

Urinalysis shows rectangular, envelope-shaped crystals. His creatinine 3 months ago was 1.2 mg/dL. What is the most likely cause of the laboratory abnormalities seen in this patient?

A. Diabetic ketoacidosis
B. Ethylene glycol poisoning
C. Lactic acidosis
D. Salicylate poisoning
E. Uremic acidosis

Submit

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- A. Diabetic ketoacidosis [0%]
- ✔ B. Ethylene glycol poisoning [80%]
- C. Lactic acidosis [1%]
- D. Salicylate poisoning [8%]
- E. Uremic acidosis [8%]

Omitted
Correct answer
B

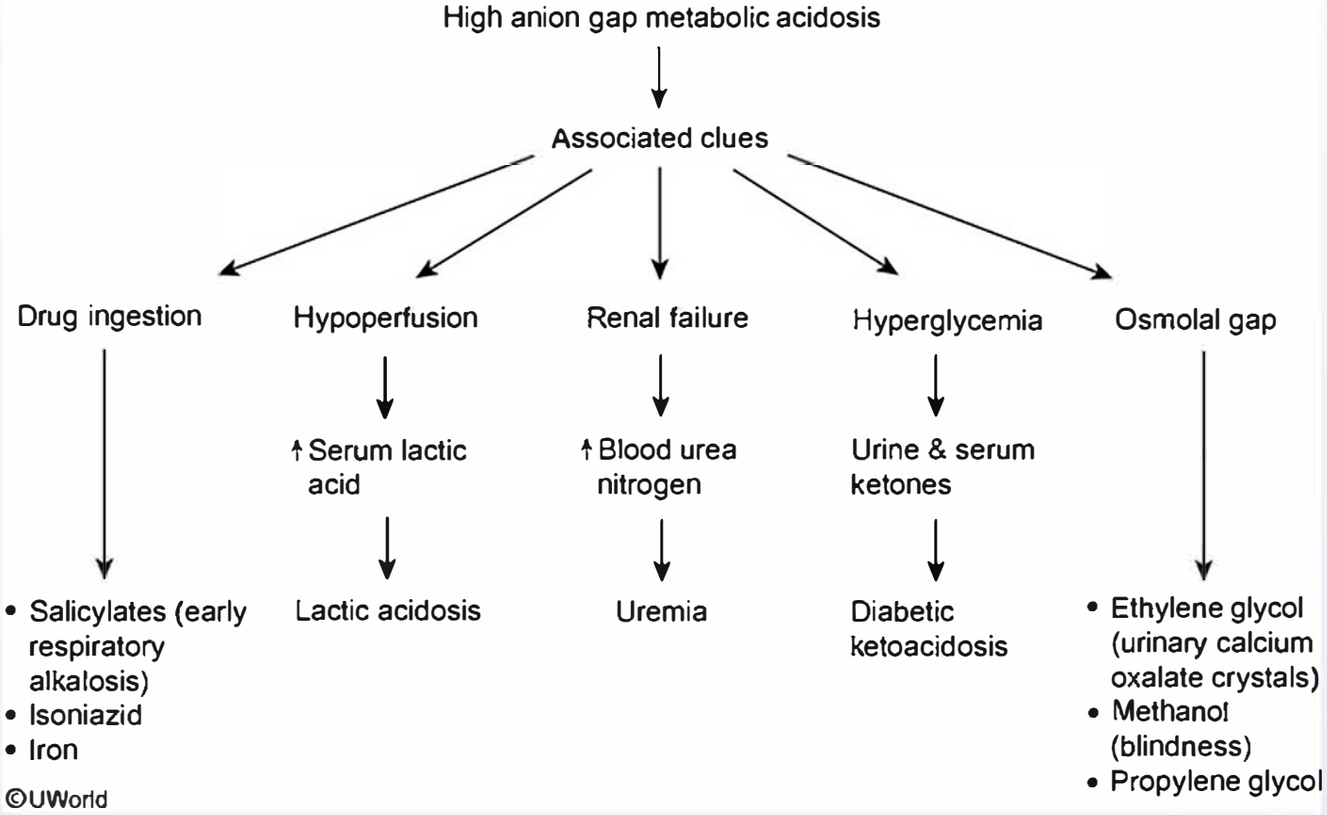
80%
Answered correctly

5 Seconds
Time Spent

09/23/2018
Last Updated

Explanation

Workup of high anion gap metabolic acidosis



This patient has metabolic acidosis (pH <7.35, primary decrease in HCO_3^-) with an increased **anion gap** (141 – [100 + 13] = 28). His measured serum osmolality (often obtained in patients with anion gap metabolic acidosis and suspected **ingestion**) is also elevated (350 mOsm/kg H_2O). An **osmolal gap**, found to be elevated in this patient, should be calculated as follows:

• Propylene glycol

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Osmolal gap = measured serum osmolality − calculated serum osmolality
= 350 – 297 = 53 mOsm/kg H_2O (normal <10),

where calculated serum osmolality = (2*sodium) + (glucose/18) + (blood urea nitrogen/2.8) = (2*141) + (90/18) + (28/2.8) = 297 mOsm/kg H_2O.

A **combination** of high anion gap and osmolal gap metabolic acidosis is seen with acute ethanol (most common), methanol, or ethylene glycol poisoning. The patient's urinalysis shows rectangular, **envelope-shaped calcium oxalate crystals**, which are classically observed in patients with **ethylene glycol** poisoning, most commonly from **antifreeze** ingestion. **Acute renal failure** is the major complication of ethylene glycol intoxication (methanol intoxication can lead to blindness).

(Choice A) Metabolic acidosis in diabetic ketoacidosis is accompanied by high blood glucose levels. This patient is euglycemic.

(Choice C) Lactic acidosis is most commonly seen in states of hypoperfusion and shock (eg, sepsis, cardiogenic shock). Lactic acid levels may also be elevated in patients with end-stage liver disease as lactic acid is metabolized in the liver.

(Choice D) Aspirin (salicylate) toxicity causes a mixed anion gap metabolic acidosis and respiratory alkalosis with no osmolal gap.

(Choice E) Uremia (renal failure) causes an anion gap metabolic acidosis because acid excretion (through formation of ammonium) is impaired. However, uremia does not cause an osmolal gap.

Educational objective:
Ethylene glycol, methanol, and ethanol intoxication cause metabolic acidosis with both an anion gap and an osmolal gap. Calcium oxalate crystals (rectangular, envelope-shaped) are seen in patients with ethylene glycol (antifreeze) poisoning. Ethylene glycol

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(Choice E) Uremia (renal failure) causes an anion gap metabolic acidosis because acid excretion (through formation of ammonium) is impaired. However, uremia does not cause an osmolal gap.

Educational objective:
Ethylene glycol, methanol, and ethanol intoxication cause metabolic acidosis with both an anion gap and an osmolal gap. Calcium oxalate crystals (rectangular, envelope-shaped) are seen in patients with ethylene glycol (antifreeze) poisoning. Ethylene glycol intoxication can result in renal failure; methanol intoxication can cause blindness.

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ketoacidosis

(urinary calcium oxalate crystals)

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Exhibit Display

Anion gap metabolic acidosis	
Calculation	Anion gap = Sodium – (Chloride + Bicarbonate) (Normal = 10-14)
Common causes Mnemonic: MUDPILES	• Methanol • Uremia • Diabetic ketoacidosis • Propylene glycol/paraldehyde • Isoniazid/iron • Lactic acidosis • Ethylene glycol (antifreeze) • Salicylates (aspirin)

Add To Flash Card

respiratory

ketoacidosis

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This patient ha
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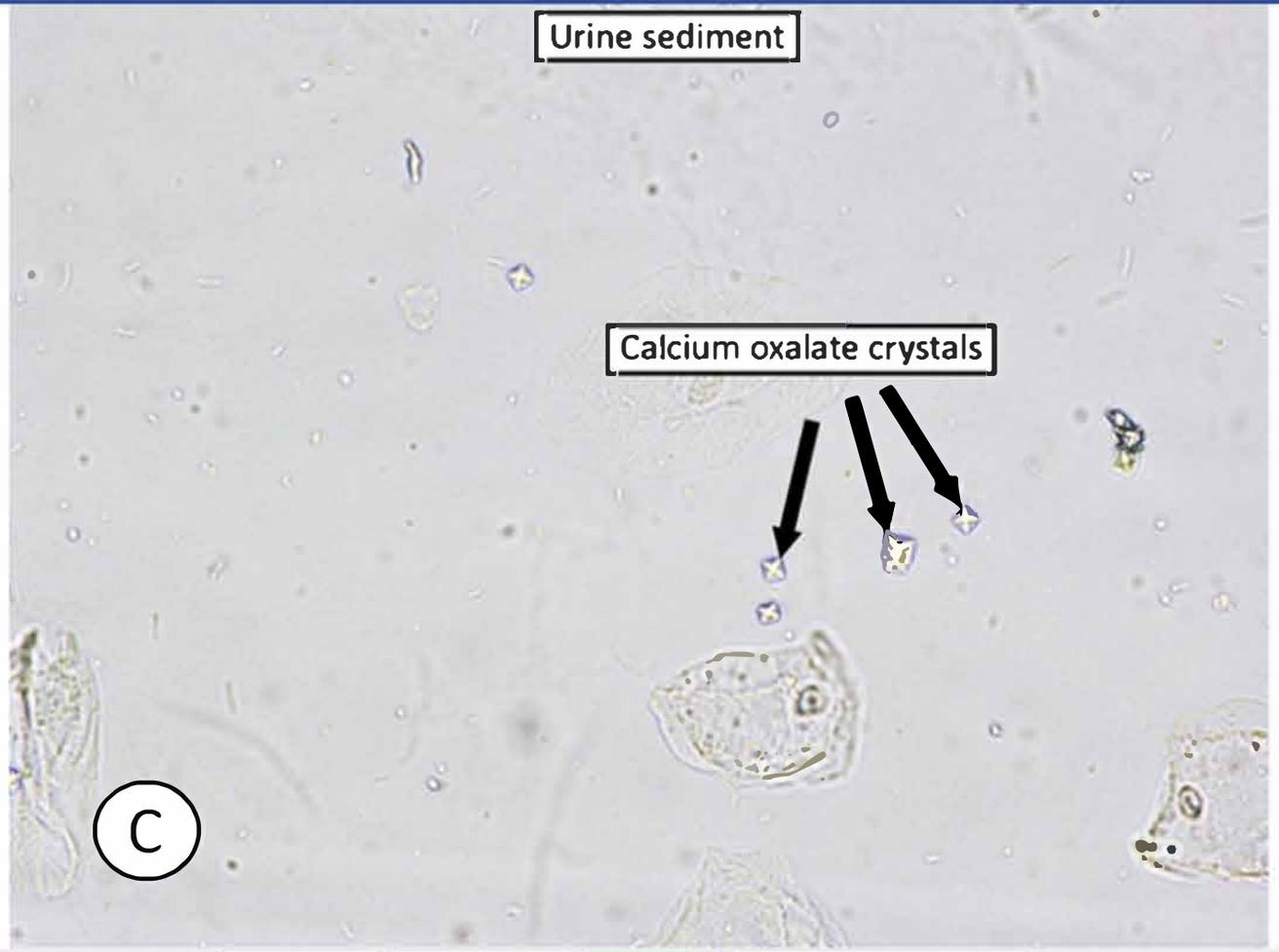
Exhibit Display



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A 65-year-old woman comes to the physician due to a 2-month history of fatigue and weight gain. The patient has rheumatoid arthritis, peptic ulcer disease, and hypertension. She takes hydrochlorothiazide and acetaminophen. She does not use tobacco, alcohol, or illicit drugs. The patient's blood pressure is 120/70 mm Hg, pulse is 80/min, and respirations are 14/min. Physical examination shows generalized edema; liver is palpated 5 cm below the costal margin. Urinalysis shows 4+ proteinuria. Ultrasound of the kidneys shows bilateral enlargement. Renal biopsy was performed. Which of the following is the most likely finding on pathological examination?

A. Glomerular crescent formation on light microscopy
B. Glomerular deposits seen after special staining
C. Hyalinosis of walls of afferent and efferent arterioles
D. Linear glomerular deposits seen on immunofluorescence microscopy
E. Normal light microscopy findings

Submit

A 65-year-old woman comes to the physician due to a 2-month history of fatigue and weight gain. The patient has rheumatoid arthritis, peptic ulcer disease, and hypertension. She takes hydrochlorothiazide and acetaminophen. She does not use tobacco, alcohol, or illicit drugs. The patient's blood pressure is 120/70 mm Hg, pulse is 80/min, and respirations are 14/min. Physical examination shows generalized edema; liver is palpated 5 cm below the costal margin. Urinalysis shows 4+ proteinuria. Ultrasound of the kidneys shows bilateral enlargement. Renal biopsy was performed. Which of the following is the most likely finding on pathological examination?

- A. Glomerular crescent formation on light microscopy [15%]
- ✔ B. Glomerular deposits seen after special staining [36%]
- C. Hyalinosis of walls of afferent and efferent arterioles [23%]
- D. Linear glomerular deposits seen on immunofluorescence microscopy [10%]
- E. Normal light microscopy findings [13%]

Omitted
Correct answer
B

36%
Answered correctly

3 Seconds
Time Spent

09/05/2018
Last Updated

Explanation

	AL amyloidosis	AA amyloidosis

Explanation

	AL amyloidosis	AA amyloidosis
Associated conditions	• Multiple myeloma • Waldenström macroglobulinemia	• Chronic inflammatory conditions: **rheumatoid arthritis**, inflammatory bowel disease • Chronic infections: osteomyelitis, tuberculosis
Composition of amyloid	• Light chains (usually lambda)	• Abnormally folded proteins: beta-2 microglobulin, apolipoprotein or transthyretin

AA amyloidosis = inflammatory amyloidosis; **AL amyloidosis** = amyloid light-chain amyloidosis.

This patient has nephrotic syndrome, evidenced by edema and a high degree of proteinuria. Common causes of nephrotic syndrome in adults are membranous glomerulopathy, focal segmental glomerulosclerosis, minimal change disease, and amyloidosis.

Amyloidosis is the most probable cause of nephrotic syndrome in this patient. Clues to this diagnosis include a history of rheumatoid arthritis (that predisposes to amyloidosis), enlarged kidneys, and hepatomegaly. The typical findings on renal biopsy are **amyloid deposits that stain with Congo red** and demonstrate a characteristic **apple-green birefringence under polarized light**. These amyloid deposits are seen in the glomerular basement membrane, blood vessels, and interstitium of the kidneys and can be seen on electron microscopy as randomly arranged **thin fibrils**. The deposits may consist of light chains (AL amyloidosis) or abnormal proteins (AA amyloidosis). Rheumatoid arthritis is the most common cause of AA amyloidosis in the United States.

(Choice A) Crescent formation on light microscopy is the characteristic finding in rapidly progressive glomerulonephritis.

Amyloidosis is the most probable cause of nephrotic syndrome in this patient. Clues to this diagnosis include a history of rheumatoid arthritis (that predisposes to amyloidosis), enlarged kidneys, and hepatomegaly. The typical findings on renal biopsy are **amyloid deposits that stain with Congo red** and demonstrate a characteristic **apple-green birefringence under polarized light**. These amyloid deposits are seen in the glomerular basement membrane, blood vessels, and interstitium of the kidneys and can be seen on electron microscopy as randomly arranged **thin fibrils**. The deposits may consist of light chains (AL amyloidosis) or abnormal proteins (AA amyloidosis). Rheumatoid arthritis is the most common cause of AA amyloidosis in the United States.

(Choice A) Crescent formation on light microscopy is the characteristic finding in rapidly progressive glomerulonephritis.

(Choice C) Hyalinosis that affects both afferent and efferent arterioles is pathognomonic of diabetic nephropathy.

(Choice D) Linear deposits on immunofluorescence microscopy are typical for antiglomerular basement membrane disease (eg, Goodpasture's syndrome). Granular deposits are usually present in immune complex glomerulonephritis (eg, lupus nephritis, IgA nephropathy, postinfectious glomerulonephritis).

(Choice E) Normal light microscopy findings in a patient with nephrotic syndrome usually suggest minimal change disease.

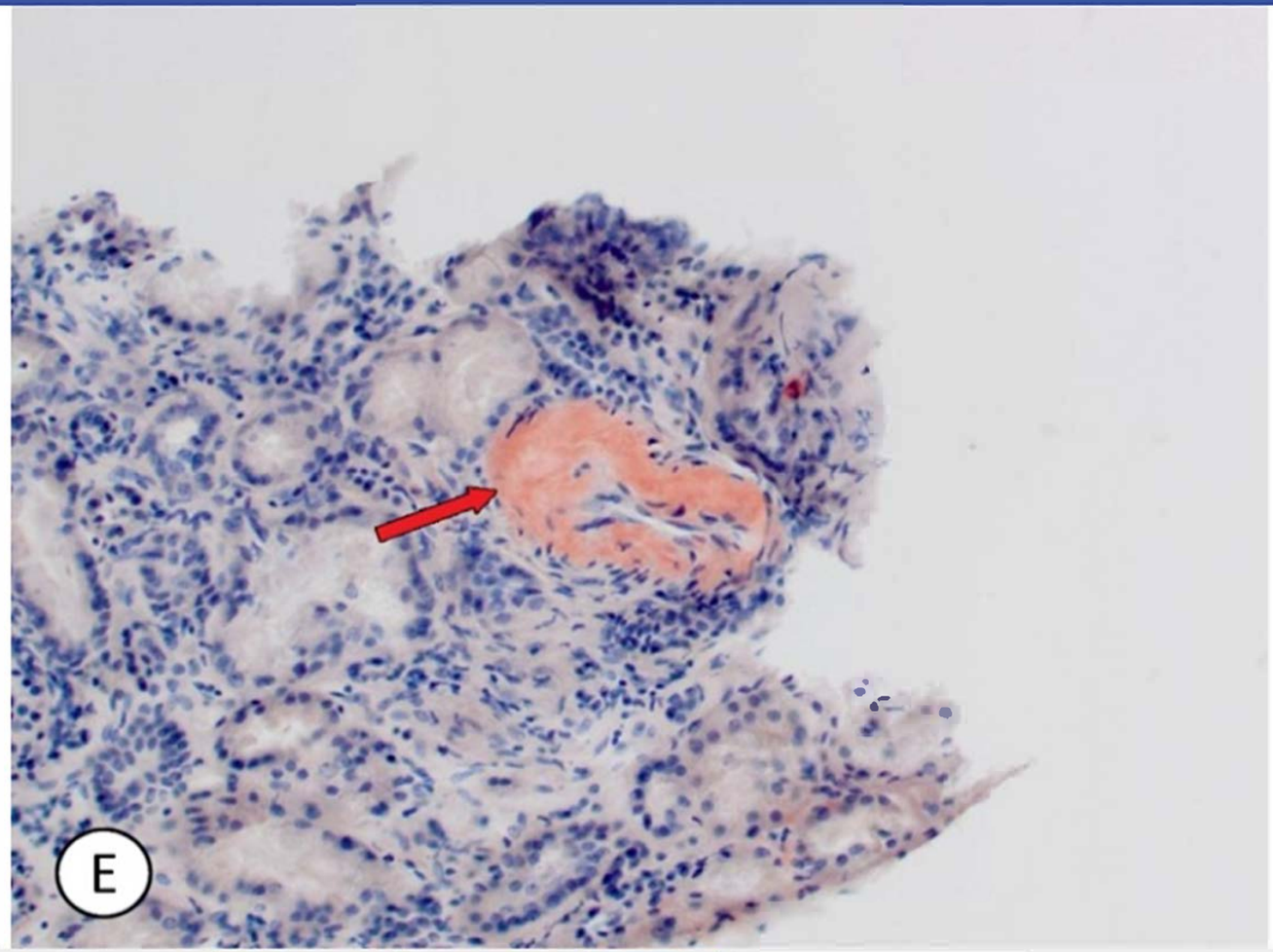
Educational objective:
Rheumatoid arthritis predisposes to amyloidosis. Renal involvement is characterized by nephrotic syndrome. The classic pathologic finding is amyloid deposits that stain with Congo red and demonstrate apple-green birefringence under polarized light. Multiple myeloma is the most common cause of AL amyloidosis, and rheumatoid arthritis is the most common cause of AA amyloidosis.

References

- Nephrotic syndrome secondary to amyloidosis.
- Immunoglobulin light chain amyloidosis: 2014 update on diagnosis, prognosis, and treatment.

- Abnormally folded proteins:

Exhibit Display



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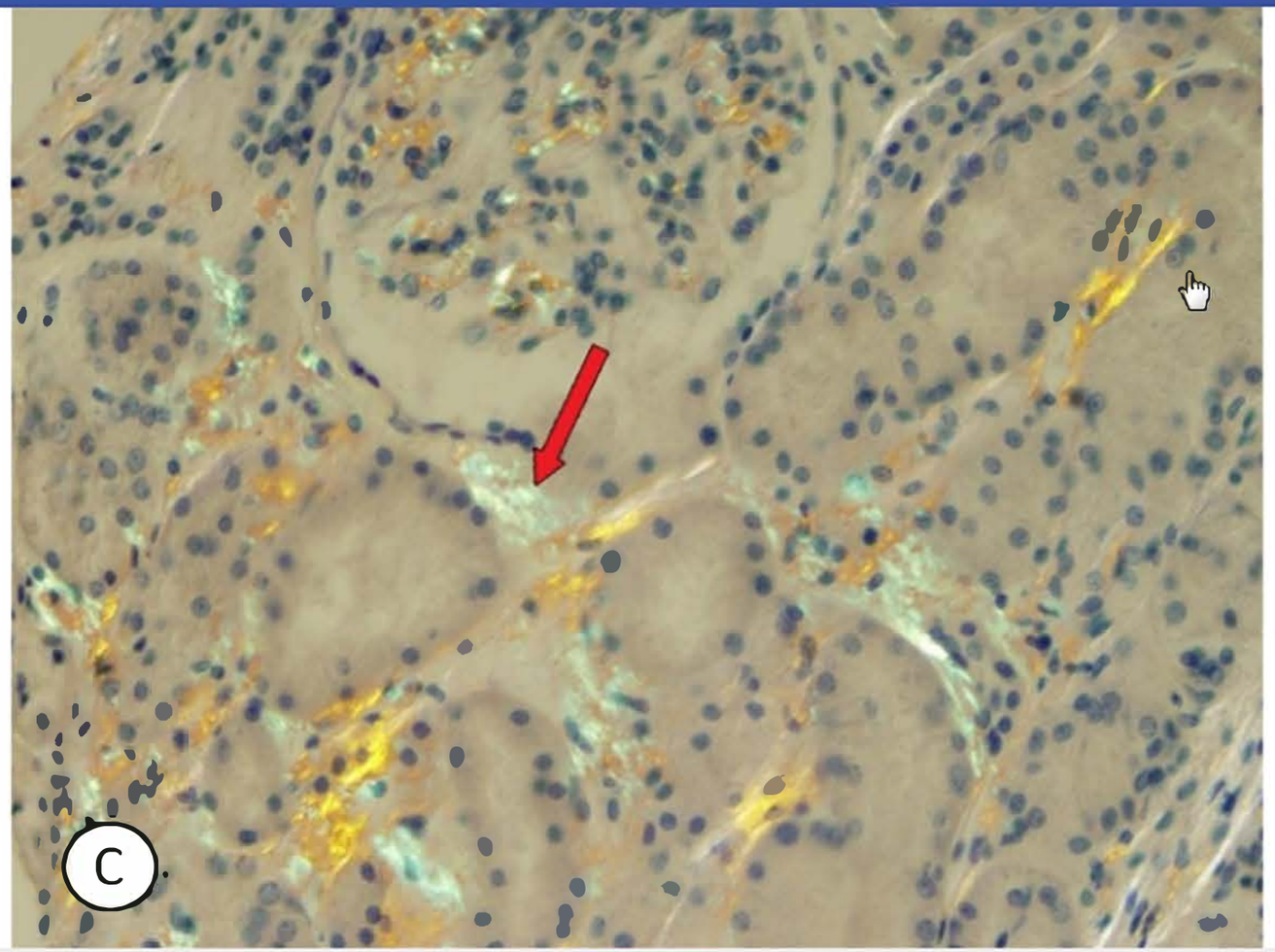

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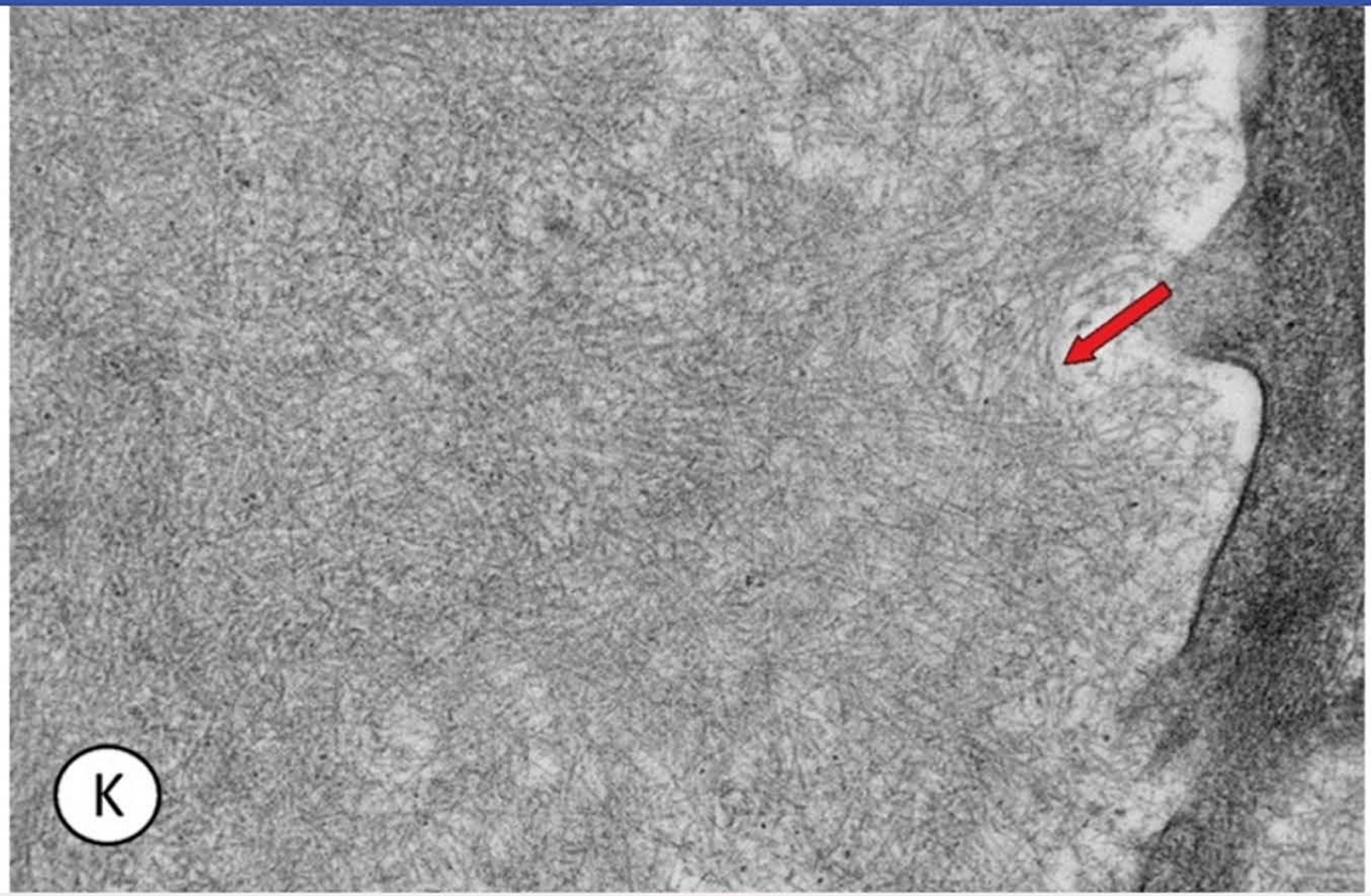

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myeloma is the most common cause of AL amyloidosis, and rheumatoid arthritis is the most common cause of AA amyloidosis

Arterial blood gas results on room air from a patient newly admitted to the intensive care unit are as follows:

pH	7.42
$PaCO_2$	25 mm Hg
PaO_2	100 mm Hg
Bicarbonate	14 mEq/L

Which of the following patients is most likely to have these laboratory values?

A. 36-year-old patient with stridor after an allergic reaction
B. 42-year-old patient with acute asthma exacerbation
C. 42-year-old patient with aspirin toxicity
D. 52-year-old patient with persistent vomiting
E. 64-year-old patient with excessive diuresis

Submit

Arterial blood gas results on room air from a patient newly admitted to the intensive care unit are as follows:

pH	7.42
$PaCO_2$	25 mm Hg
PaO_2	100 mm Hg
Bicarbonate	14 mEq/L

Which of the following patients is most likely to have these laboratory values?

- A. 36-year-old patient with stridor after an allergic reaction [3%]
- B. 42-year-old patient with acute asthma exacerbation [11%]
- ✔ C. 42-year-old patient with aspirin toxicity [71%]
- D. 52-year-old patient with persistent vomiting [7%]
- E. 64-year-old patient with excessive diuresis [5%]

Omitted
Correct answer
C

71%
Answered correctly

3 Seconds
Time Spent

12/23/2018
Last Updated

Explanation

Explanation

Acid-base disorders	
Primary disorder	**Appropriate compensation**
Metabolic acidosis	$PaCO_2$ = 1.5 (serum HCO_3) + 8 ± 2
Metabolic alkalosis	↑ $PaCO_2$ by 0.7 mm Hg for every 1 mEq/L rise in serum HCO_3
Acute respiratory acidosis	↑ Serum HCO_3 by 1 mEq/L for every 10 mm Hg rise in $PaCO_2$
Acute respiratory alkalosis	↓ Serum HCO_3 by 2 mEq/L for every 10 mm Hg decrease in $PaCO_2$

This patient's arterial blood gas shows a normal pH (normal range 7.35-7.45). However, the abnormal $PaCO_2$ **and** HCO_3- suggest a mixed acid-base disorder. The first step is to determine the primary acid-base disorder. Primary respiratory alkalosis decreases arterial $PaCO_2$. This leads to metabolic (renal) compensation that decreases the serum HCO_3- by 2 mEq/L for every 10 mm Hg change in the $PaCO_2$. However, the compensatory mechanisms are not perfect and would not bring the pH to normal or overcorrect. If this patient had only an acute respiratory alkalosis, the serum HCO_3- should be 21 mEq/L (↓ of 3 mEq/L HCO_3- for 15 mm change in $PaCO_2$). Because the serum HCO_3- is much lower, this patient also has a concurrent metabolic acidosis.

Primary metabolic acidosis decreases the serum HCO_3-. The body compensates by increasing respirations to lower the $PaCO_2$. However, the pH remains in the acidemic range instead of normal. The Winter's formula (shown below) is used to determine appropriate respiratory compensation in metabolic acidosis. This patient's expected arterial $PaCO_2$ is 29±2 mm Hg (1.5 * [14] + 8). Because this patient's $PaCO_2$ is lower, there is a mixed metabolic acidosis and respiratory alkalosis (instead of physiologic metabolic or respiratory compensation).

appropriate respiratory compensation in metabolic acidosis. This patient's expected arterial $PaCO_2$ is 29±2 mm Hg (1.5 * [14] + 8). Because this patient's $PaCO_2$ is lower, there is a mixed metabolic acidosis and respiratory alkalosis (instead of physiologic metabolic or respiratory compensation).

$$\text{Arterial } PaCO_2 = 1.5\ (HCO_3\text{-}) + 8 \pm 2$$

Aspirin toxicity is the most likely cause of this patient's mixed respiratory alkalosis and metabolic acidosis. Aspirin (salicylate) directly stimulates the medullary respiratory center to cause tachypnea and respiratory alkalosis. In addition, aspirin causes an anion gap metabolic acidosis due to increased production and decreased renal elimination of organic acids (eg, lactic acid, ketoacids).

(Choice A) An allergic reaction with stridor indicates laryngeal edema. This would impair ventilation and cause CO_2 retention with resultant respiratory acidosis.

(Choice B) Asthma exacerbation usually leads to an acute respiratory alkalosis due to tachypnea. Most patients have a slightly decreased serum HCO_3-. However, this patient's serum HCO_3- is too low to account for metabolic compensation.

(Choices D and E) Persistent vomiting and excessive diuresis cause volume contraction with an increased serum HCO_3- and metabolic alkalosis. A patient with persistent vomiting would also most likely have hypochloremic metabolic alkalosis resulting from H+ losses in the gastric contents.

Educational objective:
Aspirin intoxication causes a mixed respiratory alkalosis and metabolic acidosis. Respiratory alkalosis is due to increased respiratory drive. Metabolic acidosis is due to increased production and decreased renal elimination of organic acids (eg, lactic acid, ketoacids).

References

- Acid-base disturbances in the salicylate-intoxicated adult.

A 28-year-old woman comes to the emergency department due to worsening headaches. She was first evaluated for headache 4 weeks ago. At that time, examination was unremarkable and her blood pressure was 132/86 mm Hg. The patient was advised to take ibuprofen. However, she continues to have worsening headache and reports fatigue. The patient has no fever, nausea, vomiting, abdominal pain, chest pain, or dyspnea. She was treated for sunburn on the face and arms 6 months ago and for sinus infection 4 weeks ago. The patient does not use tobacco, alcohol, or illicit drugs. Temperature is 37.2 C (99 F), blood pressure is 170/110 mm Hg, pulse is 82/min, and respirations are 14/min. Examination shows bilateral pitting ankle edema. Normal heart and vesicular breath sounds are heard on auscultation. The abdomen is soft and nontender. Laboratory results are as follows:

Complete blood count	
Hemoglobin	11 g/dL
Platelets	75,000/mm^3
Leukocytes	7,500/mm^3
Serum chemistry	
Blood urea nitrogen	40 mg/dL
Creatinine	2.5 mg/dL
Urinalysis	
Protein	3+
Bacteria	none

Protein	3+
Bacteria	none
Red blood cells	20-30/hpf
Casts	erythrocyte casts

Immunologic and rheumatologic studies

C3 (complement)	30 mg/dL (88-206 mg/dL)

Which of the following is the most likely diagnosis?

A. Drug-induced interstitial nephritis
B. Granulomatosis with angiitis
C. Hemolytic uremic syndrome
D. Hypertensive emergency
E. Poststreptococcal glomerulonephritis
F. Systemic lupus erythematosus

Submit

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Bacteria	none
Red blood cells	20-30/hpf
Casts	erythrocyte casts

Immunologic and rheumatologic studies

C3 (complement)	30 mg/dL (88-206 mg/dL)

Which of the following is the most likely diagnosis?

- A. Drug-induced interstitial nephritis [5%]
- B. Granulomatosis with angiitis [5%]
- C. Hemolytic uremic syndrome [5%]
- D. Hypertensive emergency [2%]
- E. Poststreptococcal glomerulonephritis [21%]
- ✔ F. Systemic lupus erythematosus [58%]

Omitted
Correct answer
F

58%
Answered correctly

5 Seconds
Time Spent

10/31/2018
Last Updated

Explanation

Systemic lupus erythematosus	
Clinical presentation	• Gradual symptom onset • Malar or discoid rash • Joint, renal, serosal &/or neurologic involvement
Laboratory abnormalities	• Anemia, leukopenia, thrombocytopenia • Positive **ANA**, **anti–double-stranded DNA**, anti-Smith • Low complement levels, increased immune complexes

ANA = antinuclear antibodies.

This patient's headache and constellation of findings are highly suggestive of **systemic lupus erythematosus** (SLE), an autoimmune disorder affecting **multiple organ systems**. Signs include:

- **Photosensitive skin** (sunburn)
- Thrombocytopenia
- **Glomerulonephritis** (renal failure with erythrocyte casts, proteinuria, hypertension) with significantly low complement (C3)

SLE can also affect the central nervous system, resulting in cognitive deficits, strokes, seizures, or headaches (due to vasculitis).

Immune complexes composed of double-stranded DNA (dsDNA) and anti-dsDNA antibodies deposit in the mesangium and/or subendothelial space. The immune complexes trigger an intense inflammatory reaction with activation of the complement system, **lowering C3 and C4** levels. Immune complexes may also deposit in the subepithelial space and cause membranous glomerulonephritis, presenting with nephrotic syndrome without hypocomplementemia.

(Choice A) Drug-induced interstitial nephritis can also present with acute kidney injury; however, urinalysis shows white blood cell

Immune complexes composed of double-stranded DNA (dsDNA) and anti-dsDNA antibodies deposit in the mesangium and/or subendothelial space. The immune complexes trigger an intense inflammatory reaction with activation of the complement system, **lowering C3 and C4** levels. Immune complexes may also deposit in the subepithelial space and cause membranous glomerulonephritis, presenting with nephrotic syndrome without hypocomplementemia.

(Choice A) Drug-induced interstitial nephritis can also present with acute kidney injury; however, urinalysis shows white blood cell casts and eosinophiluria. Patients may also have fever, rash, and eosinophilia. Thrombocytopenia and erythrocyte casts are not common.

(Choice B) Granulomatosis with angiitis (which commonly involves ear, nose, throat, and pulmonary manifestations) may also present with renal involvement and nephritic syndrome. However, the complement pathway is not activated and C3 level is normal.

(Choice C) Hemolytic uremic syndrome typically occurs after infection with Shiga-toxin-producing *Escherichia coli*. Patients present with microangiopathic hemolytic anemia, thrombocytopenia, and renal failure (generally from thrombotic angiopathy rather than glomerulonephritis). Erythrocyte casts are not typical. This patient has no risk factors for hemolytic uremic syndrome, and her other clinical features (eg, photosensitive rash) are more consistent with SLE.

(Choice D) Hypertensive emergency is a severe or sudden rise in blood pressure with evidence of end-organ damage (eg, renal failure, encephalopathy, myocardial ischemia). In cases of renal failure, hypocomplementemia is not seen.

(Choice E) Poststreptococcal glomerulonephritis also presents with hypertension, acute renal failure, erythrocyte casts in urine, and low serum C3 level. However, thrombocytopenia and photosensitive rash are not typical features.

Educational objective:
Systemic lupus erythematosus is an autoimmune disorder affecting multiple organ systems. Renal involvement may present with nephritic syndrome with low serum C3 and C4 levels. Nephrotic syndrome may also be seen.

A 52-year-old woman with known liver cirrhosis comes to the emergency department with bright red bleeding per rectum for 2 days. She also has increasing abdominal distension and discomfort. The patient has been on daily furosemide and spironolactone as well as water and sodium restriction. She had variceal banding after an episode of esophageal bleeding due to esophageal varices 3 months ago. The patient's temperature is 36.5 C (97.7 F), blood pressure is 91/55 mm Hg, pulse is 116/min, and respirations are 18/min. Her abdomen is distended and nontender with positive shifting dullness and has normal bowel sounds. There is 2+ peripheral pitting edema. Laboratory results are as follows:

Hemoglobin	8.2 g/dL
Leukocytes	4,200/mm^3
Serum sodium	128 mEq/L
Serum potassium	5.5 mEq/L
Serum chloride	103 mEq/L
Blood urea nitrogen	72 mg/dL
Serum creatinine	2.1 mg/dL

After blood transfusion and holding diuretics for a day, the patient's urine output decreases despite adequate intravenous volume resuscitation. Urine dipstick is negative for protein and blood. Urine sodium is 5 mEq/L. Which of the following is the most likely cause of this patient's acute kidney injury?

A. Glomerular disease
B. Interstitial nephritis
C. Obstructive uropathy

Hemoglobin	8.2 g/dL
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- A. Glomerular disease
- B. Interstitial nephritis
- C. Obstructive uropathy
- D. Renal hypoperfusion
- E. Renal vein thrombosis

Submit

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A. Glomerular disease [3%]
B. Interstitial nephritis [5%]
C. Obstructive uropathy [4%]

Leukocytes	4,200/mm^3
Serum sodium	128 mEq/L
Serum potassium	5.5 mEq/L
Serum chloride	103 mEq/L
Blood urea nitrogen	72 mg/dL
Serum creatinine	2.1 mg/dL

After blood transfusion and holding diuretics for a day, the patient's urine output decreases despite adequate intravenous volume resuscitation. Urine dipstick is negative for protein and blood. Urine sodium is 5 mEq/L. Which of the following is the most likely cause of this patient's acute kidney injury?

- A. Glomerular disease [3%]
- B. Interstitial nephritis [5%]
- C. Obstructive uropathy [4%]
- ✔ D. Renal hypoperfusion [82%]
- E. Renal vein thrombosis [5%]

Omitted
Correct answer
D

82%
Answered correctly

5 Seconds
Time Spent

09/11/2018
Last Updated

Explanation

Hepatorenal syndrome	
Risk factors	• Advanced cirrhosis with portal hypertension & edema
Precipitating factors	• Reduced renal perfusion • GI bleed, vomiting, sepsis, excessive diuretic use, SBP • Reduced glomerular pressure & GFR ◦ NSAID use (constricts afferent arterioles)
Diagnosis	• Renal hypoperfusion • FeNa <1% (or urine Na <10 mEq/L) • Absence of tubular injury • No RBC, protein, or granular casts in urine • No improvement in renal function with fluids
Treatment	• Address precipitating factors (eg, hypovolemia, anemia, infection) • Splanchnic vasoconstrictors (midodrine, octreotide, norepinephrine) • Liver transplantation

FeNa = fractional excretion of sodium; **GFR** = glomerular filtration rate; **GI** = gastrointestinal; **RBC** = red blood cells; **SBP** = spontaneous bacterial peritonitis.

The most likely diagnosis in this patient is renal hypoperfusion secondary to hepatorenal syndrome. Patients with severe liver cirrhosis have increased nitric oxide generation in the splanchnic circulation secondary to portal hypertension. This is thought to cause systemic vasodilation, which reduces peripheral vascular resistance and blood pressure, causing renal hypoperfusion.

The most likely diagnosis in this patient is renal hypoperfusion secondary to hepatorenal syndrome. Patients with severe liver cirrhosis have increased nitric oxide generation in the splanchnic circulation secondary to portal hypertension. This is thought to cause systemic vasodilation, which reduces peripheral vascular resistance and blood pressure, causing renal hypoperfusion. Reduced renal perfusion would then activate compensatory pathways (the renin-angiotensin-aldosterone system, sympathetic nervous system, and antidiuretic hormone) that increase water and sodium retention and worsen volume overload. Any factor that may further reduce glomerular capillary pressure (such as hypotension from gastrointestinal bleeding) causes an acute decline in glomerular filtration and can precipitate hepatorenal syndrome.

Laboratory results are similar to those of prerenal azotemia: elevated serum creatinine (>1.5 mg/dL) and a very low urine sodium level, typically <10 mEq/L. The urine sediment is usually bland (no red cells, casts, or protein), reflecting absence of intrinsic renal pathology. However, patients do not respond to intravenous fluids and withdrawal of diuretics, and renal function continues to decline.

(Choice A) Membranoproliferative glomerulonephritis is associated with hepatitis C infection. Glomerular disease is unlikely in this patient as the urine dipstick does not show protein or blood.

(Choice B) Interstitial nephritis usually presents with eosinophils or leukocytes in the urine. This patient's very low urine sodium makes a prerenal cause, such as hepatorenal syndrome, more likely than an intrinsic renal process such as interstitial nephritis.

(Choice C) Acute kidney injury is not seen in obstructive uropathy unless both ureters are obstructed.

(Choice E) Renal vein thrombosis usually occurs in patients with a predisposing condition, such as hypercoagulability, trauma, nephrotic syndrome, or severe dehydration. Acute renal vein thrombosis presents with abdominal pain and hematuria.

Educational objective:
Hepatorenal syndrome is seen in patients with severe liver cirrhosis secondary to systemic and renal hypoperfusion. Patients have acute renal failure (creatinine >1.5 mg/dL) with a very low urine sodium level, typically <10 mEq/L, and an absence of blood, casts, or protein in urine. Renal function does not improve with intravenous fluid resuscitation.

A 60-year-old male is hospitalized with generalized tonic-clonic seizures. He is a heavy alcohol user and he has had previous hospitalizations for alcohol withdrawal and alcohol-related seizures. On the second day of his hospitalization, his temperature is 37.1° C (98.9° F), blood pressure is 155/92 mmHg, pulse is 108/min, and respirations are 14/min. Examination shows no abnormalities. Laboratory studies show:

Serum sodium	140 mEq/L
Serum potassium	5.4 mEq/L
Bicarbonate	20 mEq/L
BUN	36 mg/dL
Serum creatinine	2.4 mg/dL
Urinalysis	
Glucose	Negative
Ketones	Trace
Leukocyte esterase	Negative
Blood	Large

Urine sediment microscopy shows 5-10 WBCs, 0-1 RBCs and some epithelial cells. Which of the following is the most likely diagnosis?

BUN	36 mg/dL
Serum creatinine	2.4 mg/dL
Urinalysis	
Glucose	Negative
Ketones	Trace
Leukocyte esterase	Negative
Blood	Large

Urine sediment microscopy shows 5-10 WBCs, 0-1 RBCs and some epithelial cells. Which of the following is the most likely diagnosis?

- A. Hepatorenal syndrome
- B. Hydronephrosis
- C. Post-infectious glomerulonephritis
- D. Renal artery stenosis
- E. Rhabdomyolysis

Submit

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Urine sediment microscopy shows 5-10 WBCs, 0-1 RBCs and some epithelial cells. Which of the following is the most likely diagnosis?

Urinalysis

Glucose	Negative
Ketones	Trace
Leukocyte esterase	Negative
Blood	Large

Urine sediment microscopy shows 5-10 WBCs, 0-1 RBCs and some epithelial cells. Which of the following is the most likely diagnosis?

- A. Hepatorenal syndrome [21%]
- B. Hydronephrosis [5%]
- C. Post-infectious glomerulonephritis [6%]
- D. Renal artery stenosis [6%]
- ✔ E. Rhabdomyolysis [59%]

Omitted
Correct answer
E

59%
Answered correctly

7 Seconds
Time Spent

12/16/2018
Last Updated

Omitted
Correct answer
E

59%
Answered correctly

7 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

This patient's serum creatinine is elevated, although without prior labs for comparison, it is difficult to determine if this abnormality is new or old. The most marked other abnormality is the presence of a large amount of blood on urinalysis, although there are only 0-1 RBCs seen on sediment microscopy. A large amount of myoglobin in the urine likely accounts for this discrepancy, secondary to rhabdomyolysis from the patient's recent tonic-clonic seizure. A standard urinalysis is not able to distinguish between hemoglobin and myoglobin due to chemical similarities, but microscopic exam of the urine for RBCs can add clarity. Large amounts of myoglobin in the urinary system can result in tubular injury and acute renal failure.

(Choice A) This patient could potentially have liver disease given his history of heavy alcohol use, although the patient's urinalysis results are more suggestive of rhabdomyolysis as the cause of his renal failure.

(Choice B) Hydronephrosis would not alone account for all of the abnormalities described above.

(Choice C) Post-infectious glomerulonephritis could present with hematuria, but this patient's absence of RBCs on urine microscopy is more consistent with myoglobinuria.

(Choice D) Renal artery stenosis can lead to renal failure, but it would be difficult to tie this in with the patient's tonic-clonic seizure.

Educational objective:
One should suspect myoglobinuria whenever test results demonstrate a large amount of blood on urinalysis with a relative absence of RBCs on urine microscopy. Myoglobinuria is usually caused by rhabdomyolysis, which frequently leads to acute renal failure.

A 54-year-old woman comes to the emergency department due to several hours of severe epigastric abdominal pain. The patient began having mild colicky upper abdominal pain after dinner last night and took acetaminophen before going to bed. In the night, the patient awoke with severe abdominal pain radiating to her back. She has also had nausea and several episodes of vomiting. The patient has a history of occasional upper abdominal pain after eating but has never had such severe symptoms. She has no other medical issues and takes no medications. Temperature is 37.6 C (99.6 F), blood pressure is 110/66 mm Hg, pulse is 118/min, and respirations are 24/min. The patient appears to be in moderate distress. Mucous membranes are dry. The abdomen is distended with marked epigastric tenderness. Bowel sounds are decreased. Laboratory results are as follows:

Complete blood count	
Hematocrit	48%
Leukocytes	18,800/mm³
Liver function studies	
Total bilirubin	2.2 mg/dL
Alkaline phosphatase	370 U/L
Lipase	2,192 U/L

Which of the following sets of renal findings are most expected in this patient?

	Renin secretion	Efferent arteriolar resistance	Tubular sodium reabsorption
○ A.	↑	↑	↓

Leukocytes	18,800/mm³

Liver function studies

Total bilirubin	2.2 mg/dL
Alkaline phosphatase	370 U/L
Lipase	2,192 U/L

Which of the following sets of renal findings are most expected in this patient?

	Renin secretion	Efferent arteriolar resistance	Tubular sodium reabsorption
A.	↑	↑	↓
B.	↑	↑	↑
C.	↑	↓	↑
D.	↓	↑	↓
E.	↓	↓	↓
F.	↓	↓	↑

Submit

A 54-year-old woman comes to the emergency department due to several hours of severe epigastric abdominal pain. The patient began having mild colicky upper abdominal pain after dinner last night and took acetaminophen before going to bed. In the night, the patient awoke with severe abdominal pain radiating to her back. She has also had nausea and several episodes of vomiting. The patient has a history of occasional upper abdominal pain after eating but has never had such severe symptoms. She has no other medical issues and takes no medications. Temperature is 37.6 C (99.6 F), blood pressure is 110/66 mm Hg, pulse is 118/min, and respirations are 24/min. The patient appears to be in moderate distress. Mucous membranes are dry. The abdomen is distended with marked epigastric tenderness. Bowel sounds are decreased. Laboratory results are as follows:

Complete blood count

Hematocrit	48%
Leukocytes	18,800/mm³

Liver function studies

Total bilirubin	2.2 mg/dL
Alkaline phosphatase	370 U/L
Lipase	2,192 U/L

Which of the following sets of renal findings are most expected in this patient?

	Renin secretion	Efferent arteriolar resistance	Tubular sodium reabsorption
A.	↑	↑	↓

Lipase	2,192 U/L

Which of the following sets of renal findings are most expected in this patient?

	Renin secretion	Efferent arteriolar resistance	Tubular sodium reabsorption
A. [4%]	↑	↑	↓
✔ B. [74%]	↑	↑	↑
C. [16%]	↑	↓	↑
D. [1%]	↓	↑	↓
E. [2%]	↓	↓	↓
F. [0%]	↓	↓	↑

Omitted
Correct answer
B

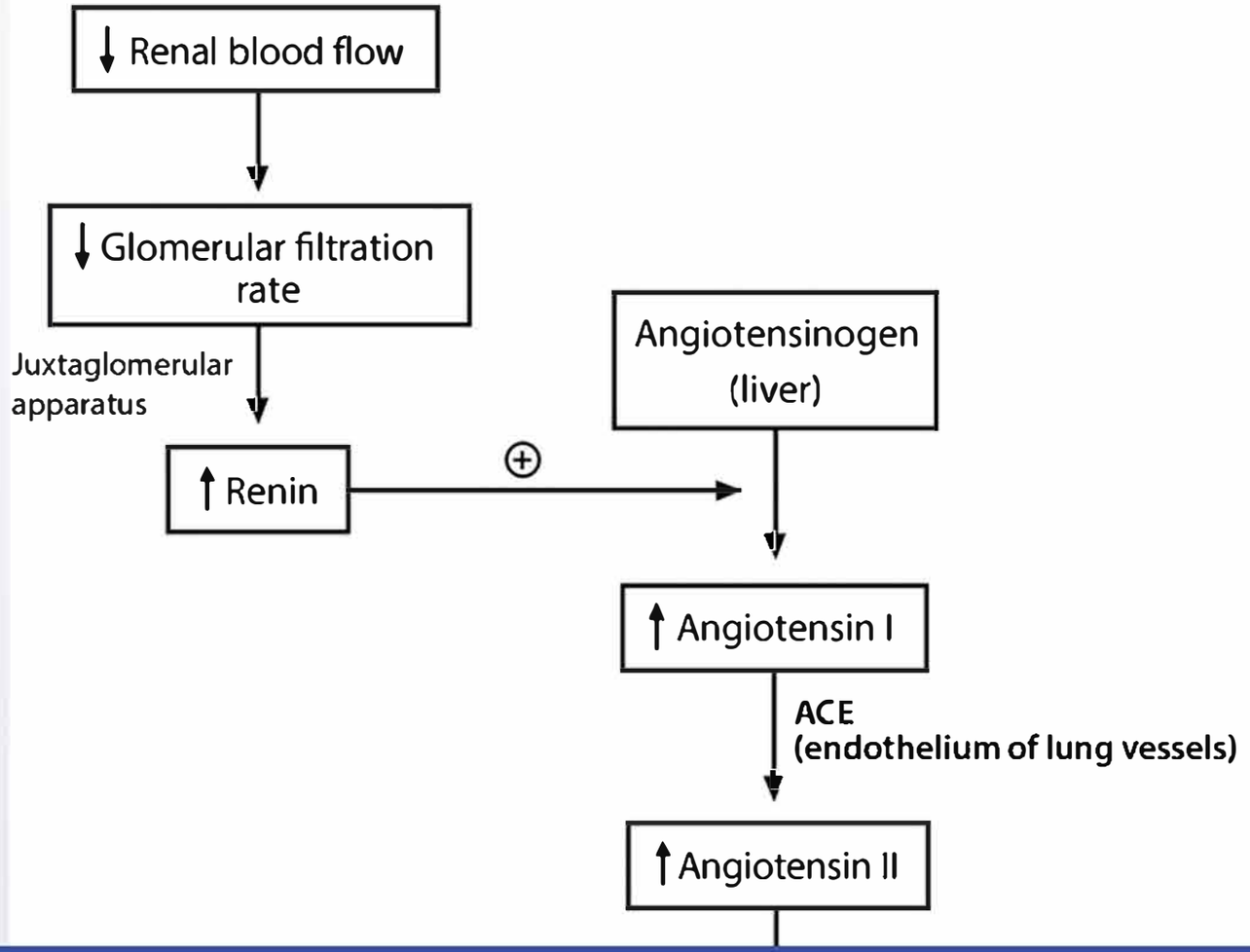
74%
Answered correctly

5 Seconds
Time Spent

07/28/2018
Last Updated

Explanation

Renin-angiotensin system



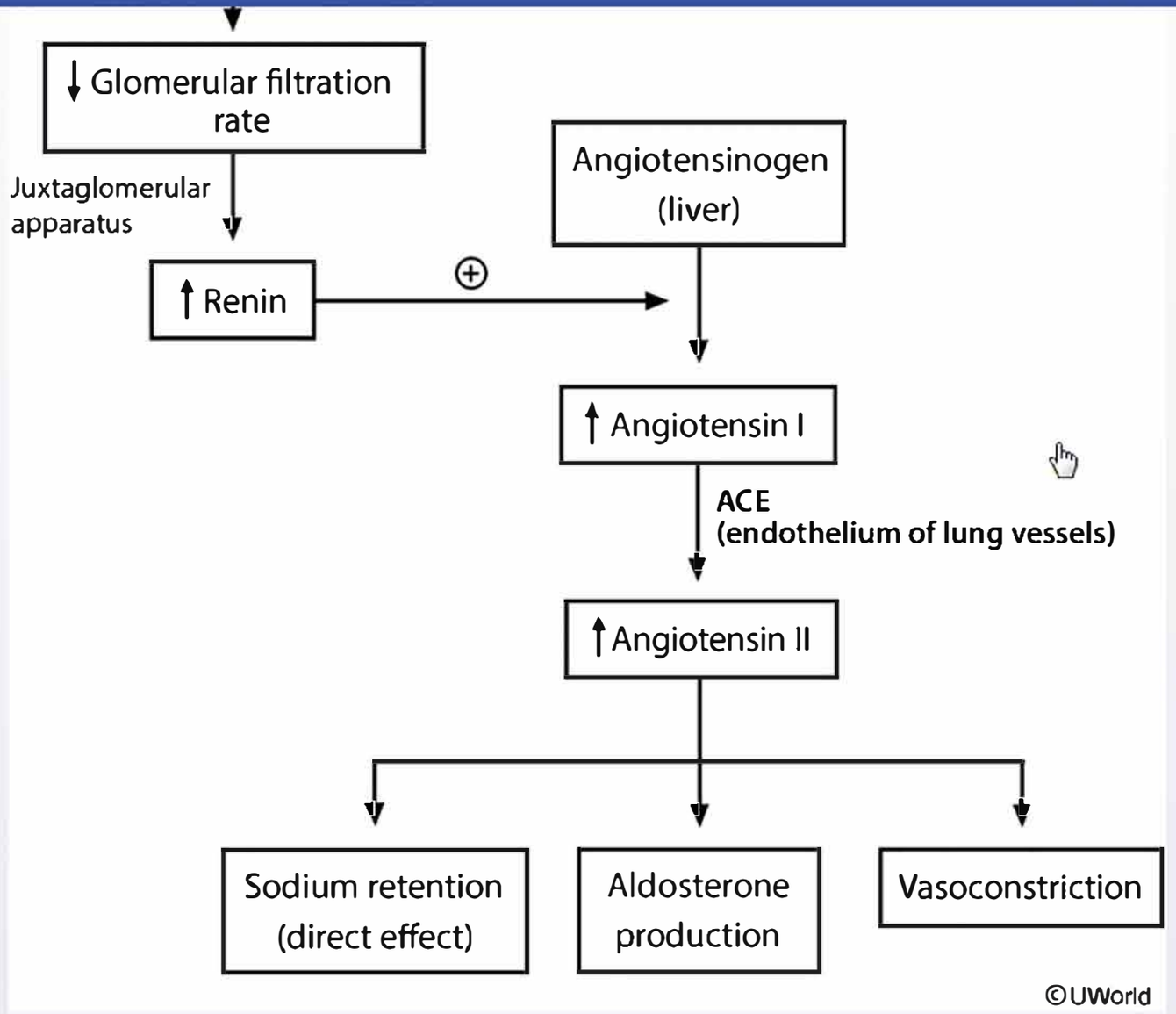
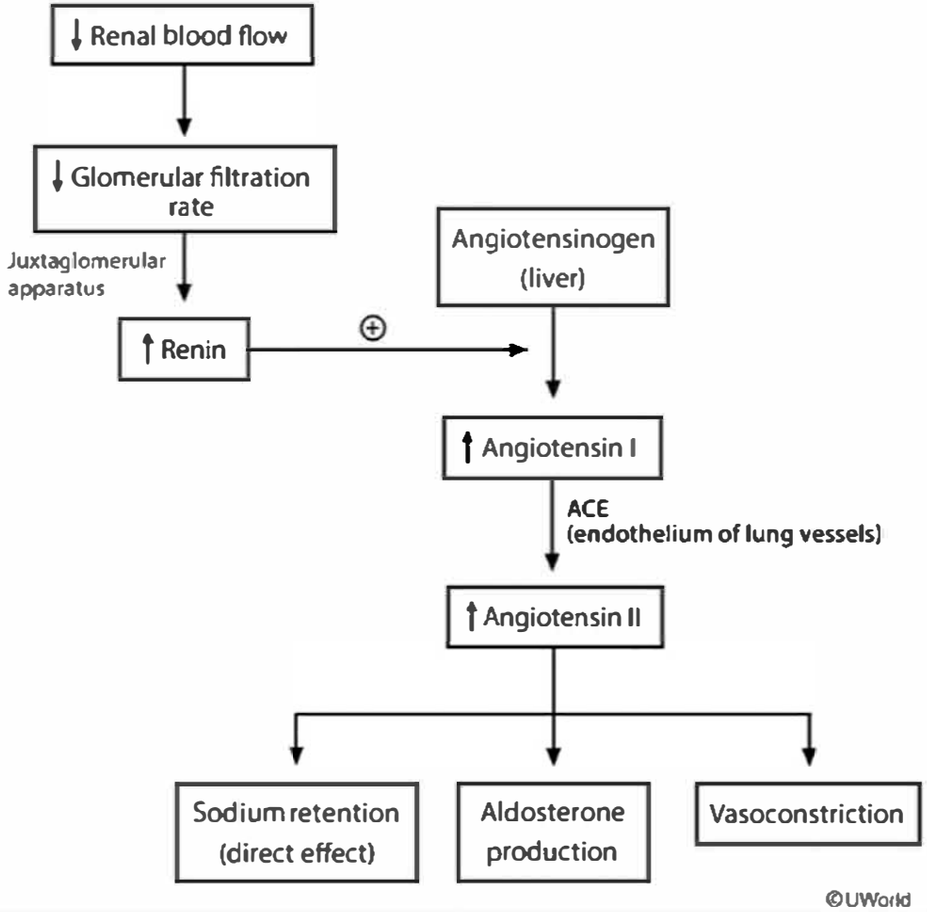

↓ Glomerular filtration rate
Juxtaglomerular apparatus
↑ Renin
⊕
Angiotensinogen (liver)
↑ Angiotensin I
ACE
(endothelium of lung vessels)
↑ Angiotensin II
Sodium retention (direct effect)
Aldosterone production
Vasoconstriction
©UWorld

Exhibit Display

Renin-angiotensin system



Zoom In | Zoom Out | Reset | Add To Flash Card

This patient has a history of colicky abdominal pain after eating, which raises suspicion for gallstones. She then developed clinical manifestations (severe epigastric pain, vomiting, elevated lipase) consistent with **acute gallstone pancreatitis**.

Patients with pancreatitis typically develop severe **hypovolemia** due to vomiting, inability to tolerate oral fluids, and third-space extravasation. Manifestations include dry mucous membranes, hemoconcentration, tachycardia, hypotension, and/or shock. The kidneys respond by **releasing renin** from the juxtaglomerular cells. Renin converts angiotensinogen to angiotensin I, which is then converted into angiotensin II by ACE. Angiotensin II has the following systemic effects:

- **Vasoconstriction:** Increases efferent and systemic arteriolar resistance, which improves glomerular filtration rate and blood pressure
- **Sodium and water reabsorption:** Directly increases proximal tubule sodium reabsorption
- **Aldosterone secretion:** Increases sodium and water reabsorption in the distal tubule

Therefore, this patient is likely to have increased renin, efferent arteriolar resistance, and tubular sodium reabsorption.

Educational objective:
Hypovolemia stimulates the renin-angiotensin system, which helps improve intravascular volume, glomerular filtration rate, and blood pressure via vasoconstriction and kidney-mediated sodium and water reabsorption.

References

- Fluid resuscitation in acute pancreatitis.

A 34-year-old man is being evaluated for possible end-stage renal disease. He has a long history of diabetes, type 1. He previously developed chronic renal insufficiency despite being on enalapril and insulin. His renal function is getting worse day by day. A nephrologist is currently managing his renal condition. Which of the following long-term treatments would give the best survival rate for this patient?

A. Hemodialysis
B. Peritoneal dialysis
C. Renal transplantation from a cadaver
D. Renal transplantation from a living related donor
E. Renal transplantation from a living unrelated donor

Submit

A 34-year-old man is being evaluated for possible end-stage renal disease. He has a long history of diabetes, type 1. He previously developed chronic renal insufficiency despite being on enalapril and insulin. His renal function is getting worse day by day. A nephrologist is currently managing his renal condition. Which of the following long-term treatments would give the best survival rate for this patient?

- A. Hemodialysis [5%]
- B. Peritoneal dialysis [1%]
- C. Renal transplantation from a cadaver [1%]
- ✔ D. Renal transplantation from a living related donor [89%]
- E. Renal transplantation from a living unrelated donor [2%]

Omitted
Correct answer
D

89%
Answered correctly

3 Seconds
Time Spent

10/10/2018
Last Updated

Explanation

End stage renal disease is a progressive condition that is fatal if left untreated. Once end stage renal disease develops, there are only two treatment options available: dialysis or renal transplantation. The choice depends on the patient and co-morbid conditions; however, if both options are available, renal transplantation is preferred, as it is associated with better survival and quality of life.

The advantages of renal transplantation over dialysis are:

Explanation

End stage renal disease is a progressive condition that is fatal if left untreated. Once end stage renal disease develops, there are only two treatment options available: dialysis or renal transplantation. The choice depends on the patient and co-morbid conditions; however, if both options are available, renal transplantation is preferred, as it is associated with better survival and quality of life.

The advantages of renal transplantation over dialysis are:

1. Better survival and quality of life.
2. Anemia, bone disease, and hypertension persist in spite of dialysis; these are better controlled with transplantation.
3. Transplant patients have a return of normal endocrine, sexual, and reproductive functions, and enhanced energy levels; thus, returning to fulltime employment and more strenuous physical activity is possible.
4. In diabetics, autonomic neuropathy persists or worsens after dialysis; whereas, it stabilizes or improves with transplantation.
5. Expected survival rate after transplantation is 95% at one year and 88% at five years.

The major disadvantages of renal transplantation are difficulty in finding a donor, surgical risk and cost, and side effects of immunosuppression. Transplantation from a living related donor has the least graft rejection and best graft survival, followed by a living non-related donor, and cadaver graft **(Choices C and E).**

(Choices A and B) Dialysis options include hemodialysis (home or in-center) or peritoneal dialysis (chronic ambulatory or cyclic peritoneal dialysis). In the US, 85% of patients have in-center hemodialysis, 15% have peritoneal dialysis, and approximately 1% have home hemodialysis. The choice depends on the patient. Peritoneal hemodialysis provides the patient with more control and mobility, but the risk of peritonitis is high. The five-year survival rate in non-diabetic patients who are on dialysis is 30-40%; whereas, in diabetics, it is 20%.

Educational Objective:
Patients with end stage renal disease have only two treatment options: dialysis or renal transplantation. Renal transplantation is

A 70-year-old man comes to the physician complaining of 4-6 months of almost continuous urinary dribbling. It occurs during the day and at night and is progressively worsening. He has no dysuria or hematuria. The patient has a 20-year history of type 2 diabetes, hypertension, alcoholic hepatitis, and coronary artery disease. He had a gastric emptying study done a few weeks ago due to continuous nausea and early satiety. He had laser photocoagulation of both eyes for diabetic retinopathy. The patient has a 50-pack-year smoking history and drinks 4-6 beers daily. His medications include insulin glargine, lispro insulin, metformin, aspirin, metoprolol, lisinopril, and erythromycin. Physical examination shows a normal-size prostate, decreased sensation in both legs below the knees, and absent Achilles tendon and knee reflexes bilaterally. Postvoid residual volume is 550 mL.

Urinalysis results are as follows:

Specific gravity	1.020
Blood	Trace
Glucose	Positive
Ketones	Negative
Protein	Moderate
Leukocyte esterase	Negative
Nitrites	Negative
White blood cells	1-2/hpf
Red blood cells	3-4/hpf

Which of the following is the most likely cause of this patient's incontinence?

A 70-year-old man comes to the physician complaining of 4-6 months of almost continuous urinary dribbling. It occurs during the day and at night and is progressively worsening. He has no dysuria or hematuria. The patient has a 20-year history of type 2 diabetes, hypertension, alcoholic hepatitis, and coronary artery disease. He had a gastric emptying study done a few weeks ago due to continuous nausea and early satiety. He had laser photocoagulation of both eyes for diabetic retinopathy. The patient has a 50-pack-year smoking history and drinks 4-6 beers daily. His medications include insulin glargine, lispro insulin, metformin, aspirin, metoprolol, lisinopril, and erythromycin. Physical examination shows a normal-size prostate, decreased sensation in both legs below the knees, and absent Achilles tendon and knee reflexes bilaterally. Postvoid residual volume is 550 mL.

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Red blood cells	3-4/hpf

Which of the following is the most likely cause of this patient's incontinence?

A. Diabetic nephropathy

Leukocyte esterase	Negative
Nitrites	Negative
White blood cells	1-2/hpf
Red blood cells	3-4/hpf

Which of the following is the most likely cause of this patient's incontinence?

- A. Diabetic nephropathy
- B. Neurogenic bladder dysfunction
- C. Overflow incontinence due to medication
- D. Overflow incontinence from bladder outlet obstruction
- E. Pelvic floor muscle weakness
- F. Urethral instability
- G. Urinary diverticulum
- H. Urinary fistula
- I. Urinary tract infection

Submit

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Nitrites	Negative
White blood cells	1-2/hpf
Red blood cells	3-4/hpf

Which of the following is the most likely cause of this patient's incontinence?

- A. Diabetic nephropathy [9%]
- ✔ B. Neurogenic bladder dysfunction [82%]
- C. Overflow incontinence due to medication [2%]
- D. Overflow incontinence from bladder outlet obstruction [4%]
- E. Pelvic floor muscle weakness [0%]
- F. Urethral instability [0%]
- G. Urinary diverticulum [0%]
- H. Urinary fistula [0%]
- I. Urinary tract infection [0%]

Omitted
Correct answer
B

82%
Answered correctly

4 Seconds
Time Spent

11/30/2018
Last Updated

Clinical features of diabetic autonomic neuropathy	
Cardiovascular	• Tachycardia, impaired exercise tolerance • Postural hypotension with loss of diurnal blood pressure variation
Peripheral nerves	• Dry skin, pruritus, callus formation • Foot ulcers & poor wound healing • Charcot arthropathy (increased fracture risk with resultant secondary ulceration)
Gastrointestinal	• Gastroparesis with delayed gastric emptying • Esophageal dysmotility with possible dyspepsia • Intestinal involvement with possible diarrhea, constipation, or fecal incontinence
Genitourinary	• Erectile dysfunction & retrograde ejaculation in men, decreased libido & dyspareunia in women • Decreased ability to sense full bladder leading to incomplete emptying & decreased urination • Eventual recurrent urinary tract infections &/or overflow incontinence (eg, dribbling, poor urinary stream)

This patient's presentation is consistent with likely diabetic autonomic neuropathy (DAN) affecting the genitourinary tract. Risk factors

This patient's presentation is consistent with likely diabetic autonomic neuropathy (DAN) affecting the genitourinary tract. Risk factors for DAN include poor glucose control and other vascular risk factors (eg, hypertension, elevated triglycerides, smoking, obesity). This patient's microvascular complications (peripheral neuropathy, retinopathy, and nephropathy) indicate poorly controlled diabetes.

Parasympathetic innervation of the bladder regulates detrusor muscle contraction and internal sphincter relaxation to allow for voiding urine. DAN can cause a neurogenic bladder with decreased ability to sense a full bladder, incomplete emptying, urinary retention, and distended bladder. Patients with a higher bladder than urethral pressure develop overflow incontinence and lose urine until the pressures equalize. The symptoms can occur cyclically both day and night. Physical examination may show a distended bladder with high post-void residual urine volume (>50 mL).

(Choice A) This patient's urinalysis showing proteinuria suggests glomerular disease due to diabetic nephropathy. However, diabetic nephropathy does not typically cause urinary incontinence.

(Choice C) Medications (eg, anticholinergics, antipsychotics, tricyclic antidepressants, sedative-hypnotics) can block the parasympathetic pathways that initiate micturition and lead to overflow incontinence. However, this patient's current medications are not usually associated with incontinence.

(Choice D) Bladder outlet obstruction due to an enlarged prostate is the most common cause of overflow incontinence in men. This patient's normal prostate exam makes this less likely than a neurogenic cause, especially in the setting of retinopathy, neuropathy and nephropathy.

(Choice E) Pelvic floor weakness commonly causes stress incontinence in women. Patients develop incontinence due to increased intraabdominal pressure after coughing or sneezing. Stress incontinence can occur in older men after a radical or transurethral prostatectomy. However, patients with this condition usually have normal post-void residual volume.

(Choice F) Urethral instability refers to involuntary fluctuations in the urethral pressure with or without urinary incontinence. This can occur even in normal individuals. However, it usually does not cause urinary retention or a high post-void residual volume.

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(Choice F) Urethral instability refers to involuntary fluctuations in the urethral pressure with or without urinary incontinence. This can occur even in normal individuals. However, it usually does not cause urinary retention or a high post-void residual volume.

(Choice G) Urinary diverticulum is an outpouching of the bladder or urethra into the adjacent tissues. It is more common in women than men. Patients usually develop post-void dribbling, dysuria, and dyspareunia. The urinary stasis can lead to possible recurrent cystitis/urinary tract infection but does not cause a high post-void residual volume.

(Choice H) Urinary fistula is a communication between the urethra/bladder and uterus or vagina. It occurs more commonly in women after pelvic surgery. Patients develop continuous leakage of urine without significant urinary retention.

(Choice I) Urinary tract infection may cause incontinence without typical dysuria in patients with neuropathy, but patients will still have an abnormal urinalysis (eg, pyuria, positive nitrite/leukocyte esterase) and sometimes systemic symptoms (eg fever, chills).

Educational objective:
Diabetic autonomic neuropathy can affect the genitourinary tract to cause a neurogenic bladder with urinary retention and distended bladder. Patients can then develop overflow incontinence (eg, dribbling, poor urinary stream) with a high post-void residual volume.

References

- Bladder dysfunction in diabetes mellitus.

A 45-year-old man comes to the emergency department with severe right flank pain. He is tossing in the bed from the pain. He has a history of hypertension, diet-controlled type 2 diabetes mellitus, and lower-extremity joint pains. Abdominal radiographs show no abnormalities, but CT scan shows a 5-mm stone in the right ureter and several smaller stones in both renal pelvices. Urinalysis results are as follows:

Urine pH	4.5 (normal 5-6)
White blood cells	Absent
Red blood cells	50-100/hpf
Bacteria	Absent
Nitrites	Negative
Esterase	Negative

The patient subsequently passes a stone and analysis shows it to be composed of 100% uric acid. Besides high fluid intake, which of the following would be most helpful for this patient to prevent recurrent stones?

A. Calcium-restricted diet
B. Furosemide
C. High-protein diet
D. Hydrochlorothiazide
E. Potassium citrate

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Bacteria	Absent
Nitrites	Negative
Esterase	Negative

The patient subsequently passes a stone and analysis shows it to be composed of 100% uric acid. Besides high fluid intake, which of the following would be most helpful for this patient to prevent recurrent stones?

- A. Calcium-restricted diet [3%]
- B. Furosemide [12%]
- C. High-protein diet [2%]
- D. Hydrochlorothiazide [28%]
- ✔ E. Potassium citrate [53%]

Omitted

Omitted
Correct answer
E

53%
Answered correctly

4 Seconds
Time Spent

10/23/2018
Last Updated

Explanation

There are 3 likely possibilities when a patient has symptoms consistent with typical renal colic but no stones are identified on conventional radiographs:

1. Radiolucent stones (uric acid stones, xanthine stones)
2. Calcium stones <1-3 mm in diameter
3. Non-stone ureteral obstruction (eg, blood clot, tumor)

This patient has uric acid stones, which account for approximately 10%-15% of nephrolithiasis cases. These are most commonly seen in patients with unusually low urine pH (which may be due to a defect in renal ammonia excretion) and hyperuricosuria. Uric acid stones are radiolucent but can often be seen on renal ultrasound or CT scan.

Treatment of uric acid stones includes hydration, **alkalinization** of the urine, and a low-purine diet. Alkalinization of the urine to pH 6.0- 6.5 with oral potassium citrate is recommended as uric acid stones are highly soluble in alkaline urine. In addition to alkalinizing the urine, citrate is a stone inhibitor and reduces crystallization. Allopurinol can be added if there are recurrent symptoms despite initial measures, especially if hyperuricosuria or hyperuricemia occurs.

(Choice A) A calcium-restricted diet is not beneficial in the management of uric acid stones. It is not advised even in patients with calcium stones, as calcium restriction can potentially cause a negative calcium balance and hyperoxaluria due to the resulting increase in gastrointestinal absorption of oxalate.

(Choice B) Furosemide increases urinary calcium excretion, and the resulting hypercalciuria can increase the risk of calcium stone

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(Choice B) Furosemide increases urinary calcium excretion, and the resulting hypercalciuria can increase the risk of calcium stone formation.

(Choice C) A purine-restricted (not high-protein) diet is indicated in patients with uric acid stones secondary to hyperuricosuria.

(Choice D) Hydrochlorothiazide decreases urinary calcium excretion and is used in the management of recurrent hypercalciuric renal stones.

Educational objective:
Uric acid stones are often radiolucent but may be seen on ultrasound or CT scan. They are highly soluble in alkaline urine; alkalinization of the urine to pH 6.0-6.5 with oral potassium citrate is the treatment of choice.

References

- Uric acid stones and hyperuricosuria.

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A 58-year-old man with Burkitt lymphoma is admitted to the hospital for chemotherapy. Medical history includes hypertension, stable angina, and hyperlipidemia. Two days after starting chemotherapy, the patient reports nausea and generalized weakness. Laboratory results are as follows:

Serum chemistry	
Sodium	140 mEq/L
Potassium	6.8 mEq/L
Bicarbonate	18 mEq/L
Blood urea nitrogen	24 mg/dL
Creatinine	1.6 mg/dL
Calcium	8 mg/dL
Glucose	160 mg/dL

ECG shows peaked T waves. Which of the following interventions should be employed next to rapidly lower serum potassium in this patient?

A. Calcium carbonate [22%]
B. Furosemide [2%]
C. Hemodialysis [11%]
D. High-dose inhaled beta-2 agonist [3%]
E. Insulin and glucose [56%]

Potassium	6.6 mEq/L
Bicarbonate	18 mEq/L
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- E. Insulin and glucose
- F. Normal saline
- G. Sodium polystyrene sulfonate

Submit

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- D. High-dose inhaled beta-2 agonist [3%]
- ✔ E. Insulin and glucose [56%]
- F. Normal saline [2%]
- G. Sodium polystyrene sulfonate [1%]

Omitted
Correct answer
E

56%
Answered correctly

4 Seconds
Time Spent

11/22/2018
Last Updated

Clinical features of hyperkalemia	
Sequence of ECG changes	• Tall **peaked T waves** with shortened QT interval • **PR prolongation & QRS widening** • Disappearance of P wave • Conduction blocks, ectopy, or **sine wave** pattern
Cardiac membrane stabilization	• **Calcium** infusion
Rapidly acting treatment options	• Insulin with glucose • Beta-2 adrenergic agonists • Sodium bicarbonate
Removal of potassium from the body (slow-acting)	• Diuretics • Cation exchange resins • Hemodialysis

This patient with Burkitt lymphoma has **hyperkalemia** likely due to tumor lysis syndrome from chemotherapy. Indications for emergent treatment of hyperkalemia include presence of hyperkalemia-related ECG changes or marked elevation (>6.5 mEq/L) or rapid rise in serum potassium level. Hyperkalemia is life threatening due to its adverse **cardiac effects**, including significant bradycardia (sinus node dysfunction, atrioventricular block) and ventricular arrhythmias. Goals of treatment include stabilizing the cardiac membrane, lowering serum potassium levels by shifting potassium intracellularly, and lowering total body potassium content.

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Serum potassium may be lowered faster by stimulating an intracellular potassium shift than by removing potassium from the body. **Insulin** (administered intravenously) can move potassium intracellularly within **minutes**. Glucose is given along with insulin to avoid hypoglycemia. Inhaled beta agonists are also rapidly acting agents that shift potassium intracellularly. However, their use in patients with active coronary artery disease (eg, stable angina) can cause tachycardia and precipitate angina. Therefore, insulin is the preferred agent in this patient **(Choice D)**.

Intracellular shifts are usually transient, so efforts to decrease total body potassium must still be made, although this occurs over a longer time frame.

(Choice A) Administration of intravenous calcium chloride or gluconate (not calcium carbonate, which is used as an oral calcium supplement) helps stabilize the cardiac membrane, making it resistant to the effects of hyperkalemia. However, calcium has no effect on serum potassium level.

(Choice B) Furosemide is a loop diuretic that promotes renal potassium excretion. Onset of action is delayed by approximately 30 minutes.

(Choice C) Hemodialysis is the most definitive way to remove potassium from the body in patients with renal failure. However, the process often takes time to prepare (eg, catheter placement, dialysis machine setup).

(Choice F) Normal saline may be used to manage prerenal azotemia with or without hyperkalemia. Normal saline has no direct effect on serum potassium.

(Choice G) Sodium polystyrene sulfonate is a potassium-binding resin that decreases total body potassium content. Sodium is exchanged for potassium in the gut. Sodium polystyrene sulfonate takes at least 1-2 hours to take effect.

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Educational objective:
Hyperkalemia is a medical emergency. Therapy involves 3 goals: Stabilizing the cardiac membrane with calcium, shifting potassium intracellularly, and decreasing the total body potassium content. Insulin/glucose administration is the quickest way to lower serum potassium concentration.

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(Choice C) Hemodialysis is the most definitive way to remove potassium from the body in patients with renal failure. However, the

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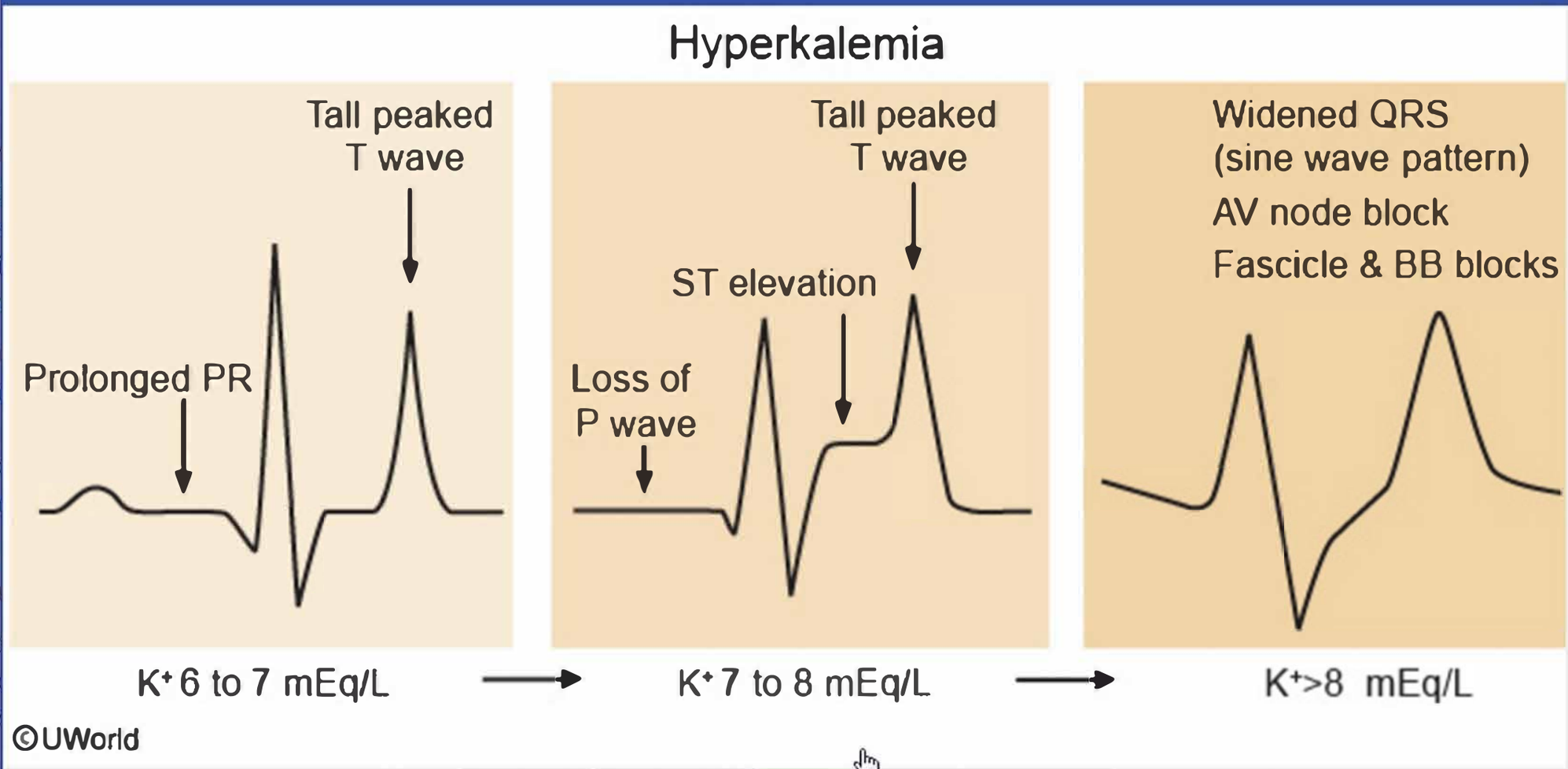

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A 25-year-old woman comes to the physician complaining of a 3-day history of burning with urination and increased urinary frequency. She has suprapubic discomfort but no unusual vaginal discharge. The patient has been sexually active and monogamous with her husband for the past 4 years. She has never been pregnant and uses condoms for contraception. Her last menstrual period was 2 weeks ago, and her last sexual encounter was 2 days ago. She takes no medications and has no known drug allergies. Her temperature is 37.1 C (98.9 F), blood pressure is 110/70 mm Hg, pulse is 68/min, and respirations are 15/min. Examination shows suprapubic tenderness without flank tenderness. The rest of the examination is within normal limits. Her urine pregnancy test is negative. Laboratory results are as follows:

Urinalysis

Specific gravity	1.020
Blood	Trace
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Positive
Nitrites	Positive

Which of the following is the most appropriate next step in management of this patient?

A. Intramuscular ceftriaxone and oral doxycycline
B. Oral amoxicillin
C. Oral levofloxacin

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B. Oral amoxicillin
C. Oral levofloxacin
D. Oral nitrofurantoin
E. Urine culture

Submit

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Urinalysis

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Blood	Trace
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Leukocyte esterase	Positive
Nitrites	Positive

Which of the following is the most appropriate next step in management of this patient?

A. Intramuscular ceftriaxone and oral doxycycline [1%]
B. Oral amoxicillin [5%]
C. Oral levofloxacin [16%]

Urinalysis

Specific gravity	1.020
Blood	Trace
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Positive
Nitrites	Positive

Which of the following is the most appropriate next step in management of this patient?

- A. Intramuscular ceftriaxone and oral doxycycline [1%]
- B. Oral amoxicillin [5%]
- C. Oral levofloxacin [16%]
- ✔ D. Oral nitrofurantoin [60%]
- E. Urine culture [15%]

Omitted
Correct answer
D

60%
Answered correctly

5 Seconds
Time Spent

09/12/2018
Last Updated

Explanation

Treatment of acute cystitis & pyelonephritis in nonpregnant women	
Uncomplicated cystitis	• **Nitrofurantoin** for 5 days (avoid in suspected pyelonephritis or creatinine clearance <60 mL/min) • **Trimethoprim-sulfamethoxazole** for 3 days (avoid if local resistance rate >20%) • **Fosfomycin** single dose • Fluoroquinolones only if above options cannot be used • Urine culture needed only if initial treatment fails
Complicated cystitis*	• **Fluoroquinolones**** (5-14 days), extended-spectrum antibiotic (eg, ampicillin/gentamicin) for more severe cases • Obtain sample for urine culture prior to initiating therapy & adjust antibiotic as needed
Pyelonephritis	• **Outpatient:** Fluoroquinolones (eg, ciprofloxacin, levofloxacin) • **Inpatient:** Intravenous antibiotics (eg, fluoroquinolone, aminoglycoside ± ampicillin) • Obtain sample for urine culture prior to initiating therapy & adjust antibiotic as needed

*Associated with diabetes, pregnancy, renal failure, urinary tract obstruction, indwelling catheter, urinary procedure (eg, cystoscopy), immunosuppression & hospital-acquired.

**Do not use fluoroquinolones in pregnancy. Consider cefpodoxime, cephalexin, amoxicillin-clavulanate & fosfomycin.

This patient's clinical presentation (dysuria, urinary frequency, suprapubic tenderness) suggests **uncomplicated cystitis**. Uncomplicated cystitis commonly occurs in otherwise healthy patients and has a **low risk** of treatment failure. In patients with suggestive clinical findings (eg, dysuria, urgency), urinalysis will confirm the diagnosis and patients can be treated without a urine culture. Culture is reserved for those who fail initial therapy **(Choice E)**. Preferred treatment options include trimethoprim-

This patient's clinical presentation (dysuria, urinary frequency, suprapubic tenderness) suggests **uncomplicated cystitis**. Uncomplicated cystitis commonly occurs in otherwise healthy patients and has a **low risk** of treatment failure. In patients with suggestive clinical findings (eg, dysuria, urgency), urinalysis will confirm the diagnosis and patients can be treated without a urine culture. Culture is reserved for those who fail initial therapy **(Choice E)**. Preferred treatment options include trimethoprim-sulfamethoxazole (3 days), nitrofurantoin (5 days), and fosfomycin (single dose). Fluoroquinolones are also effective but are typically reserved for patients who cannot take the above options (eg, sulfa allergy) or for settings with high local resistance rates **(Choice C)**.

Complicated cystitis refers to infections associated with factors that increase the risk of antibiotic resistance or treatment failure. Such factors include diabetes, chronic kidney disease, pregnancy, immunocompromised state, or urinary tract obstruction; hospital-acquired infection; or infection associated with a procedure (eg, cystoscopy) or indwelling foreign body (eg, catheter, stent). These patients should have **urine culture** prior to therapy. Complicated cystitis in otherwise stable patients may be managed with oral **fluoroquinolones**, but more severe cases may require intravenous broad-spectrum antibiotics (eg, ceftriaxone) while awaiting culture results.

Pyelonephritis (ie, urinary tract infection with fever and flank pain/tenderness) also requires urine culture prior to starting treatment. Stable patients with uncomplicated pyelonephritis can be treated with oral antibiotics (usually a fluoroquinolone), but unstable patients and those with complicated infection require intravenous antibiotics (eg, ceftriaxone).

(Choice A) Intramuscular ceftriaxone with oral doxycycline is often used empirically for treating suspected chlamydial and/or gonococcal infections. Doxycycline is not reliably effective for cystitis, and oral medications are preferred over parenteral for uncomplicated cystitis.

(Choice B) Amoxicillin is not recommended for empiric treatment of cystitis due to antibiotic resistance. Amoxicillin/clavulanate may be considered but is associated with significant side effects (eg, diarrhea).

Educational objective:
Uncomplicated cystitis commonly occurs in otherwise healthy patients and has a low risk of treatment failure. Urinalysis confirms the

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Educational objective:
Uncomplicated cystitis commonly occurs in otherwise healthy patients and has a low risk of treatment failure. Urinalysis confirms the diagnosis. Patients can be treated without a urine culture, which may be done later in those who fail initial therapy. Oral trimethoprim/sulfamethoxazole, nitrofurantoin, and fosfomycin are effective first-line treatment options.

References

- Nitrofurantoin compares favorably to recommended agents as empirical treatment of uncomplicated urinary tract infections in a decision and cost analysis.
- International clinical practice guidelines for the treatment of acute uncomplicated cystitis and pyelonephritis in women: A 2010 update by the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases.

A 26-year-old man comes to the emergency department because of a sudden onset of severe right-sided flank pain. The pain is colicky and radiates from the flank to the scrotum. He also has nausea, vomiting and dark-colored urine. He has never had these symptoms before. His temperature is 37° C (98.6° F), blood pressure is 126/70 mm Hg, pulse is 90/min, and respirations are 18/min. Examination shows no abnormalities. He is given adequate analgesia. Non-contrast helical CT shows a 4 mm radiopaque stone in the right upper ureter. Laboratory studies show serum calcium of 9.8 mg/dL, serum creatinine of 0.9 mg/dL, and BUN of 15 mg/dL. Urinalysis shows hematuria but no casts. Which of the following is the most appropriate next step in management?

- A. 24 hr urine collection for metabolic evaluation
- B. Reassurance
- C. Fluid intake greater than 2 L/day
- D. Intake of potassium citrate
- E. Restriction of dietary oxalate

Submit

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- A. 24 hr urine collection for metabolic evaluation [3%]
- B. Reassurance [7%]
- ✔ C. Fluid intake greater than 2 L/day [81%]
- D. Intake of potassium citrate [4%]
- E. Restriction of dietary oxalate [3%]

Omitted
Correct answer
C

81%
Answered correctly

2 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

This patient has the classic clinical presentation of nephrolithiasis. The following are important concepts in the management of such patients.

Explanation

This patient has the classic clinical presentation of nephrolithiasis. The following are important concepts in the management of such patients.

1. Imaging study - CT scan of the abdomen without contrast is the investigation of choice because of its high sensitivity and specificity. It has the advantage over the plain abdominal x-ray (KUB) in detecting the radiolucent stones.

2. Narcotics and NSAIDs - These are equally effective in relieving the pain of acute renal colic; however, in patients with normal renal function, NSAIDs are preferred over narcotics because the latter can exacerbate nausea and vomiting.

3. Size of the stone - Stones measuring less than 5mm in diameter typically pass spontaneously with conservative management. This includes a fluid intake of greater than 2L daily. Increased hydration increases the urinary flow rate and lowers the urinary solute concentration, thus preventing stone formation.

4. Urology referral - Urgent urologic evaluation is warranted in patients with anuria, urosepsis, or acute renal failure.

(Choice A) A detailed metabolic evaluation is not needed when a patient presents with his first renal stone. In patients with recurrent renal stones, 24-hr urine is collected to identify any underlying metabolic disorder. A complete urinary evaluation includes measurement of calcium, citrate, creatinine, uric acid, oxalate, pH and sodium levels.

(Choice B) Reassurance alone is not appropriate. Although his renal stone is relatively small, he still requires conservative management (i.e., adequate hydration).

(Choice D) Potassium citrate is the appropriate treatment when a patient presents with a history of recurrent stone formation due to citrate deficiency.

(Choice E) Restriction of dietary oxalate is helpful when a patient presents with a history of recurrent calcium stone formation due to hyperoxaluria.

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Educational Objective:
Hydration is the cornerstone of therapy for renal stone disease. A detailed metabolic evaluation is not needed when a patient presents with his first renal stone.

A 57-year-old woman is admitted to the ICU after being involved in a highway motor vehicle accident. She was hypotensive at the scene and received 7 liters of fluids, which included crystalloids, blood, and fresh frozen plasma. She apparently had significant external blood loss from multiple fractures and skin loss. She undergoes surgery, after which she is transferred to the ICU and receives continuous IV fluids and vasopressors. Her laboratory studies 24 hours after the accident show the following:

Hb	9.5 g/dL
WBC	15,000/cmm
Platelets	130,000/cmm
BUN	34 mg/dL
Serum Creatinine	2.2 mg/dL

Which of the following is the most likely microscopic finding on urinalysis?

A. Broad cast
B. Muddy brown cast
C. RBC casts
D. WBC casts
E. Fatty casts
F. Eosinophils

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Platelets	130,000/cmm
BUN	34 mg/dL
Serum Creatinine	2.2 mg/dL

Which of the following is the most likely microscopic finding on urinalysis?

- A. Broad cast [4%]
- ✔ B. Muddy brown cast [75%]
- C. RBC casts [9%]
- D. WBC casts [2%]
- E. Fatty casts [5%]
- F. Eosinophils [2%]

Omitted
Correct answer
B

75%
Answered correctly

26 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The clinical picture of this patient is highly suggestive of acute tubular necrosis (ATN) following hypovolemic shock. Her serum BUN and Cr ratio is less than 20:1. Other findings that support this diagnosis are:

1. Urine osmolality of 300-350 mOsm/L (but never <300)
2. Urine Na of >20 mEq/L
3. FENa >2%

Prolonged hypotension from any cause can lead to ATN. The hallmark findings on urinalysis are muddy brown granular casts consisting of renal tubular epithelial cells; this is a nonspecific, but very sensitive finding for ATN.

(Choice A) Broad casts are seen in patients with chronic renal failure (CRF). These arise in the dilated tubules of enlarged nephrons that have undergone compensatory hypertrophy in response to the reduced renal mass. Waxy casts, which are shiny and translucent, are also generally seen in chronic renal disease.

(Choice C) RBC casts are indicative of glomerular disease or vasculitis.

(Choice D) WBC casts are definitive evidence that urinary WBCs originate in the kidney. These are seen in cases of interstitial nephritis, pyelonephritis, etc.

(Choice E) Fatty casts are seen in conditions causing nephrotic syndrome. Hyaline casts are composed almost entirely of protein and pass unchanged along the urinary tract; these may be seen in asymptomatic individuals and in patients with pre-renal azotemia.

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Educational Objective:
Muddy brown granular cast - Acute tubular necrosis
RBC casts - Glomerulonephritis
WBC casts - Interstitial nephritis and pyelonephritis
Fatty casts - Nephrotic syndrome
Broad and waxy casts - Chronic renal failure

*Extremely high yield question for the USMLE!!!

A 24-year-old woman is brought to the emergency department due to feeling lightheaded every time she stands up. The patient has no significant medical problems. She left her boyfriend 4 months ago as he was "eating too much" and has since had decreased appetite and lost 5 kg (11 lb). The patient has also missed her last 2 menstrual periods. She does not use tobacco, alcohol, or illicit drugs. Family history is significant for coronary artery disease and congestive heart failure. Her temperature is 36.7 C (98 F), blood pressure is 100/70 mm Hg, and pulse is 88/min while supine. On standing, blood pressure is 80/55 mm Hg and pulse is 120/min. Other than dry skin and mucous membranes, the patient's physical examination is unremarkable. Initial laboratory results are as follows:

Serum chemistry

Sodium	132 mEq/dL
Potassium	2.8 mEq/dL
Chloride	88 mEq/dL

Urine electrolytes

Sodium	73 mEq/L (~20 mEq/L)
Potassium	68 mEq/L (5-15 mEq/L)

Which of the following is the most likely underlying etiology for this patient's symptoms?

A. Cerebral salt wasting
B. Diuretic abuse
C. Laxative abuse

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- D. Low caloric intake
- E. Mineralocorticoid deficiency
- F. Self-induced vomiting

Submit

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Which of the following is the most likely underlying etiology for this patient's symptoms?

A. Cerebral salt wasting [1%]
✔ B. Diuretic abuse [71%]
C. Laxative abuse [6%]

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- A. Cerebral salt wasting [1%]
- ✔ B. Diuretic abuse [71%]
- C. Laxative abuse [6%]
- D. Low caloric intake [4%]
- E. Mineralocorticoid deficiency [2%]
- F. Self-induced vomiting [13%]

Omitted
Correct answer
B

71%
Answered correctly

5 Seconds
Time Spent

10/26/2018
Last Updated

Explanation

This young woman has dizziness due to **orthostatic hypotension**. In addition, she has abnormal serum and urine electrolytes, including hyponatremia, hypokalemia, hypochloremia, and increased urinary sodium and potassium. **Diuretic abuse**, which leads to hypovolemia, best explains the laboratory findings in this patient as follows:

- Normally, dehydrated patients with hyponatremia are expected to have low urine sodium (<20 mEq/L); however, this patient has **elevated urine sodium**, which suggests **salt wasting** (eg, diuretic use, cerebral salt wasting [CSW], adrenal insufficiency).
- Normally, patients with hypokalemia respond by reducing urine potassium excretion, except in cases of renal potassium wasting (eg, diuretic use, hyperaldosteronism, renal tubular acidosis).
- Hypochloremia is likely a result of diuresis and contraction alkalosis; urine chloride levels can vary.

Surreptitious diuretic use is sometimes employed as a means to lose weight. Patients may gain access to these medications from family members who have been appropriately prescribed these medications.

(Choice A) CSW presents with hypovolemia and hyponatremia with high urine sodium (>20 mEq/L). However, CSW always occurs due to a neurologic insult (injury or surgery).

(Choice C) Laxative abuse will also lead to hypotension and hypovolemia. However, elevated urine sodium indicates that sodium loss is through the urinary tract (eg, diuretic use) rather than the gastrointestinal tract (eg, laxative use).

(Choice D) Low caloric intake causes weight loss secondary to wasting of muscle mass and fat and could also lead to electrolyte abnormalities. However, elevated urine sodium and potassium are not expected.

(Choice E) Mineralocorticoid deficiency can lead to wasting of sodium and water, but serum potassium levels are increased in such patients.

(Choice F) Self-induced vomiting may lead to hypovolemia, hypokalemia, and hyponatremia. However, urinary sodium will be low as the kidneys try to conserve water by maximally resorbing sodium and water.

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Educational objective:
Diuretic abuse leads to increased excretion of water and electrolytes by the kidneys, resulting in dehydration, weight loss, orthostatic hypotension, hyponatremia, and hypokalemia. Urinary sodium and potassium will be elevated. Patients sometimes abuse diuretics to induce weight loss.

References

- Diuretic abuse and central pontine myelinolysis.

E. Mineralocorticoid deficiency [2%]

F. Se

Omitted

Correct answe

B

Explanatio

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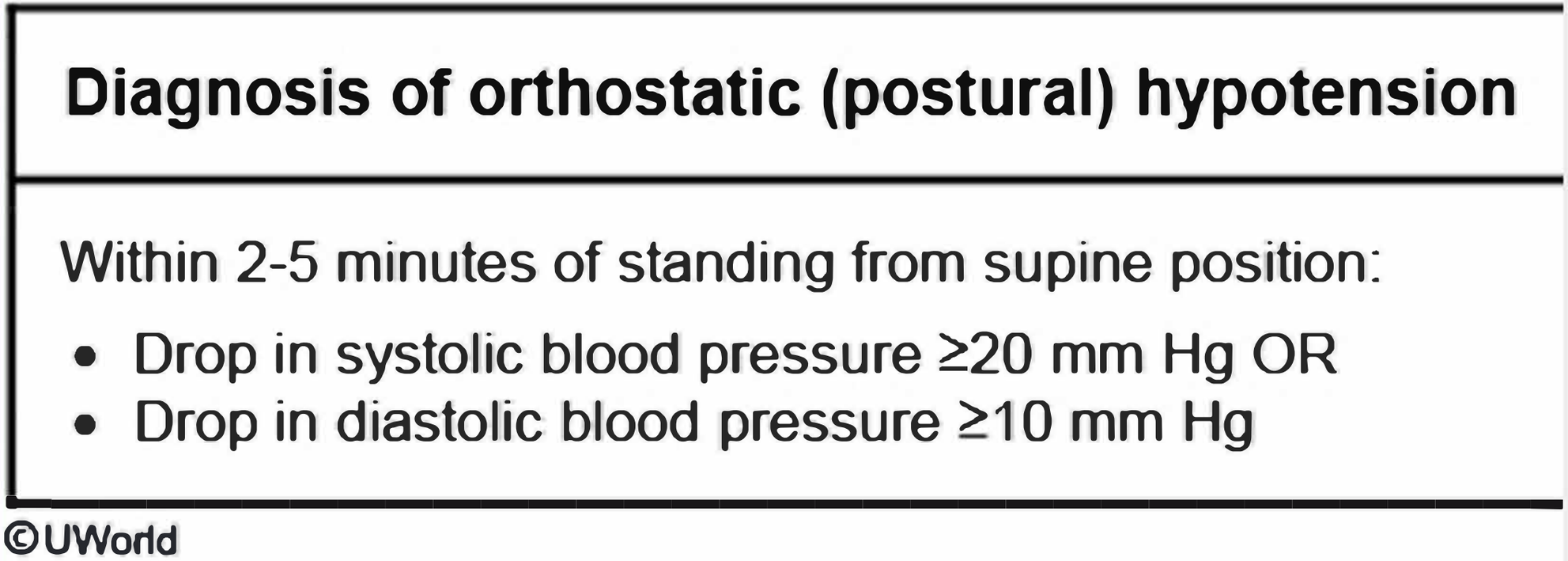

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A 24-year-old man is brought to the emergency department with severe upper back pain and some abdominal discomfort after involvement in a motor vehicle accident. Blood pressure is 115/78 mm Hg, pulse is 55/min, and respirations are 16/min. Pulse oximetry is 96% on room air. The patient's neck and body have been immobilized, and he has 2 large-bore intravenous lines in place. He is alert, fully oriented, and conversant. There are several lacerations on the face and anterior and posterior chest, but the abdomen and lower body have no superficial signs of injury. Air entry is bilaterally symmetric. There is weakness and decreased pain sensation in both legs. Proprioceptive sensation is preserved. Laboratory workup is sent. Chest x-ray and CT scans of the abdomen and spine are scheduled urgently. Which of the following is the most appropriate next step in management of this patient?

- A. Bladder catheterization
- B. Femoral line placement
- C. Intravenous atropine
- D. Intravenous cefazolin
- E. Nasogastric tube placement

Submit

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- ✔ A. Bladder catheterization [82%]
- B. Femoral line placement [4%]
- C. Intravenous atropine [5%]
- D. Intravenous cefazolin [2%]
- E. Nasogastric tube placement [5%]

Omitted
Correct answer
A

82%
Answered correctly

2 Seconds
Time Spent

07/11/2018
Last Updated

Explanation

All trauma patients should be evaluated for cardiorespiratory stability and have the spine immobilized until spinal injury has been ruled

Explanation

All trauma patients should be evaluated for cardiorespiratory stability and have the spine immobilized until spinal injury has been ruled out. Airway protection and mechanical ventilation are not needed if the patient is awake and has a normal respiratory examination. Additional intravenous (IV) access, such as a femoral line, is not required in hemodynamically stable patients if adequate peripheral IV access is established **(Choice B)**.

After cardiorespiratory and spine stability have been established, a focused neurologic examination is performed as part of the primary survey. This patient has weakness and decreased pain sensation in both legs, suggesting spinal cord injury. In the absence of obvious pelvic injury and blood at the urethral meatus, such patients should have a urinary catheter placed to assess for urinary retention and prevent possible bladder injury from acute distension (which is likely causing the abdominal discomfort in this patient). Imaging is then performed to diagnose and evaluate spinal cord damage. Surgical intervention is indicated in patients with acute cord compression with neurologic defects or unstable vertebral fracture/dislocation.

(Choice C) IV atropine or external pacing is indicated only for symptomatic bradycardia. Symptoms include lightheadedness, presyncope, or syncope.

(Choice D) IV cefazolin is commonly used for antimicrobial prophylaxis before surgery to prevent wound infections. It is usually given within 60 minutes of the procedure.

(Choice E) Nasogastric tube placement is indicated for bowel obstruction, enteral nutrition, and gastric lavage but is not otherwise recommended for routine use. This patient is alert, can swallow, and does not have an immediate need for nasogastric tube placement.

Educational objective:
In patients with traumatic spinal cord injuries, urinary catheter placement can assess for urinary retention and prevent acute bladder distension and damage.

A 58-year-old man with a history of extensive alcohol use is admitted with the diagnosis of decompensated liver cirrhosis and ascites.
The laboratory panel is shown below on the day of admission and 3 days later.

	Admission	3 days later
pH	7.34	7.47
Bicarbonate (mEq/L)	19	31
Serum sodium (mEq/L)	133	135
Serum potassium (mEq/L)	4.6	3.1
Serum chloride (mEq/L)	101	92
Blood urea nitrogen (mg/dl)	34	45
Serum creatinine (mg/dl)	1.7	2.1

Which of the following best explains the acid-base status change in this patient?

A. Acute kidney injury
B. Bowel ischemia
C. Loop diuretic therapy

Bicarbonate (mEq/L)	19	31
Serum sodium (mEq/L)	133	135
Serum potassium (mEq/L)	4.6	3.1
Serum chloride (mEq/L)	101	92
Blood urea nitrogen (mg/dl)	34	45
Serum creatinine (mg/dl)	1.7	2.1

Which of the following best explains the acid-base status change in this patient?

- A. Acute kidney injury
- B. Bowel ischemia
- C. Loop diuretic therapy
- D. Opioid medication use
- E. Right lower-lobe atelectasis

Submit

A 58-year-old man with a history of extensive alcohol use is admitted with the diagnosis of decompensated liver cirrhosis and ascites. The laboratory panel is shown below on the day of admission and 3 days later.

	Admission	3 days later
pH	7.34	7.47
Bicarbonate (mEq/L)	19	31
Serum sodium (mEq/L)	133	135
Serum potassium (mEq/L)	4.6	3.1
Serum chloride (mEq/L)	101	92
Blood urea nitrogen (mg/dl)	34	45
Serum creatinine (mg/dl)	1.7	2.1

Which of the following best explains the acid-base status change in this patient?

A. Acute kidney injury [31%]
B. Bowel ischemia [1%]
✔ C. Loop diuretic therapy [64%]

Bicarbonate (mEq/L)	19	31
Serum sodium (mEq/L)	133	135
Serum potassium (mEq/L)	4.6	3.1
Serum chloride (mEq/L)	101	92
Blood urea nitrogen (mg/dl)	34	45
Serum creatinine (mg/dl)	1.7	2.1

Which of the following best explains the acid-base status change in this patient?

- A. Acute kidney injury [31%]
- B. Bowel ischemia [1%]
- ✔ C. Loop diuretic therapy [64%]
- D. Opioid medication use [1%]
- E. Right lower-lobe atelectasis [1%]

Omitted
Correct answer
C

64%
Answered correctly

6 Seconds
Time Spent

10/09/2018
Last Updated

Explanation

On admission, this patient's acid-base status was borderline acidemic; now he is mildly alkalemic. The most likely intervention to have resulted in this change is administration of a loop diuretic. Volume overload and ascites are common complications in patients with decompensated liver cirrhosis, and loop diuretics such as furosemide are a common treatment, often in combination with spironolactone.

Loop diuretics function by inhibiting the Na-K-2Cl carrier in the loop of Henle, which results in increased loss of sodium in the urine. The increased sodium delivery to the distal tubule subsequently results in elevated hydrogen and potassium ion secretion in the urine. Loop diuretics also result in volume contraction and increased aldosterone levels, further promoting the secretion of hydrogen ions in the urine. This patient's elevation in blood urea nitrogen and creatinine is likely secondary to reduction in circulating blood volume and subsequent mild acute kidney injury. This patient's mild hyponatremia on admission is due to volume overload (dilutional), which has improved with diuresis (excess water diuresis compared to sodium loss).

(Choice A) This patient has likely developed mild acute kidney injury as evidenced by his mild increase in blood urea nitrogen and creatinine. However, acute kidney injury tends to cause an anion gap metabolic acidosis and hyperkalemia.

(Choice B) Bowel ischemia results in an anion gap acidosis from increased circulating lactate; this patient has a metabolic alkalosis.

(Choice D) Opioid medication may result in hypoventilation and subsequent respiratory acidosis. Although this can result in a compensatory increase in bicarbonate level, this patient's pH of 7.47 is more consistent with a primary metabolic alkalosis.

(Choice E) Atelectasis is not a common cause of acid-base disturbances.

Educational objective:
Loop diuretics are frequently administered to cirrhotic patients with volume overload and ascites. Potential side effects include hypokalemia, metabolic alkalosis, and prerenal kidney injury.

A 38-year-old woman comes to the emergency department for right flank pain radiating to the groin. She also has hematuria but no dysuria or urinary frequency. She has no other medical problems and takes no medications. Her temperature is 36.7 C (98 F), blood pressure is 110/80 mm Hg, and pulse is 68/min. The patient has no costovertebral angle tenderness. Abdominal imaging shows a 6-mm calculus in the distal right ureter with no hydronephrosis. The patient's symptoms improve with intravenous hydration and analgesics. Which of the following medications will be most helpful in facilitating stone passage?

- A. Bethanechol
- B. Finasteride
- C. Furosemide
- D. Imipramine
- E. Oxybutynin
- F. Phenazopyridine
- G. Tamsulosin

Submit

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- A. Bethanechol [16%]
- B. Finasteride [2%]
- C. Furosemide [20%]
- D. Imipramine [1%]
- E. Oxybutynin [8%]
- F. Phenazopyridine [5%]
- ✔ G. Tamsulosin [44%]

Omitted
Correct answer
G

44%
Answered correctly

4 Seconds
Time Spent

10/30/2018
Last Updated

Explanation

This patient has ureteric colic secondary to a distal ureteral stone. Uncomplicated stones <1 cm can be managed conservatively

Explanation

This patient has ureteric colic secondary to a distal **ureteral stone**. Uncomplicated stones <1 cm can be managed conservatively with hydration, analgesics, and alpha blockers. Alpha receptors are found on the distal ureter, base of the detrusor, bladder neck, and urethra. Sympathetic activation stimulates alpha receptors to maintain high muscular tone for normal urinary continence. Reflex ureteral spasm secondary to stone impaction causes the typical waxing and waning pain seen in ureteral colic. Tamsulosin is an alpha 1 antagonist that relaxes ureteral muscle and decreases intraureteral pressure. This facilitates stone passage and reduces the need for analgesics.

(Choices A and E) Cholinergic receptors are found on the dome of the bladder, contraction of which facilitates voiding. These receptors are not known to be present on the ureter. Bethanechol is a cholinergic agent used to treat urinary retention or atonic bladder. Oxybutynin is an anticholinergic agent that inhibits cholinergic input during bladder filling. It helps improve bladder capacity and is used to treat overactive bladder (detrusor instability).

(Choice B) Finasteride is a 5-alpha-reductase inhibitor that blocks the conversion of testosterone to dihydrotestosterone. It is used as a second-line agent in the management of benign prostatic hyperplasia.

(Choice C) Furosemide is a loop diuretic that promotes calciuria, which can predispose to renal calculi.

(Choice D) Imipramine is an antidepressant used to treat childhood enuresis. It has anticholinergic effects and promotes urinary retention.

(Choice F) Phenazopyridine is an analgesic for urinary tract mucosa frequently used to treat the dysuria of cystitis (eg, after infection or instrumentation). It may worsen stone formation (mechanism unknown).

Educational objective:
Alpha-1 receptor blockers such as tamsulosin act on the distal ureter, lowering muscle tone and reducing reflex ureteral spasm secondary to stone impaction. These agents facilitate stone passage and reduce the need for analgesics.

A 9-month-old infant is brought to the emergency department with lethargy and tachypnea. He was healthy before developing fever and diarrhea four days ago. He has been taking some formula, but has had two to three episodes of diarrhea with each bottle. He has lost three pounds (1.4 kg) since his routine check-up two weeks ago. He has had one wet diaper in the past twenty four hours. On examination, his temperature is 102.5° F (39.1° C), pulse is 200/min, respiratory rate is 42/min, and blood pressure is 70/45 mm Hg. He is lethargic with decreased tone and decreased deep tendon reflexes. His mucous membranes are dry. Cardiopulmonary exam reveals tachycardia and tachypnea. His abdominal exam is unremarkable. Capillary refill is four seconds. Laboratory results are shown below.

Chemistry panel

Serum sodium	165 mEq/L
Serum potassium	4.5 mEq/L
Chloride	108 mEq/L
Bicarbonate	14 mEq/L
Blood urea nitrogen (BUN)	20 mg/dL
Serum creatinine	0.8 mg/dL
Calcium	10.0 mg/dL
Blood glucose	98 mg/dL

Which of the following fluids should be used as a bolus in the resuscitation of this infant?

A. 0.9% saline

Serum sodium	165 mEq/L
Serum potassium	4.5 mEq/L
Chloride	108 mEq/L
Bicarbonate	14 mEq/L
Blood urea nitrogen (BUN)	20 mg/dL
Serum creatinine	0.8 mg/dL
Calcium	10.0 mg/dL
Blood glucose	98 mg/dL

Which of the following fluids should be used as a bolus in the resuscitation of this infant?

- A. 0.9% saline
- B. 0.45% saline
- C. 5% dextrose
- D. 5% albumin
- E. Packed red blood cells

Submit

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Serum creatinine	0.8 mg/dL
Calcium	10.0 mg/dL
Blood glucose	98 mg/dL

Which of the following fluids should be used as a bolus in the resuscitation of this infant?

A. 0.9% saline [67%]

Serum potassium	4.5 mEq/L
Chloride	108 mEq/L
Bicarbonate	14 mEq/L
Blood urea nitrogen (BUN)	20 mg/dL
Serum creatinine	0.8 mg/dL
Calcium	10.0 mg/dL
Blood glucose	98 mg/dL

Which of the following fluids should be used as a bolus in the resuscitation of this infant?

- ✔ A. 0.9% saline [67%]
- B. 0.45% saline [24%]
- C. 5% dextrose [6%]
- D. 5% albumin [1%]
- E. Packed red blood cells [0%]

Omitted
Correct answer
A

67%
Answered correctly

5 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

The signs and symptoms of hypernatremia are mainly neurologic and include lethargy, altered mental status, irritability, and seizures. Hypernatremia can also cause muscle cramps, muscle weakness, and decreased deep tendon reflexes.

Hypovolemic hypernatremia develops secondary to renal losses (eg, diuretic use, glycosuria) or extrarenal losses (eg, gastrointestinal upset, excessive sweating). Hypervolemic hypernatremia occurs due to exogenous sodium intake or mineralocorticoid excess (eg, hyperaldosteronism).

When treating a patient with hypernatremia, the sodium must be slowly returned to normal. In this infant with hypernatremia and dehydration, the initial goal is to stabilize him with fluid resuscitation as needed. When giving intravenous fluid boluses, only isotonic solutions such as normal saline or lactated Ringer's should be used.

(Choices B and C) Half normal saline and 5% dextrose are hypotonic solutions. As such, they should never be used for initial resuscitation because they quickly exit the intravascular system and lower the sodium too rapidly. Precipitous drops in sodium levels can cause cerebral edema.

(Choice D) Multiple studies have demonstrated that the expensive colloid solutions are no better than crystalloids at fluid resuscitation.

(Choice E) Packed red blood cells may be appropriate in initial fluid resuscitation when bleeding is a major issue. Because of the limited availability of packed red blood cells on short notice, however, isotonic solutions remain the mainstay of initial fluid resuscitation (even in individuals with massive bleeding).

Educational objective:
Isotonic solutions such as normal saline are the fluid of choice for initial resuscitation in severe hypovolemic hypernatremia.

A 3-year-old girl is brought to the physician with dysuria. Her parents say that she has been crying with urination, even though "a small amount comes out at a time." She has no vomiting, nausea, or fever. In the past year, the girl had 3 bladder infections that were treated with antibiotics; the last infection was 2 months ago. She also has a history of constipation since starting cow's milk at age 1 and takes a laxative as needed to help with bowel movements. Her temperature is 37.2 C (99 F), blood pressure is 80/50 mm Hg, and pulse is 110/min. Examination shows suprapubic tenderness and small anal fissures at 12 o'clock and 2 o'clock. Rectal examination shows normal anal wink and tone; hard stool is palpated in the rectal vault. Urinalysis shows positive leukocyte esterase, positive nitrites, and white blood cells 60/hpf. Urine culture shows 100,000 colonies of *Escherichia coli*. Renal ultrasound and voiding cystoureterogram are normal. Which of the following is the most likely mechanism for this patient's infection?

- A. Inadequate treatment of prior infection
- B. Neurologic impairment
- C. Renal insufficiency
- D. Sexual abuse
- E. Urinary reflux
- F. Urinary stasis

Submit

A 3-year-old girl is brought to the physician with dysuria. Her parents say that she has been crying with urination, even though "a small amount comes out at a time." She has no vomiting, nausea, or fever. In the past year, the girl had 3 bladder infections that were treated with antibiotics; the last infection was 2 months ago. She also has a history of constipation since starting cow's milk at age 1 and takes a laxative as needed to help with bowel movements. Her temperature is 37.2 C (99 F), blood pressure is 80/50 mm Hg, and pulse is 110/min. Examination shows suprapubic tenderness and small anal fissures at 12 o'clock and 2 o'clock. Rectal examination shows normal anal wink and tone; hard stool is palpated in the rectal vault. Urinalysis shows positive leukocyte esterase, positive nitrites, and white blood cells 60/hpf. Urine culture shows 100,000 colonies of *Escherichia coli*. Renal ultrasound and voiding cystoureterogram are normal. Which of the following is the most likely mechanism for this patient's infection?

- A. Inadequate treatment of prior infection [5%]
- B. Neurologic impairment [7%]
- C. Renal insufficiency [0%]
- D. Sexual abuse [14%]
- E. Urinary reflux [15%]
- ✔ F. Urinary stasis [57%]

Omitted
Correct answer
F

57%
Answered correctly

3 Seconds
Time Spent

10/11/2018
Last Updated

Explanation

Explanation

Pediatric constipation	
Risk factors	• Initiation of solid food & cow's milk • Toilet training • School entry
Clinical features	• Painful/hard bowel movements • Stool withholding • Encopresis
Complications	• Anal fissures • Hemorrhoids • Enuresis/urinary tract infections
Treatment	• ↑ Dietary fiber & water intake • Limit cow's milk intake to <24 oz • Laxatives • ± Suppositories, enema

This child has recurrent cystitis, which is characterized by **suprapubic pain**, **dysuria**, **pyuria**, and **bacteriuria**. Recurrent cystitis in toddlers is often caused by constipation as **fecal retention** can cause rectal distension, which in turn **compresses the bladder** and prevents complete voiding. The residual urine is a potential breeding ground for bacteria that ascend to the urethra from the perineum.

Risk factors of constipation include dietary changes, such as transition from breast milk to cow's milk and solid foods. Signs of

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Risk factors of constipation include dietary changes, such as transition from breast milk to cow's milk and solid foods. Signs of constipation include straining or pain with defecation, passage of firm **pellet-like stools**, and anal fissures and hemorrhoids. Prevention and treatment of recurrent cystitis requires adequate treatment of constipation.

(Choice A) Inadequate treatment of a prior infection is unlikely due to the 2-month gap between the current and last illness.

(Choice B) Spina bifida and Hirschsprung disease (congenital aganglionic megacolon) are associated with neurologic impairment. These are unlikely in this patient with an otherwise normal examination and constipation onset correlating with dietary changes.

(Choice C) Renal insufficiency is a potential complication of recurrent pyelonephritis (urinary tract infection involving the kidney) and can manifest as poor urine output, malnutrition, poor growth, hypertension, and anemia. It is not a direct cause of recurrent cystitis.

(Choice D) Sexual activity is associated with recurrent cystitis in women, but it is rarely the cause in children. The absence of vaginal pain, behavioral changes, and genital, perineal, or anal injury makes this diagnosis unlikely. Anal fissures are a common manifestation of chronic constipation.

(Choice E) Renal ultrasound is performed to rule out hydronephrosis. **Voiding cystoureterogram** is the imaging study of choice to detect structural abnormalities (eg, **vesicoureteral reflux**). An underlying anatomical problem (eg, vesicoureteral reflux) is a common cause of recurrent urinary tract infections in infants. However, this child had normal imaging studies.

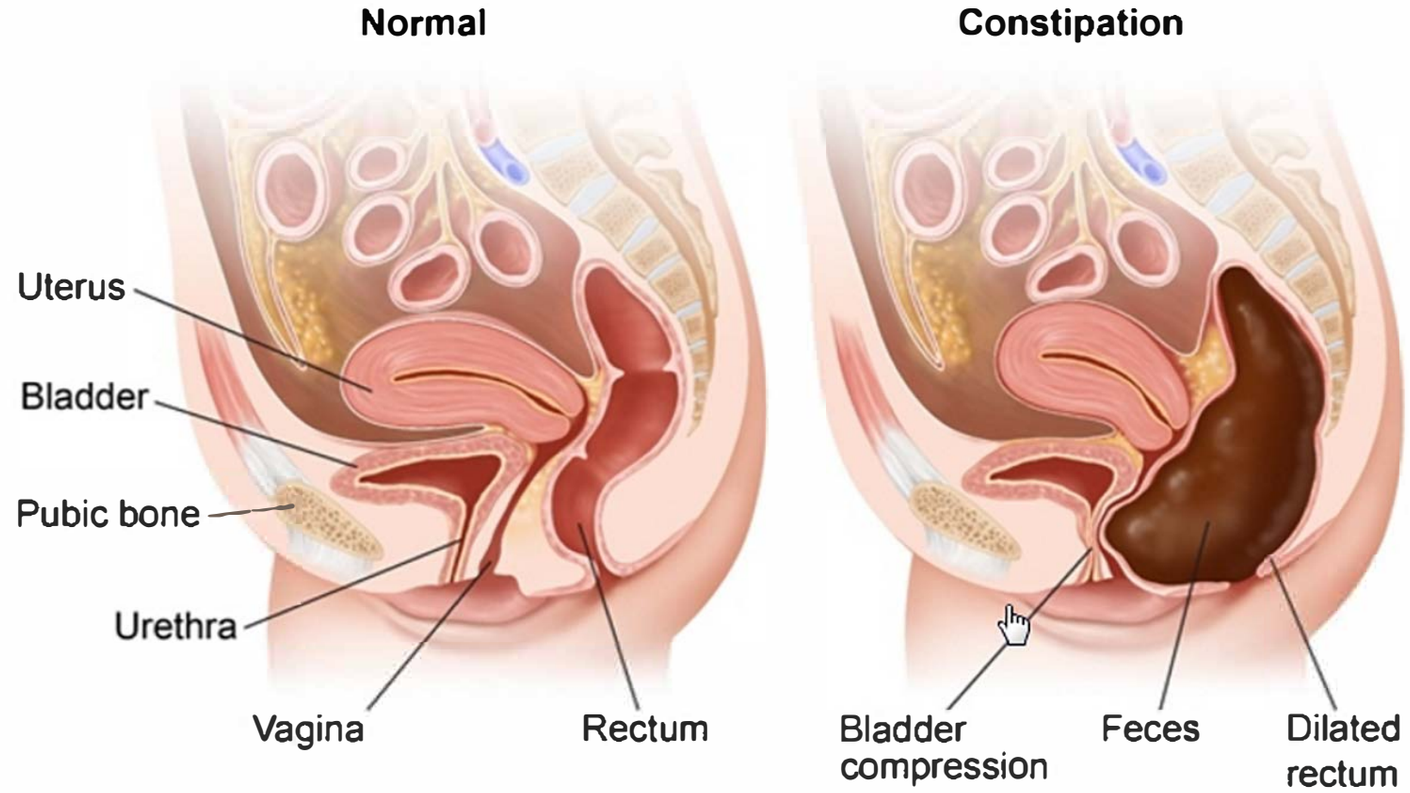
Educational objective:
Chronic constipation is a risk factor for recurrent cystitis in toddlers. Impacted stool can cause rectal distension, which in turn compresses the bladder, prevents complete voiding, and leads to urinary stasis.

References

Risk factors	• Toilet training

Exhibit Display

Chronic constipation



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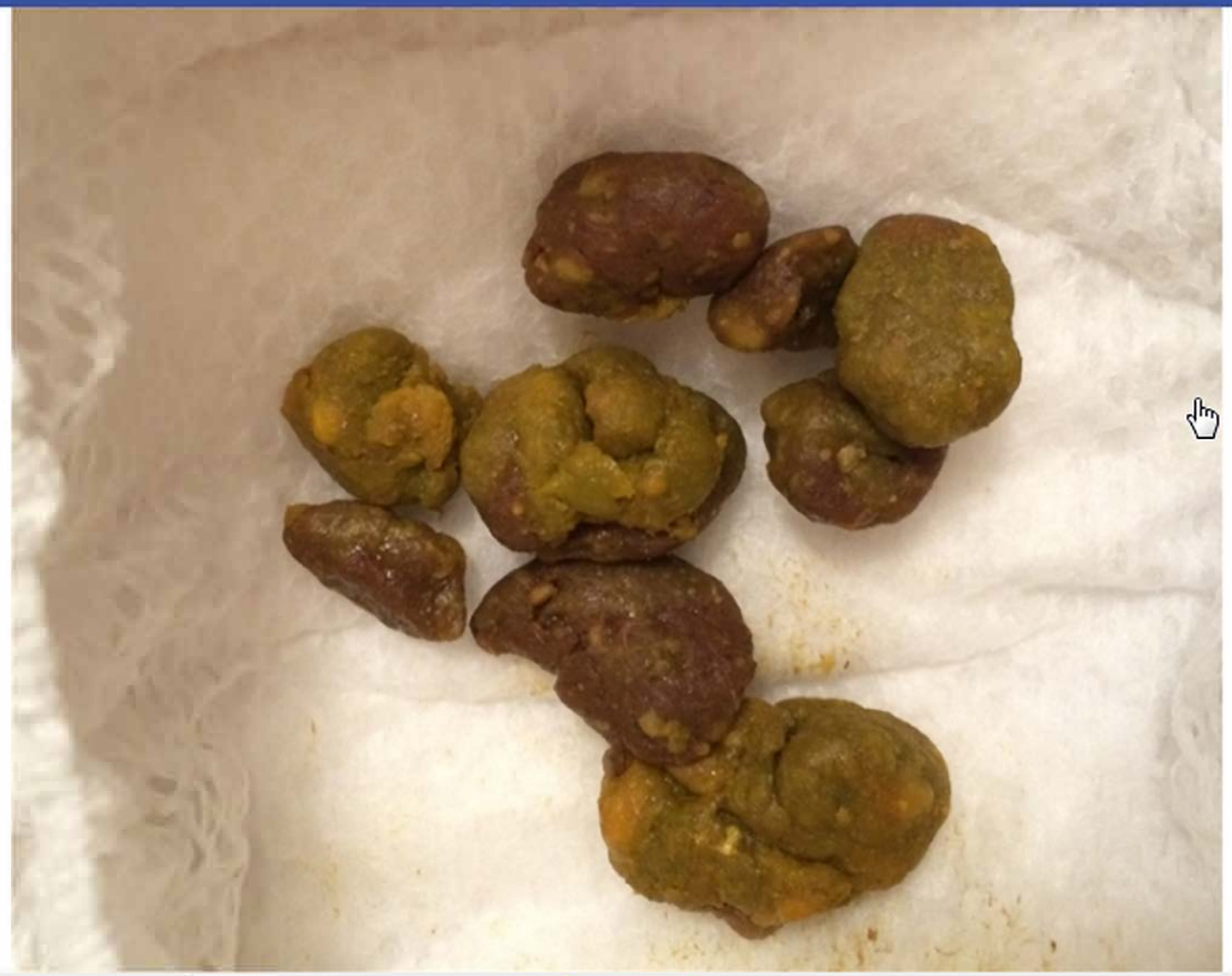

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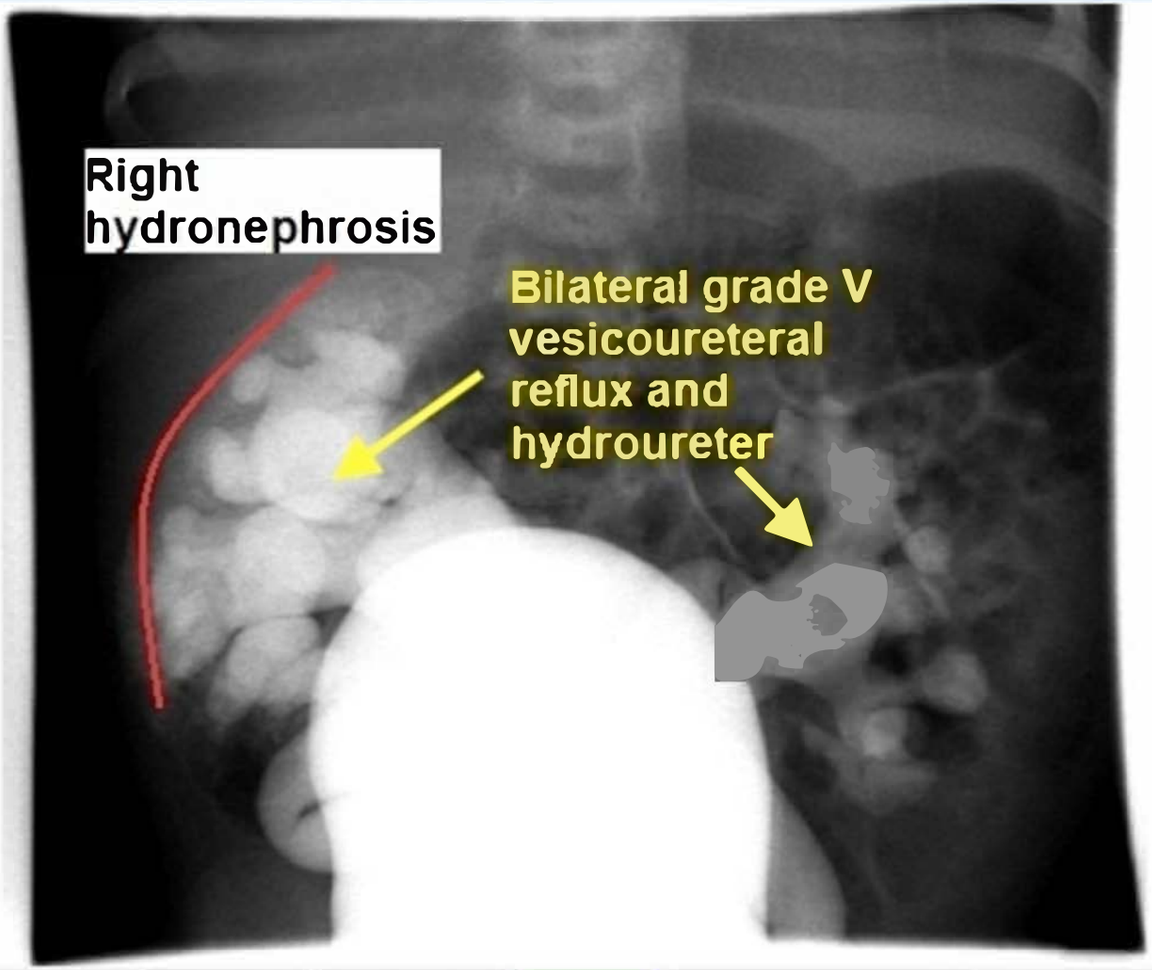

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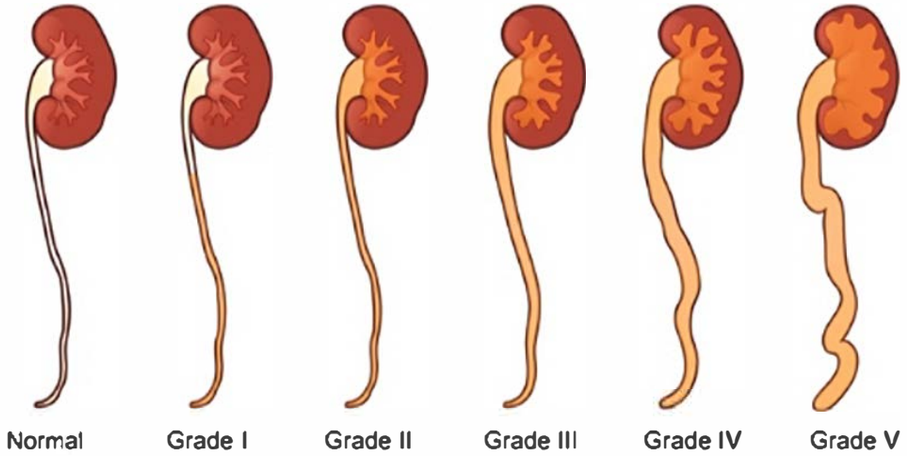
Vesicoureteral reflux



Grade	Description
I	Into a nondilated ureter
II	Into the pelvis & calyces without dilation
III	Mild to moderate dilation of the ureter, renal pelvis & calyces, with minimal blunting of the fornices
IV	Moderate ureteral tortuosity & dilation of the pelvis & calyces
V	Gross dilation of the ureter, pelvis & calyces; loss of papillary impressions; ureteral tortuosity

Zoom In | Zoom Out | Reset | Add To Flash Card

This child has recurrent cystitis, which is characterized by **suprapubic pain**, **dysuria**, **pyuria**, and **bacteriuria**. Recurrent cystitis in toddlers is often [illegible] prevents complete emptying of the bladder and perineum.

Risk factors of [illegible] constipation in[illegible] Prevention and[illegible]

(Choice A) In[illegible]

(Choice B) Sp[illegible] These are unli[illegible]

(Choice C) Re[illegible] can manifest a[illegible]

(Choice D) Se[illegible] pain, behaviora[illegible] manifestation o[illegible]

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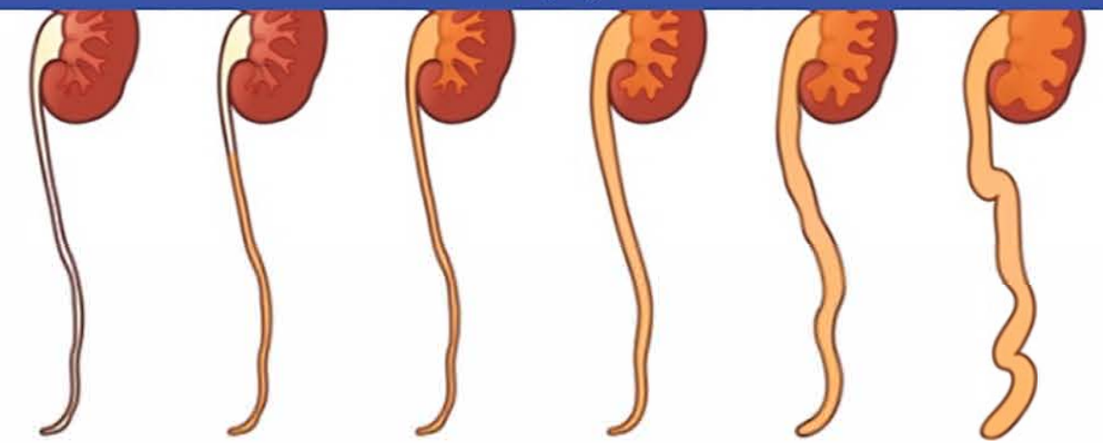

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Zoom In | Zoom Out | Reset | Add To Flash Card

A 7-month-old boy is brought to the physician due to fever, fussiness, and decreased wet diapers for the past week. He has no medical problems and takes no medications. His immunizations are up-to-date. His temperature is 39.4 C (103 F). Examination shows a tired-appearing boy with an uncircumcised penis. Laboratory results are as follows:

Complete blood count	
Hemoglobin	13 g/dL
Hematocrit	40%
Platelets	205,000/μL
Leukocytes	15,800/μL
Neutrophils	80%
Serum chemistry	
Sodium	135 mEq/L
Potassium	4.5 mEq/L
Chloride	100 mEq/L
Bicarbonate	26 mEq/L
Blood urea nitrogen	20 mg/dL
Creatinine	1.4 mg/dL

Which of the following is the best next step in management of this patient?

Neutrophils	80%

Serum chemistry

Sodium	135 mEq/L
Potassium	4.5 mEq/L
Chloride	100 mEq/L
Bicarbonate	26 mEq/L
Blood urea nitrogen	20 mg/dL
Creatinine	1.4 mg/dL

Which of the following is the best next step in management of this patient?

A. Abdominal ultrasound
B. Clean-catch urinalysis and urine culture
C. CT scan of the abdomen
D. Intravenous pyelogram
E. Urethral catheterization, urinalysis, and urine culture

Submit

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Which of the following is the best next step in management of this patient?

- A. Abdominal ultrasound [6%]
- B. Clean-catch urinalysis and urine culture [22%]
- C. CT scan of the abdomen [0%]
- D. Intravenous pyelogram [1%]
- ✔ E. Urethral catheterization, urinalysis, and urine culture [69%]

Omitted
Correct answer
E

69%
Answered correctly

4 Seconds
Time Spent

07/11/2018
Last Updated

Explanation

Diagnostic tests in urinary tract infections	
Serum BUN & creatinine	Estimate renal function
Urine dipstick	Qualitative measurement of urine properties
Urinalysis	Quantitative measurement of urine properties
Urine culture	Identification, quantification & susceptibility testing of bacterial colonies

Urinary tract infections (UTIs) in infants and toddlers must be diagnosed and treated promptly as they usually involve the kidneys (pyelonephritis). Risk factors include girls at any age (short urethra), uncircumcised boys age ≤1, and underlying renal anomaly (eg, vesicoureteral reflux, posterior urethral valves). During infancy, symptoms are nonspecific and vague (eg, fever, fussiness, decreased urine output); abdomen/flank pain and dysuria can be difficult to recognize as infants are nonverbal. The presence of **fever >39 C (102.2 F)** in any child age <3 should prompt evaluation for occult UTI.

Serum blood urea nitrogen (BUN) and creatinine and urinalysis are quick, noninvasive, preliminary tests that should be done in all infants with illnesses involving the urinary tract. The BUN and creatinine provide a general sense of the patient's hydration status and degree of renal impairment. Urine dipsticks are also commonly performed, but they have a high rate of false-positive and negative results. Microscopic **urinalysis** is more accurate as it provides quantitative data on the degree of inflammation of the urinary tract (eg, number of white blood cells). A urine **culture** can identify bacteria type and antibiotic susceptibility. Patients who have received multiple antibiotic courses are at risk for resistant organisms.

degree of renal impairment. Urine dipsticks are also commonly performed, but they have a high rate of false-positive and negative results. Microscopic **urinalysis** is more accurate as it provides quantitative data on the degree of inflammation of the urinary tract (eg, number of white blood cells). A urine **culture** can identify bacteria type and antibiotic susceptibility. Patients who have received multiple antibiotic courses are at risk for resistant organisms.

A mid-stream clean-catch **(Choice B)** urine specimen is appropriate testing for patients who do not wear diapers. The external genitalia should be thoroughly cleaned to prevent contamination by skin flora. However, infants and toddlers in diapers should undergo **straight catheterization** of the urethra to obtain a sterile urine specimen. Clean-catch specimens are unreliable in diapered patients due to a high likelihood of stool or skin flora confounding the result.

(Choice A) Abdominal ultrasonography is the preferred imaging screening modality for renal disease in children due to its lack of radiation. However, the priority in this patient is to obtain urinalysis and urine culture, followed by antibiotics.

(Choice C) Noncontrast abdominal CT scan is the gold standard for diagnosing nephrolithiasis. This patient's fever, leukocytosis, elevated creatinine, and young age make UTI more likely.

(Choice D) Intravenous pyelogram was previously the gold standard for delineating renal disease. It is now rarely used due to substantial radiation exposure and ready access to renal ultrasound and CT imaging.

Educational objective:
Urinalysis and urine culture should be performed as preliminary studies in all children with suspected urinary tract infection. Patients in diapers should undergo straight catheterization to obtain a sterile specimen and avoid contamination with stool or skin flora.

References

- Urinary tract infection: clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months.

A 15-year-old boy comes to the physician because of hematuria and lower abdominal pain. This is his third episode of hematuria in the past 2 years. He has a family history of renal disease. His temperature is 37.1° C (98.9° F), blood pressure is 140/90 mm Hg, pulse is 80/min, and respirations are 14/min. Examination shows mild sensorineural deafness bilaterally. Urinalysis shows hematuria and proteinuria. Laboratory studies show BUN of 50 mg/dL and serum creatinine of 3.1 mg/dL; serum complement levels are normal. Renal biopsy shows foam cells, and immunofluorescence shows no immunoglobulins or complement. Electron microscopy shows alternating areas of thinned and thickened capillary loops with splitting of GBM. Which of the following is the most likely diagnosis?

- A. Alport's syndrome
- B. Acute interstitial nephritis
- C. Acute post infectious glomerulonephritis
- D. Anti-glomerular basement membrane disease
- E. Benign recurrent hematuria
- F. Goodpasture's syndrome
- G. Henoch-Schonlein purpura
- H. Idiopathic anti-GBM antibody mediated glomerulonephritis
- I. IgA nephropathy
- J. Mixed essential cryoglobulinemia
- K. Microscopic polyangiitis
- L. Systemic lupus erythematosus

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- K. Microscopic polyangiitis
- L. Systemic lupus erythematosus
- M. Thin basement membrane disease
- N. Wegener's granulomatosis

Submit

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- A. Alport's syndrome [87%]
- B. Acute interstitial nephritis [0%]
- C. Acute post infectious glomerulonephritis [0%]
- D. Anti-glomerular basement membrane disease [1%]
- E. Benign recurrent hematuria [0%]
- F. Goodpasture's syndrome [1%]
- G. Henoch-Schonlein purpura [0%]
- H. Idiopathic anti-GBM antibody mediated glomerulonephritis [1%]
- I. IgA nephropathy [1%]
- J. Mixed essential cryoglobulinemia [0%]
- K. Microscopic polyangiitis [0%]
- L. Systemic lupus erythematosus [0%]

- ✔ A. Alport's syndrome [87%]
- B. Acute interstitial nephritis [0%]
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- D. Anti-glomerular basement membrane disease [1%]
- E. Benign recurrent hematuria [0%]
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- J. Mixed essential cryoglobulinemia [0%]
- K. Microscopic polyangiitis [0%]
- L. Systemic lupus erythematosus [0%]
- M. Thin basement membrane disease [1%]
- N. Wegener's granulomatosis [0%]

Omitted
Correct answer
A

87%
Answered correctly

18 Seconds
Time Spent

08/09/2018
Last Updated

E. Systemic lupus erythematosus [0%]

M. Thin basement membrane disease [1%]

N. Wegener's granulomatosis [0%]

Omitted
Correct answer
A

87%
Answered correctly

18 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The above vignette illustrated the classic presentation of Alport's syndrome. This is a familial disorder which usually presents in childhood as recurrent gross hematuria and proteinuria. Sensorineural deafness usually occurs. Electron microscopy findings include alternating areas of thinned and thickened capillary loops with splitting of the glomerular basement membrane (GBM).

(Choice M) Thin basement membrane disease is also a familial disorder, but it presents in adulthood as microscopic hematuria without proteinuria. Renal biopsy reveals a markedly thinned basement membrane.

(Choice E) Benign recurrent hematuria is asymptomatic. Renal biopsy is normal in most cases. This condition has an excellent prognosis.

Educational Objective:
Suspect Alport's syndrome in patients with recurrent episodes of hematuria, sensorineural deafness and a family history of renal failure.

A 5-year-old girl with chronic renal insufficiency is brought to the physician for a follow-up visit. Since birth, she has had multiple episodes of urinary tract infections, for which she takes trimethoprim-sulfamethoxazole daily for prophylaxis. She has a history of poor growth and mild hypertension but is otherwise developmentally normal. Her mother lost custody of the girl 3 years ago due to failure to comply with recommended treatment and prophylaxis of her infections. The girl has since lived with her grandmother. Examination shows mild bilateral lower-extremity edema but no other abnormalities. Urinalysis shows mild proteinuria but no white blood cells or bacteria. Renal scintigraphy with dimercaptosuccinic acid shows bilateral focal parenchymal scarring and blunted calyces. Which of the following is the most likely predisposing factor for this patient's recurrent infections?

- A. Common variable immunodeficiency
- B. Neurogenic bladder
- C. Posterior urethral valves
- D. Polycystic kidney disease
- E. Recurrent sexual abuse
- F. Unilateral renal agenesis
- G. Vesicoureteral reflux

Submit

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- A. Common variable immunodeficiency [1%]
- B. Neurogenic bladder [0%]
- C. Posterior urethral valves [11%]
- D. Polycystic kidney disease [5%]
- E. Recurrent sexual abuse [0%]
- F. Unilateral renal agenesis [0%]
- ✔ G. Vesicoureteral reflux [81%]

Omitted
Correct answer
G

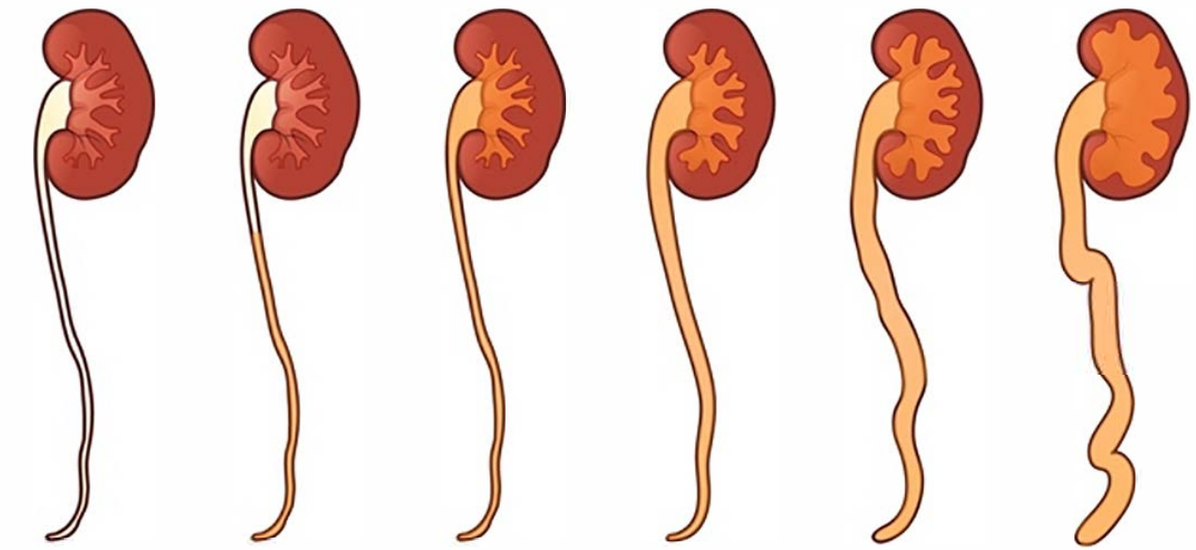
81%
Answered correctly

3 Seconds
Time Spent

08/13/2018
Last Updated

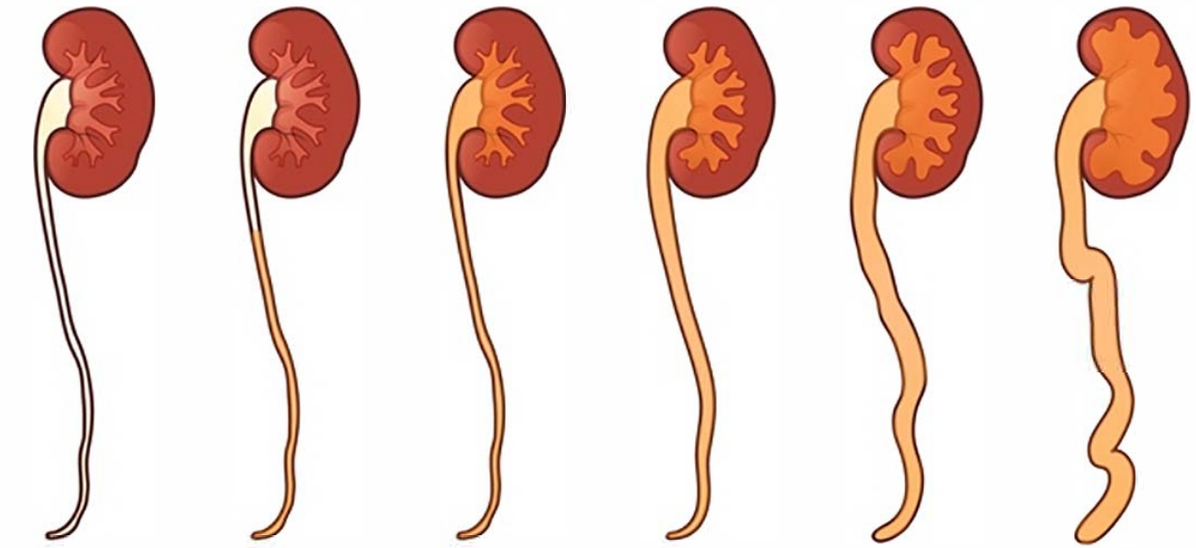
Explanation

Vesicoureteral reflux



Normal Grade I Grade II Grade III Grade IV Grade V

Grade	Description
I	Into a nondilated ureter
II	Into the pelvis & calyces without dilation
III	Mild to moderate dilation of the ureter, renal pelvis & calyces, with minimal blunting of the fornices
IV	Moderate ureteral tortuosity & dilation of the pelvis & calyces
V	Gross dilation of the ureter, pelvis & calyces; loss of papillary



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III	Mild to moderate dilation of the ureter, renal pelvis & calyces, with minimal blunting of the fornices
IV	Moderate ureteral tortuosity & dilation of the pelvis & calyces
V	Gross dilation of the ureter, pelvis & calyces; loss of papillary impressions; ureteral tortuosity

Recurrent urinary tract infections (UTIs) in infants and children are a serious problem as they often involve the kidney and signify a congenital urinary tract anomaly. One of the most common abnormalities is primary **vesicoureteral reflux** (VUR). Normal urine should have unidirectional flow from the kidneys, ureters, bladder, and out the urethra. Patients with severe VUR have urinary reflux from the bladder into the kidney, and the regurgitant urine causes dilation of the ureters (hydroureter) and kidneys (hydronephrosis).

The definitive diagnosis of VUR is made by contrast **voiding cystourethrogram**. Renal ultrasound is performed to screen for hydronephrosis. Recurrent and/or chronic pyelonephritis can lead to blunting of calices (calyceal clubbing) and focal parenchymal scarring. Renal scintigraphy with dimercaptosuccinic acid is the preferred modality for long-term evaluation for renal scarring. Renal function should be followed by serial creatinine. Patients should be monitored closely for complications of **chronic renal insufficiency**, such as hypertension and anemia.

(Choice A) Patients with common variable immunodeficiency typically have recurrent sinopulmonary or gastrointestinal infections.

(Choice B) Neurogenic bladder can cause recurrent UTIs due to urine stasis and secondary reflux from inadequate voiding. It is unlikely due to lack of other neurologic deficits in this patient.

(Choice C) **Posterior urethral valves** are the most common cause of chronic renal insufficiency/failure in children. This distal urinary tract obstruction can cause secondary urinary reflux but the condition affects only boys.

(Choice D) Autosomal recessive polycystic kidney disease manifests in infancy as large flank masses, respiratory distress from pulmonary hypoplasia, and Potter faces (flattened ears/nose, micrognathia from oligohydramnios). Autosomal dominant polycystic kidney disease is usually asymptomatic in childhood.

(Choice E) Although frequent sexual intercourse is a risk factor for recurrent UTIs in women, urinary tract anomalies are the principal cause in infants and children.

(Choice F) Most patients with a solitary kidney are asymptomatic.

Educational objective:

(Choice A) Patients with common variable immunodeficiency typically have recurrent sinopulmonary or gastrointestinal infections.

(Choice B) Neurogenic bladder can cause recurrent UTIs due to urine stasis and secondary reflux from inadequate voiding. It is unlikely due to lack of other neurologic deficits in this patient.

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(Choice E) Although frequent sexual intercourse is a risk factor for recurrent UTIs in women, urinary tract anomalies are the principal cause in infants and children.

(Choice F) Most patients with a solitary kidney are asymptomatic.

Educational objective:
Severe vesicoureteral reflux can cause recurrent or chronic pyelonephritis. Complications include parenchymal scarring, hypertension, and renal insufficiency. Definitive diagnosis is made by voiding cystourethrogram.

References

- Summary of the AUA guideline on management of primary vesicoureteral reflux in children.
- Urinary tract infection: clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months.

Recurrent urinary tract infections (UTIs) in infants and children are a serious problem as they often involve the kidney and signify a congenital urin
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(Choice D) Au
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(Choice E) Al
cause in infant

(Choice F) Mo

Educational o
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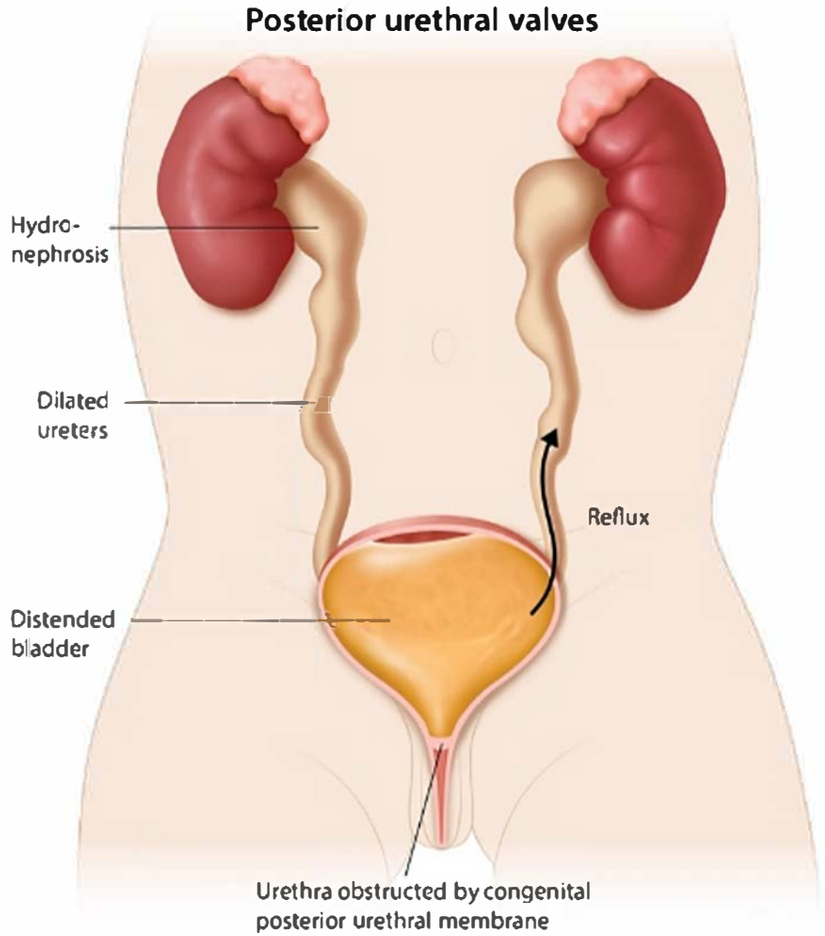

Zoom In | Zoom Out | Reset | Add To Flash Card

A 30-year-old African American man comes to the office due to a 2-week history of fatigue and ankle edema. The patient has a history of HIV. He does not use tobacco, alcohol, or drugs. Temperature is 36.7 C (98 F), blood pressure is 140/86 mm Hg, and respirations are 16/min. Physical examination shows mild ankle edema. Laboratory results are as follows:

Hemoglobin	12.5 g/dL
Leukocytes	6,000/mm^3
Platelets	140,000/mm^3
Sodium	135 mEq/L
Potassium	5.0 mEq/L
Blood urea nitrogen	28 mg/dL
Creatinine	2.4 mg/dL

Urinalysis reveals 3+ proteinuria but otherwise shows no abnormalities. CD4 count taken 3 weeks ago was 550/mm^3. Which of the following is the most probable form of kidney disease in this patient?

A. Membranous glomerulonephritis
B. Mesangioproliferative glomerulonephritis
C. Focal segmental glomerulosclerosis
D. Diffuse proliferative glomerulonephritis
E. Acute interstitial nephritis

You have not answered this question.
Do you wish to continue without answering?
YES
No

Hemoglobin	12.5 g/dL
Leukocytes	6,000/mm³
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Urinalysis reveals 3+ proteinuria but otherwise shows no abnormalities. CD4 count taken 3 weeks ago was 550/mm^3. Which of the following is the most probable form of kidney disease in this patient?

A. Membranous glomerulonephritis [14%]
B. Mesangioproliferative glomerulonephritis [4%]
✔ C. Focal segmental glomerulosclerosis [73%]
D. Diffuse proliferative glomerulonephritis [4%]
E. Acute interstitial nephritis [2%]

Leukocytes	6,000/mm³
Platelets	140,000/mm³
Sodium	135 mEq/L
Potassium	5.0 mEq/L
Blood urea nitrogen	28 mg/dL
Creatinine	2.4 mg/dL

Urinalysis reveals 3+ proteinuria but otherwise shows no abnormalities. CD4 count taken 3 weeks ago was 550/mm³. Which of the following is the most probable form of kidney disease in this patient?

- A. Membranous glomerulonephritis [14%]
- B. Mesangioproliferative glomerulonephritis [4%]
- ✔ C. Focal segmental glomerulosclerosis [73%]
- D. Diffuse proliferative glomerulonephritis [4%]
- E. Acute interstitial nephritis [2%]

Omitted
Correct answer
C

73%
Answered correctly

5 Seconds
Time Spent

12/16/2018
Last Updated

Omitted
Correct answer
C

73%
Answered correctly

5 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

HIV is associated with several forms of kidney damage. The classic and most common one is a form of collapsing **focal segmental glomerulosclerosis (FSGS)**, referred to as HIV-associated nephropathy (HIVAN). Typical presentation includes **heavy** (eg, nephrotic-range) **proteinuria** with **rapid development of renal failure**. Although associated with advanced HIV disease, HIVAN can manifest even if with normal CD4 count and suppressed HIV viral load. This condition is more prevalent among African-Americans, possibly due to differences in the apolipoprotein L1 (*APOL1*) gene.

(Choices A, B, and D) Other forms of HIV-related glomerulopathies that can present with nephrotic-range proteinuria are membranous glomerulonephritis (which tends to have a slower progression and is often seen in association with hepatitis B infection), mesangioproliferative glomerulonephritis, and diffuse proliferative glomerulonephritis. These conditions are less common than FSGS.

(Choice E) Interstitial nephritis can occur during HIV, but the clinical scenario described (eg, heavy proteinuria) is unusual for interstitial nephritis, which can cause fever, rash, eosinophilia (and possible eosinophiluria), and white cells casts.

Educational objective:
Collapsing focal segmental glomerulosclerosis is the most common form of glomerulopathy associated with HIV. Typical presentation includes heavy (eg, nephrotic-range proteinuria), azotemia, rapid development or renal failure, and normal sized kidneys.

An 83-year-old woman is brought to the emergency department from her nursing home due to poor appetite, fatigue, diarrhea, and confusion. The patient has a history of moderate dementia, hypertension, mild intermittent asthma, and bilateral knee osteoarthritis. She was recently hospitalized with pneumonia for which she was treated with broad-spectrum antibiotics. The patient was discharged to the nursing home in stable condition and had normal laboratory values 2 weeks ago. Since that time, she has had worsening diarrhea. The diarrhea is foul-smelling but does not contain any blood. She has mild abdominal discomfort. Temperature is 37.6 C (99.7 F), blood pressure is 96/54 mm Hg, pulse is 112/min, and respirations are 14/min. She has poor skin turgor. There is no peripheral edema. The patient's lungs are clear to auscultation. The abdomen is soft, but she has mild generalized tenderness without rebound or guarding. Laboratory results are as follows:

Sodium	121 mEq/L
Potassium	3.8 mEq/L
Chloride	110 mEq/L
Bicarbonate	10 mEq/L
Blood urea nitrogen	62 mg/dL
Creatinine	1.5 mg/dL
Blood glucose	98 mg/dL
Hemoglobin	12.9 g/dL
Leukocytes	18,000/mm^3
Platelets	163,000/mm^3

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Bicarbonate	10 mEq/L
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Creatinine	1.5 mg/dL
Blood glucose	98 mg/dL
Hemoglobin	12.9 g/dL
Leukocytes	18,000/mm^3
Platelets	163,000/mm^3

A stool specimen tests positive for *Clostridium difficile* toxin. Which of the following is most likely present in this patient?

A. High antidiuretic hormone, high urine sodium
B. High renin, high aldosterone, high antidiuretic hormone
C. High renin, high aldosterone, low antidiuretic hormone
D. Low antidiuretic hormone, low urine sodium
E. Low renin, low aldosterone, high antidiuretic hormone

Submit

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Leukocytes	18,000/mm³
Platelets	163,000/mm³

A stool specimen tests positive for *Clostridium difficile* toxin. Which of the following is most likely present in this patient?

- A. High antidiuretic hormone, high urine sodium [15%]
- ✔ B. High renin, high aldosterone, high antidiuretic hormone [63%]
- C. High renin, high aldosterone, low antidiuretic hormone [6%]
- D. Low antidiuretic hormone, low urine sodium [2%]
- E. Low renin, low aldosterone, high antidiuretic hormone [11%]

Omitted
Correct answer
B

63%
Answered correctly

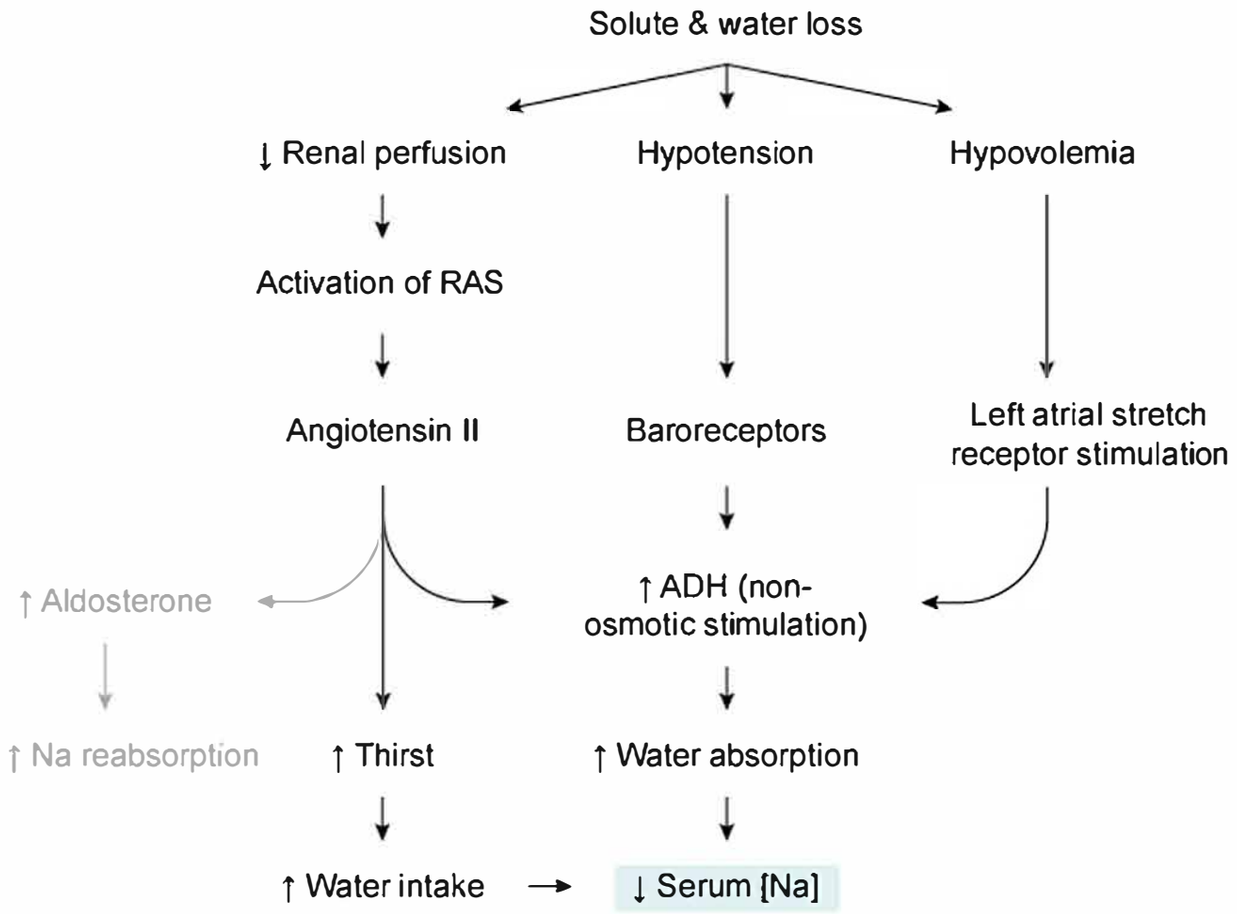
4 Seconds
Time Spent

10/22/2018
Last Updated

Explanation

Mechanism of hypovolemic hyponatremia

Mechanism of hypovolemic hyponatremia



ADH = antidiuretic hormone; **Na** = sodium; **RAS** = renin-angiotensin-aldosterone system.

An assessment of **volume status** is essential in diagnosing and treating hyponatremia (serum sodium <135 mEq/L). This patient's recent history of poor oral intake and diarrhea due to *Clostridium difficile* infection, laboratory evidence of prerenal azotemia (eg, blood urea nitrogen/creatinine ratio >20), and examination findings of tachycardia, hypotension, decreased skin turgor, and absence of peripheral edema strongly suggest hypovolemia (depletion of salt and water).

Hypovolemic hyponatremia occurs due to a multiple-pathway mechanism that illustrates the body's priority to restore euvolemia at the risk of developing hypotonicity:

- Decreased renal perfusion leads to decreased renal tubular sodium delivery, which stimulates the **renin-angiotensin-aldosterone** system and increases sodium reabsorption. (Angiotensin II also stimulates thirst, which leads to increased water intake.)
- **Nonosmotic stimulation of antidiuretic hormone (ADH)** occurs in response to angiotensin II, hypovolemia (stimulates stretch receptors in the left atrium), and **hypotension** (stimulates baroreceptors in the carotid arteries).

Consequent salt and **water retention** help correct the hypovolemia. However, in the setting of ongoing ADH secretion, **hypotonic** hypovolemic hyponatremia can develop due to retention of a relative excess of total body water. **ADH** levels will remain **high** (not low) until hypovolemia is corrected **(Choice C)**. Infusion of normal saline is the treatment of choice for hypovolemic hyponatremia as it replenishes the body's depleted salt stores, restores euvolemia, and shuts off nonosmotic stimuli for ADH release.

(Choices A and E) High ADH and high urine sodium are characteristic of the syndrome of inappropriate ADH secretion, which is a common cause of euvolemic hyponatremia. Low renin and low aldosterone would also be expected. This patient has hypovolemic, rather than euvolemic, hyponatremia and would be expected to have low urine sodium.

(Choice D) Low ADH and low urine sodium may be observed in a patient with central diabetes insipidus, which typically presents with polyuria, polydipsia, and normal to high serum sodium levels.

Educational objective:
Hypovolemic hyponatremia occurs due to nonosmotic stimulation of antidiuretic hormone (ADH) secretion in response to hypovolemia,

intake.)

- **Nonosmotic stimulation of antidiuretic hormone (ADH)** occurs in response to angiotensin II, hypovolemia (stimulates stretch receptors in the left atrium), and **hypotension** (stimulates baroreceptors in the carotid arteries).

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Educational objective:
Hypovolemic hyponatremia occurs due to nonosmotic stimulation of antidiuretic hormone (ADH) secretion in response to hypovolemia, hypotension, and decreased renal perfusion (via angiotensin II). Restoration of blood volume shuts off nonosmotic stimulation of ADH and corrects the hyponatremia.

References

- Hyponatremia: a prospective analysis of its epidemiology and the pathogenetic role of vasopressin.
- Diagnosis and management of hyponatremia in hospitalised patients.

A 51-year-old man is admitted to the hospital because of renal failure. His past medical history is significant for recurrent episodes of bilateral flank pain over the past several years as well as nocturia 2 to 3 times per night for the past 10 years. He has no weight loss. On physical examination, his blood pressure is 164/100 mm Hg. The liver is enlarged and a mass is felt at the right flank on deep palpation. Which of the following is the most likely diagnosis?

A. Horseshoe kidney
B. Nephrolithiasis
C. Papillary necrosis
D. Polycystic kidney disease
E. Renal cell carcinoma

Submit

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- A. Horseshoe kidney [3%]
- B. Nephrolithiasis [3%]
- C. Papillary necrosis [2%]
- ✔ D. Polycystic kidney disease [78%]
- E. Renal cell carcinoma [12%]

Omitted
Correct answer
D

78%
Answered correctly

2 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

This man most likely has autosomal dominant polycystic kidney disease (ADPKD). ADPKD is one of the most common hereditary diseases in the United States and accounts for 10% of dialysis patients. Patients will often have hypertension and palpable kidneys on exam. Please note: the enlarged right kidney is easier to palpate because it lies lower than the left kidney! The liver might be enlarged due to cystic involvement.

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(Choice A) Horseshoe kidney is a congenital abnormality that can cause complications such as ureteropelvic junction obstruction, renal stones, vesicoureteric reflux, and chronic urinary tract infections. Horseshoe kidney does not typically cause pain and would not be palpable on physical examination.

(Choice B) Nephrolithiasis is a common complication of PKD, occurring in 20% of affected patients. It can cause pain but would not produce a palpable renal mass unless it has caused severe hydronephrosis. Severe hydronephrosis usually causes acute renal failure, whereas the abnormalities in the patient described above have been more chronic in nature.

(Choice C) Papillary necrosis refers to ischemic necrosis in the renal papillae. Analgesic overuse is the most common cause, but diabetes mellitus, infections, urinary tract obstruction, hemoglobinopathies, cirrhosis, congestive heart failure, shock, and hemophilia can also be associated. There would not be a palpable mass on physical examination.

(Choice E) Renal cell carcinoma does not seem to occur with increased frequency in patients with APKD when compared to the general population. Also, it is less likely given no other symptoms such as weight loss or loss of appetite. RCC alone would not give renal failure.

Educational objective:
Autosomal dominant polycystic kidney disease is a heritable form of renal disease characterized by multiple renal cysts and intermittent flank pain, hematuria, urinary tract infections, and nephrolithiasis.

A 40-year-old man comes to the physician because of a two-week history of fatigue, lower extremity edema, and dark urine. He has no history of serious illnesses. He takes no medications. He does not use tobacco, alcohol, or drugs. His blood pressure is 132/83 mm Hg and pulse is 84/min. Physical examination shows symmetric pitting edema of lower extremities. Laboratory studies show a serum creatinine level of 1.1 mg/dL. Urinalysis shows 4+ proteinuria and microhematuria. Electron microscopy of the specimen obtained from kidney biopsy shows dense deposits within glomerular basement membrane. Immunofluorescence microscopy is positive for C3, not immunoglobulins. Which of the following is the most likely pathophysiologic mechanism that explains this patient's condition?

A. Anti-GBM antibodies
B. Circulating immune complexes
C. Persistent activation of the alternative complement pathway
D. Cell-mediated injury
E. Non-immunologic damage

Submit

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A. Anti-GBM antibodies [13%]
B. Circulating immune complexes [34%]
✔ C. Persistent activation of the alternative complement pathway [42%]
D. Cell-mediated injury [5%]
E. Non-immunologic damage [4%]

Omitted
Correct answer
C

42%
Answered correctly

2 Seconds
Time Spent

08/04/2018
Last Updated

Explanation

This patient with nephrotic-range proteinuria and hematuria most likely has membranoproliferative glomerulonephritis. Dense

Omitted
Correct answer
C

42%
Answered correctly

2 Seconds
Time Spent

08/04/2018
Last Updated

Explanation

This patient with nephrotic-range proteinuria and hematuria most likely has membranoproliferative glomerulonephritis. Dense intramembranous deposits that stain for C3 is a characteristic microscopic finding for membranoproliferative glomerulonephritis, type 2 (also called dense deposit disease). This condition is unique among glomerulopathies, because it is caused by IgG antibodies (termed *C3 nephritic factor*) directed against C3 convertase of the alternative complement pathway. These antibodies reacting with C3 convertase lead to persistent complement activation and kidney damage.

(Choice A) Anti-GBM antibodies are characteristic for Goodpasture's syndrome.

(Choice B) Circulating immune complexes account for the group of glomerulonephritis called immune complex-mediated glomerulopathies that include SLE, post-streptococcal glomerulonephritis, etc.

(Choice D) Cell-mediated injury may be important in idiopathic crescentic glomerulonephritis.

(Choice E) Non-immunologic kidney damage is believed to operate in diabetic nephropathy, hypertensive nephropathy, etc.

Educational objective:
Membranoproliferative glomerulonephritis, type 2, is a unique glomerulopathy that is caused by persistent activation of the alternative complement pathway.

A 65-year-old man is found confused and lethargic on the floor of his apartment. His medical history includes hypertension, diabetes mellitus, coronary artery disease, transient ischemic attacks, chronic renal disease, and osteoarthritis. The patient's temperature is 37.3 C (99.2 F) and blood pressure is 137/67 mm Hg. He has palpable pulses bilaterally. Right-sided weakness and facial droop are noted on initial assessment. Laboratory results are as follows:

Serum chemistry

Sodium	133 mEq/L
Potassium	7.4 mEq/L
Chloride	104 mEq/L
Bicarbonate	18 mEq/L
Blood urea nitrogen	36 mg/dL
Creatinine	2.9 mg/dL
Calcium	8.4 mg/dL
Glucose	208 mg/dL

ECG is shown in this **exhibit**.

Which of the following is the most appropriate next step in management of this patient?

A. Direct current cardioversion
B. Intravenous amiodarone

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ECG is shown

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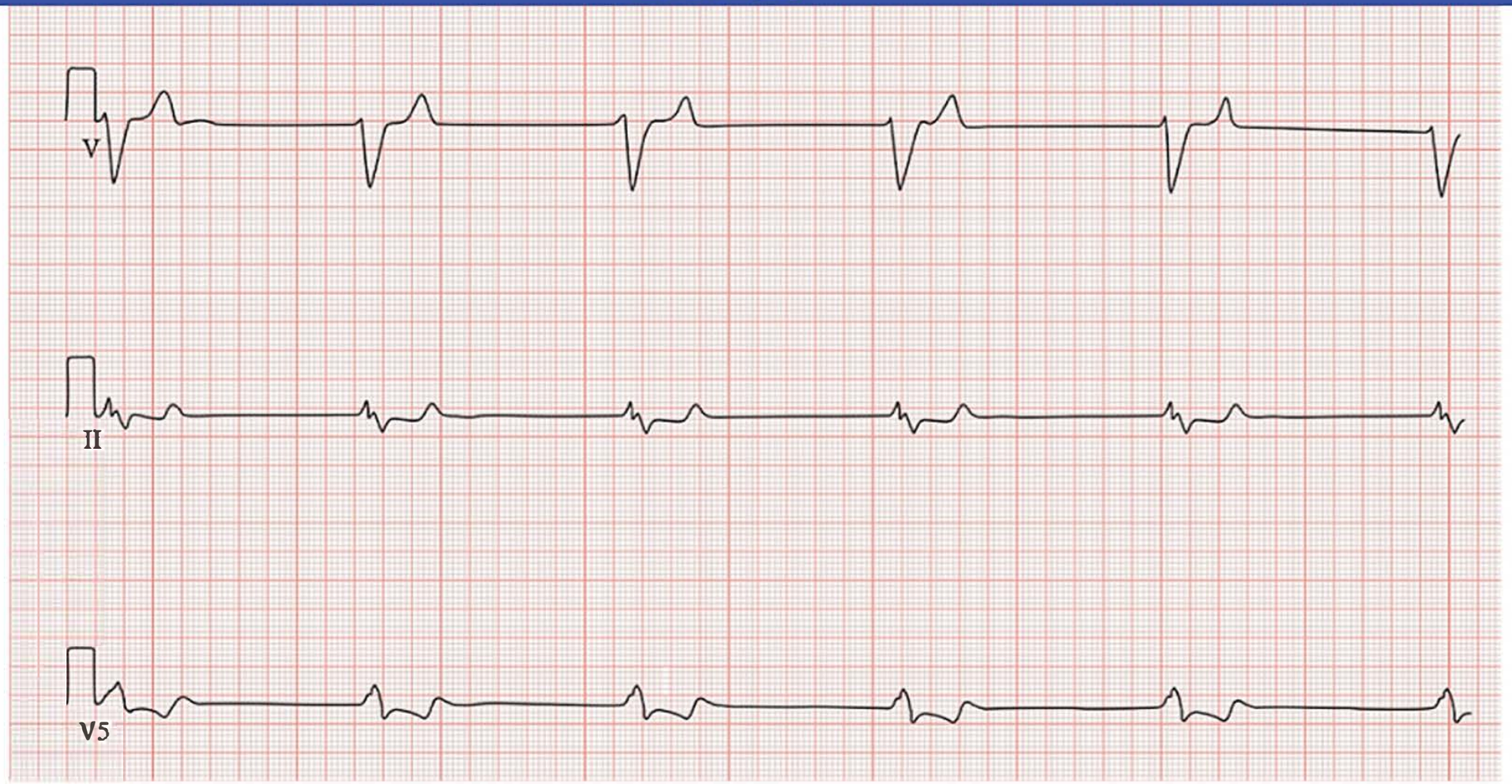

Zoom In | Zoom Out | Reset | Add To Flash Card

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- C. Intravenous calcium gluconate
- D. Sodium polystyrene sulfonate
- E. Subcutaneous insulin
- F. Transvenous pacemaker insertion

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ECG is shown in this **exhibit**.

Which of the following is the most appropriate next step in management of this patient?

A. Direct current cardioversion [5%]

B. Intravenous amiodarone [1%]

C. [illegible]

Bicarbonate	18 mEq/L
Blood urea nitrogen	36 mg/dL
Creatinine	2.9 mg/dL
Calcium	8.4 mg/dL
Glucose	208 mg/dL

ECG is shown in this **exhibit**.

Which of the following is the most appropriate next step in management of this patient?

- A. Direct current cardioversion [5%]
- B. Intravenous amiodarone [1%]
- ✔ C. Intravenous calcium gluconate [79%]
- D. Sodium polystyrene sulfonate [0%]
- E. Subcutaneous insulin [7%]
- F. Transvenous pacemaker insertion [4%]

Omitted
Correct answer
C

79%
Answered correctly

19 Seconds
Time Spent

10/28/2018
Last Updated

C

Explanation

Clinical features of hyperkalemia	
Sequence of ECG changes	• Tall **peaked T waves** with shortened QT interval • **PR prolongation** & **QRS widening** • Disappearance of P wave • Conduction blocks, ectopy, or **sine wave** pattern
Cardiac membrane stabilization	• **Calcium** infusion
Rapidly acting treatment options	• Insulin with glucose • Beta-2 adrenergic agonists • Sodium bicarbonate
Removal of potassium from the body (slow-acting)	• Diuretics • Cation exchange resins • Hemodialysis

This patient with a stroke (unilateral muscular weakness, facial droop, history of transient ischemic attacks) has acute renal failure and **hyperkalemia**. His ECG is notable for a lack of P waves, QRS widening, and bradycardia (approximate heart rate 30/min). The

This patient with a stroke (unilateral muscular weakness, facial droop, history of transient ischemic attacks) has acute renal failure and **hyperkalemia**. His ECG is notable for a lack of P waves, QRS widening, and bradycardia (approximate heart rate 30/min). The patient likely fell due to weakness and was down for a prolonged period, predisposing to rhabdomyolysis and dehydration. He developed acute-on-chronic renal failure and subsequent hyperkalemia. **ECG findings** that can be seen in hyperkalemia include peaked T waves, bradycardia from sinus node dysfunction and atrioventricular block, and arrhythmias (eg, ventricular tachycardia or fibrillation).

Emergent treatment for hyperkalemia is indicated if the serum potassium level is rapidly rising or is ≥6.5 mEq/L, or if there are **ECG changes** attributable to hyperkalemia. The **most immediate** measure is **intravenous calcium** (chloride or gluconate) administration to **stabilize the cardiac myocyte** membrane, making it resistant to the effect of hyperkalemia. Intravenous (not subcutaneous) insulin (in combination with glucose) and/or beta-agonists (eg, albuterol) are used to transiently shift potassium into cells, lowering serum potassium levels **(Choice E)**.

Definitive measures to **reduce total body potassium** are still required and include the following:

1. Reversal of correctable etiology (eg, intravenous fluids for pre-renal azotemia)
2. Diuretics (contraindicated in dehydrated patients)
3. Exchange resins (eg, sodium polystyrene sulfonate **[Choice D]**), which remove potassium via the gastrointestinal tract (effect takes hours)
4. Hemodialysis

(Choices A, B, and F) Nonpharmacologic intervention is required if the patient continues to worsen despite adequate medical therapy or if life-threatening arrhythmias are present. Examples include direct current cardioversion for ventricular fibrillation or transvenous pacemaker insertion for symptomatic bradycardia. Intravenous amiodarone is used to treat stable and unstable ventricular arrhythmias.

Educational objective:

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Educational objective:
Rapid treatment with calcium gluconate is necessary in patients with hyperkalemia who develop significant ECG changes. Temporary (eg, intravenous insulin) and definitive (eg, cation exchange resin, dialysis) measures to reduce serum potassium should follow calcium administration.

A 43-year-old woman presents to the emergency department complaining of confusion. Her other complaints include increased thirst and "using the bathroom more frequently." She denies any fever, chills, headache, chest pain, shortness of breath, or cough. Her past medical history is significant for bipolar disorder that is well-controlled by medication. She does not drink alcohol, smoke cigarettes, or use illicit drugs. Her vital signs are stable, and physical exam is unremarkable. Laboratory studies reveal the following:

Sodium	154 mEq/L
Potassium	4.1 mEq/L
Chloride	116 mEq/L
Bicarbonate	28 mEq/L
Glucose	95 mg/dL
Urine osmolality	250 mOsm/L
Plasma osmolality	326 mOsm/L

What is the most likely cause of this patient's symptoms and laboratory findings?

A. Dehydration
B. Lithium
C. Divalproic acid
D. Craniopharyngioma
E. Head trauma
F. Psychogenic polydipsia

Sodium	154 mEq/L
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What is the most likely cause of this patient's symptoms and laboratory findings?

- A. Dehydration [3%]
- ✔ B. Lithium [89%]
- C. Divalproic acid [1%]
- D. Craniopharyngioma [1%]
- E. Head trauma [1%]
- F. Psychogenic polydipsia [3%]

Sodium	134 mEq/L
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- ✔ B. Lithium [89%]
- C. Divalproic acid [1%]
- D. Craniopharyngioma [1%]
- E. Head trauma [1%]
- F. Psychogenic polydipsia [3%]

Omitted
Correct answer
B

89%
Answered correctly

5 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

Diabetes insipidus (DI) is a leading cause of euvolemic hypernatremia. It typically presents with severe polyuria and mild hypernatremia. It can be divided into two types based on urine osmolality, as well as etiology.

Based on urine osmolality, DI may be complete or partial.

1. Complete DI - the urine osmolality is less than 300 mOsm/kg (often less than 100 mOsm/kg)
2. Partial DI - urine osmolality ranges from 300-600 mOsm/kg.

The serum osmolality is elevated in both types.

Based on etiology, DI may be central or nephrogenic.

1. Central DI is due to decreased production of antidiuretic hormone (ADH). Common causes include trauma, hemorrhage, infection, and tumors.
2. Nephrogenic DI results from renal ADH resistance. Common causes include hypercalcemia, severe hypokalemia, tubulointerstitial renal disease, and medications. The most commonly implicated mediations are lithium, demeclocycline, foscarnet, cidofovir, and amphotericin.

In this vignette, the patient's clinical history, presentation and laboratory findings are suggestive of nephrogenic diabetes insipidus most likely caused by lithium, which is one of the first-line drugs for bipolar disorder.

(Choice A) Dehydration is the hallmark of hypovolemic hypernatremia. Furthermore, patients with this condition have increased urine osmolality.

(Choice C) Divalproic acid is a mood stabilizer that is also used in the treatment of bipolar disorder. Common side effects include nausea, vomiting, somnolence, and weight gain. It is not associated with DI.

(Choice D) Craniopharyngiomas are relatively rare tumors of the sella and suprasellar space. These may cause central DI. The

infection, and tumors.

2. Nephrogenic DI results from renal ADH resistance. Common causes include hypercalcemia, severe hypokalemia, tubulointerstitial renal disease, and medications. The most commonly implicated mediations are lithium, demeclocycline, foscarnet, cidofovir, and amphotericin.

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(Choice C) Divalproic acid is a mood stabilizer that is also used in the treatment of bipolar disorder. Common side effects include nausea, vomiting, somnolence, and weight gain. It is not associated with DI.

(Choice D) Craniopharyngiomas are relatively rare tumors of the sella and suprasellar space. These may cause central DI. The typical presentation includes headaches, focal neurologic changes, or visual problems; which this patient does not have.

(Choice E) Head trauma is another cause of central DI. This is an unlikely cause of the patient's condition, as there is no mention of any head trauma in the history, nor is there any external evidence of trauma on physical examination.

(Choice F) Psychogenic polydipsia results from excessive free water intake by patients who usually have an associated psychiatric condition. Although DI and psychogenic polydipsia both present with euvolemia and polyuria; patients with the former are hypernatremic, whereas those with the latter are hyponatremic.

Educational objective:
Lithium is a common cause of nephrogenic diabetes insipidus. Lithium-induced nephrogenic DI is treated with salt restriction and discontinuation of lithium.

WBC	8,000/cmm
Platelets	200,000/cmm
Serum Na	135 mEq/L
Serum albumin	2.2 g/dL
BUN	16 mg/dL
Serum creatinine	1.0 mg/dL

Urinalysis:

Glucose	Absent
Protein	4+
WBC	1-2/HPF
RBC	Absent
Casts	Fatty casts

This patient is most likely at risk for developing which of the following?

A. Rupture of brain aneurysm
B. Abdominal aortic aneurysm
C. Hypercoagulability
D. Pulmonary hemorrhage

BUN	16 mg/dL
Serum creatinine	1.0 mg/dL

Urinalysis:

Glucose	Absent
Protein	4+
WBC	1-2/HPF
RBC	Absent
Casts	Fatty casts

This patient is most likely at risk for developing which of the following?

- A. Rupture of brain aneurysm
- B. Abdominal aortic aneurysm
- C. Hypercoagulability
- D. Pulmonary hemorrhage
- E. Gall stone pancreatitis

Submit

A 35-year-old man comes to the physician due to a one-month history of weight gain and facial edema. The facial edema resolves at the end of the day, but ankle edema develops. His temperature is 37.2° C (99° F), blood pressure is 142/80 mm Hg, pulse is 80/min, and respirations are 16/min. Examination shows 2+ ankle edema. Laboratory studies show:

Hb	11.0 g/dL
WBC	8,000/cmm
Platelets	200,000/cmm
Serum Na	135 mEq/L
Serum albumin	2.2 g/dL
BUN	16 mg/dL
Serum creatinine	1.0 mg/dL

Urinalysis:

Glucose	Absent
Protein	4+
WBC	1-2/HPF
RBC	Absent
Casts	Fatty casts

This patient is most likely at risk for developing which of the following?

Serum creatinine	1.0 mg/dL

Urinalysis:

Glucose	Absent
Protein	4+
WBC	1-2/HPF
RBC	Absent
Casts	Fatty casts

This patient is most likely at risk for developing which of the following?

- A. Rupture of brain aneurysm [9%]
- B. Abdominal aortic aneurysm [6%]
- ✔ C. Hypercoagulability [62%]
- D. Pulmonary hemorrhage [9%]
- E. Gall stone pancreatitis [11%]

Omitted
Correct answer
C

62%
Answered correctly

4 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The patient in this clinical vignette has nephrotic syndrome. Nephrotic syndrome is a clinical complex characterized by:

1. Proteinuria (> 3-3.5 g/day - most important manifestation)
2. Hypoalbuminemia
3. Edema
4. Hyperlipidemia and lipiduria

The basic pathology is altered permeability of the glomerular membrane for proteins. Diseases most commonly causing nephrotic syndrome are minimal change disease (in children), membranous glomerulopathy (adults), mesangial proliferative glomerulonephritis, membranoproliferative glomerulonephritis, and focal segmental glomerulosclerosis.

Nephrotic syndrome is frequently complicated by hypercoagulation, with a consequent risk of thromboembolic complications. The etiology of hypercoagulation in nephrotic syndrome is multifactorial and includes: increased urinary loss of antithrombin 3, altered levels of protein C and S, increased platelet aggregation, hyperfibrinogenemia due to increased hepatic synthesis, and impaired fibrinolysis. Renal vein thrombosis is the most common manifestation of coagulopathy (especially with membranous glomerulopathy), but arterial thrombosis and pulmonary embolism may also occur. Coagulopathy is less common but more severe in children as compared to adults with nephrotic syndrome.

Complications of nephrotic syndrome include: protein malnutrition, iron-resistant microcytic hypochromic anemia due to transferrin loss, vitamin D deficiency due to increased urinary excretion of cholecalciferol-binding protein, decreased thyroxin levels due to loss of thyroxine-binding globulin, and increased susceptibility to infection.

(Choices A and B) Rupture of a brain aneurysm and abdominal aortic aneurysms are more likely to be seen in patients with adult polycystic kidney disease. Such patients do not present with nephrotic syndrome.

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(Choices A and B) Rupture of a brain aneurysm and abdominal aortic aneurysms are more likely to be seen in patients with adult polycystic kidney disease. Such patients do not present with nephrotic syndrome.

(Choice D) Pulmonary hemorrhage is a manifestation of Goodpasture's disease or granulomatosis with polyangiitis (Wegener's). These cause nephritic (not nephrotic) syndrome.

(Choice E) Gallstone pancreatitis is not a complication of nephrotic syndrome.

Educational Objective:
Nephrotic syndrome is a hypercoagulable condition which manifests as venous or arterial thrombosis, and even pulmonary embolism. Renal vein thrombosis is the most frequent manifestation. Complications of nephrotic syndrome include: protein malnutrition, iron-resistant microcytic hypochromic anemia, increased susceptibility to infection, and vitamin D deficiency.

pain. The patient has chronic intermittent diarrhea and lower back pain for which he takes acetaminophen. He does not use tobacco or illicit drugs. He lives alone and drinks alcohol occasionally. The patient's temperature is 37.6 C (99.7 F), blood pressure is 122/86 mm Hg, pulse is 88/min, and respirations are 25/min. Laboratory results are as follows:

Serum chemistry	
Sodium	132 mEq/L
Potassium	5.0 mEq/L
Chloride	90 mEq/L
Bicarbonate	14 mEq/L
Blood urea nitrogen	19 mg/dL
Creatinine	1.1 mg/dL
Glucose	450 mg/dL
Arterial blood gases	
pH	7.31
PaO_2	90 mm Hg
$PaCO_2$	29 mm Hg

Which of the following best describes this patient's acid-base status?

A. Normal acid-base status

Bicarbonate	14 mEq/L
Blood urea nitrogen	19 mg/dL
Creatinine	1.1 mg/dL
Glucose	450 mg/dL

Arterial blood gases

pH	7.31
PaO_2	90 mm Hg
$PaCO_2$	29 mm Hg

Which of the following best describes this patient's acid-base status?

A. Normal acid-base status
B. Primary metabolic acidosis with respiratory compensation
C. Primary metabolic acidosis without compensation
D. Primary metabolic alkalosis with renal compensation
E. Respiratory acidosis with compensation

Submit

A 32-year-old man with type 1 diabetes mellitus comes to the emergency department with a 2-day history of nausea and abdominal pain. The patient has chronic intermittent diarrhea and lower back pain for which he takes acetaminophen. He does not use tobacco or illicit drugs. He lives alone and drinks alcohol occasionally. The patient's temperature is 37.6 C (99.7 F), blood pressure is 122/86 mm Hg, pulse is 88/min, and respirations are 25/min. Laboratory results are as follows:

Serum chemistry	
Sodium	132 mEq/L
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pH	7.31
PaO_2	90 mm Hg
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Which of the following best describes this patient's acid-base status?

A. Normal acid-base status [0%]
✔ B. Primary metabolic acidosis with respiratory compensation [91%]
C. Primary metabolic acidosis without compensation [7%]
D. Primary metabolic alkalosis with renal compensation [0%]
E. Respiratory acidosis with compensation [0%]

Omitted
Correct answer
B

91%
Answered correctly

4 Seconds
Time Spent

10/31/2018
Last Updated

Explanation

This patient has classic symptoms of diabetic ketoacidosis (DKA), which usually occurs in type 1 diabetics. Typical symptoms of DKA include polyuria, polydipsia, nausea, vomiting, and abdominal pain that may mimic a surgical abdomen. DKA causes a **metabolic acidosis** (pH <7.35) with an **elevated anion gap**, which is calculated as follows:

Anion gap = measured sodium − (measured chloride + measured bicarbonate)

= 132 − (90 + 14) = 28

Metabolic acidosis leads to hyperventilation (deep, rapid breathing called Kussmaul breathing) via a chemoreceptor response, resulting in loss of CO_2. The expected change in $PaCO_2$ can be calculated using Winter's formula:

$PaCO_2 = 1.5*(HCO_3^-) + 8\ (+/-\ 2)$

$= 1.5*(14) + 8\ (+/-\ 2) = 29\ (+/-\ 2)$

This patient's $PaCO_2$ is appropriate for the degree of acidosis and lowered serum bicarbonate level, reflecting adequate **respiratory compensation**. The pH remains slightly acidic as even complete compensation does not restore the pH to normal limits.

(Choice A) Normal acid-base status is a pH of 7.35-7.45, HCO_3^- of 22-28 mEq/L, and $PaCO_2$ of 33-45 mm Hg.

(Choice C) Absence of respiratory compensation would be reflected by a normal $PaCO_2$ (33-45 mm Hg) and a pH that would be much lower than that seen in this patient.

(Choice D) Primary metabolic alkalosis is usually caused by an abnormal accumulation of HCO_3^-. As a result, the filtered load of HCO_3^- in the renal tubules is increased beyond the capability of the kidneys to reabsorb it. Therefore, in metabolic alkalosis, renal

Metabolic acidosis leads to hyperventilation (deep, rapid breathing called Kussmaul breathing) via a chemoreceptor response, resulting in loss of CO_2. The expected change in $PaCO_2$ can be calculated using Winter's formula:

$$PaCO_2 = 1.5*(HCO_3^-) + 8\ (+/-\ 2)$$

$$= 1.5*(14) + 8\ (+/-\ 2) = 29\ (+/-\ 2)$$

This patient's $PaCO_2$ is appropriate for the degree of acidosis and lowered serum bicarbonate level, reflecting adequate **respiratory compensation**. The pH remains slightly acidic as even complete compensation does not restore the pH to normal limits.

(Choice A) Normal acid-base status is a pH of 7.35-7.45, HCO_3^- of 22-28 mEq/L, and $PaCO_2$ of 33-45 mm Hg.

(Choice C) Absence of respiratory compensation would be reflected by a normal $PaCO_2$ (33-45 mm Hg) and a pH that would be much lower than that seen in this patient.

(Choice D) Primary metabolic alkalosis is usually caused by an abnormal accumulation of HCO_3^-. As a result, the filtered load of HCO_3^- in the renal tubules is increased beyond the capability of the kidneys to reabsorb it. Therefore, in metabolic alkalosis, renal HCO_3^- losses automatically increase. The patient's ABG indicates a metabolic acidosis (pH <7.35), not alkalosis.

(Choice E) Respiratory acidosis with compensation would cause a decreased pH, a primarily increased $PaCO_2$, and a secondarily increased HCO_3^-.

Educational objective:
Diabetic ketoacidosis causes an anion gap metabolic acidosis with low pH, low bicarbonate, and compensatory fall in $PaCO_2$ due to Kussmaul breathing (deep, rapid breathing). The pH is restored to near normal levels but never fully corrects to normal limits.

A 55-year-old man comes to the emergency department due to progressively worsening shortness of breath for the past 3 days. He also reports wheezing and productive cough with purulent sputum. The patient's medical history includes chronic bronchitis and benign prostatic hyperplasia. He smokes a pack of cigarettes daily. Temperature is 37.2 C (99 F), blood pressure is 150/90 mm Hg, pulse is 114/min, and respirations are 26/min. The patient's pulse oximetry shows 84% on room air. He is alert and answering questions coherently. Examination shows decreased breath sounds and diffuse wheezing over all lung fields. The patient is admitted to the hospital and started on antibiotics, systemic corticosteroids, and nebulized albuterol and ipratropium. The following day, laboratory results are as follows:

Serum chemistry	
Potassium	2.8 mEq/L
Blood urea nitrogen	28 mg/dL
Creatinine	1.1 mg/dL

Serum chemistry 2 days ago was normal. Which of the following is the most likely cause of this patient's low serum potassium?

A. Decrease in availability of insulin
B. Decrease in extracellular pH
C. Decrease in mineralocorticoid activity
D. Increased beta-adrenergic activity
E. Increased blood cell production
F. Increased gastrointestinal loss

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Creatinine	1.1 mg/dL

Serum chemistry 2 days ago was normal. Which of the following is the most likely cause of this patient's low serum potassium?

- A. Decrease in availability of insulin [2%]
- B. Decrease in extracellular pH [11%]
- C. Decrease in mineralocorticoid activity [5%]
- ✔ D. Increased beta-adrenergic activity [77%]
- E. Increased blood cell production [1%]
- F. Increased gastrointestinal loss [1%]

Omitted
Correct answer
D

77%
Answered correctly

5 Seconds
Time Spent

11/17/2018
Last Updated

Explanation

Low serum potassium may result from increased potassium entry into cells, renal potassium wasting, or gastrointestinal fluid loss.

This patient with an acute exacerbation of chronic obstructive pulmonary disease (COPD) is treated with an inhaled beta-2 adrenergic agent (albuterol). Exogenous and endogenous (eg, epinephrine) **beta-adrenergic agonists** cause a **potassium shift** into the intracellular space by stimulating the sodium-potassium ATPase pump and the sodium-potassium-2 chloride cotransporter. Adrenergic agents also stimulate release of insulin, which further promotes intracellular potassium shift, causing **hypokalemia**.

(Choice A) Insulin promotes potassium shift into the intracellular space and is used in the management of hyperkalemia. Decreased insulin availability would exacerbate hyperkalemia.

(Choice B) Decreased pH (acidosis) with COPD exacerbation may be seen in patients with CO_2 retention. However, this would result in hyperkalemia.

(Choice C) Renal potassium wasting with hypertension is a feature of primary hyperaldosteronism. Decrease in mineralocorticoid activity will lead to hyperkalemia. Diuretics also cause renal potassium loss.

(Choice E) Increased potassium uptake by cells during accelerated hematopoiesis (eg, administration of granulocyte colony-stimulating factors) may cause hypokalemia. This patient has no evidence of increased hematopoiesis.

(Choice F) Lower gastrointestinal fluid levels have a high potassium concentration; therefore, diarrhea is a direct cause of hypokalemia. Vomiting causes metabolic alkalosis, which leads to renal potassium wasting. This patient does not have any gastrointestinal fluid losses.

A 68-year-old Caucasian man is admitted with a diagnosis of left lower lobe pneumonia, and is started on antibiotics. He has a long history of diabetes, hypothyroidism, hypercholesterolemia, and hypertension. He also has diabetic retinopathy, peripheral neuropathy, and nephropathy. He has an arterio-venous fistula placed for a possible dialysis. His medications are insulin, furosemide, atorvastatin, metoprolol and levothyroxine. After having his blood drawn for some laboratory studies today, he bleeds persistently. Laboratory studies show:

Hb	11.5 g/dL
Platelets	160,000/cmm
Blood glucose	178 mg/dL
BUN	56 mg/dL
Serum creatinine	3.5 mg/dL

His baseline creatinine level is between 3.2-3.5 mg/dL. Which of the following is the most likely cause of his bleeding?

A. Disseminated intravascular coagulation
B. Platelet dysfunction
C. Factor VIII deficiency
D. Consumptive coagulopathy
E. Thrombocytopenia

Submit

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BUN	56 mg/dL
Serum creatinine	3.5 mg/dL

His baseline creatinine level is between 3.2-3.5 mg/dL. Which of the following is the most likely cause of his bleeding?

- A. Disseminated intravascular coagulation [7%]
- ✔ B. Platelet dysfunction [63%]
- C. Factor VIII deficiency [9%]
- D. Consumptive coagulopathy [16%]
- E. Thrombocytopenia [4%]

Omitted
Correct answer
B

63%
Answered correctly

3 Seconds
Time Spent

10/10/2018
Last Updated

Explanation

Abnormal hemostasis is a common manifestation seen in patients with chronic renal failure. Abnormal bleeding and bruising are characteristic of uremic coagulopathy. Nowadays, ecchymoses and epistaxis are the only major bleeding manifestations seen due to the advent of dialysis; however, GI bleeding, hemopericardium, subdural hematoma, and bleeding from surgical or invasive sites can still occur due to uremic coagulopathy.

The pathogenesis is multifactorial, but the major defect involves platelet-vessel wall and platelet-platelet interaction. Several uremic toxins have been implicated in the pathogenesis of platelet dysfunction seen in chronic renal failure (CRF), the chief among which is guanidinosuccinic acid. Activated partial thromboplastin (aPTT), prothrombin (PT), and thrombin times (TT) are generally normal. Bleeding time (BT) is reflective of platelet function, and is usually prolonged. The platelet count is normal, but there is platelet dysfunction that causes bleeding.

A number of agents such as desmopressin (DDAVP), cryoprecipitate, and conjugated estrogens have been used to correct the coagulopathy in uremic patients. DDAVP increases the release of factor VIII:von Willebrand factor multimers from endothelial storage sites.

(Choices A, C, D, and E) Disseminated intravascular coagulation, factor VIII deficiency, consumptive coagulopathy, and thrombocytopenia are not common causes of bleeding in uremic patients. Furthermore, this patient's normal platelet count makes the diagnosis of DIC unlikely.

Educational Objective:

Platelet dysfunction is the most common cause of abnormal hemostasis in patients with CRF. PT, PTT, and platelet count are normal

Abnormal hemostasis is a common manifestation seen in patients with chronic renal failure. Abnormal bleeding and bruising are characteristic of uremic coagulopathy. Nowadays, ecchymoses and epistaxis are the only major bleeding manifestations seen due to the advent of dialysis; however, GI bleeding, hemopericardium, subdural hematoma, and bleeding from surgical or invasive sites can still occur due to uremic coagulopathy.

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Educational Objective:
Platelet dysfunction is the most common cause of abnormal hemostasis in patients with CRF. PT, PTT, and platelet count are normal. BT is prolonged. DDAVP is usually the treatment of choice, if needed. DDAVP increases the release of factor VIII:von Willebrand factor multimers from endothelial storage sites. Platelet transfusion is not indicated because the transfused platelets quickly become inactive.

*Extremely high yield question for USMLE!!!

A 27-year-old man comes to the physician because of red urine. He has had no pain or burning on urination. He has infiltrative pulmonary tuberculosis (diagnosed recently). He takes isoniazid, rifampin, and pyrazinamide. He smokes two packs a day and consumes alcohol occasionally. Vital signs are stable. Physical examination shows no abnormalities. Which of the following is the most likely diagnosis?

- A. Acute cystitis
- B. Renal tuberculosis
- C. Drug reaction
- D. Nephrolithiasis
- E. Glomerulopathy

Submit

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- A. Acute cystitis [1%]
- B. Renal tuberculosis [1%]
- ✔ C. Drug reaction [95%]
- D. Nephrolithiasis [0%]
- E. Glomerulopathy [1%]

Omitted
Correct answer
C

95%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The most probable diagnosis in this patient is urine discoloration due to rifampin. It is important to remember that rifampin causes red to orange discoloration of body fluids including urine, saliva, sweat, and tears. It can also cause discoloration of soft contact lenses. Knowing this fact will help you to get an 'easy' diagnosis. Of course, more serious conditions should be ruled out, starting with simple urinalysis. Absence of significant changes on urinalysis (proteinuria, hematuria, and sterile pyuria) essentially rules out renal

B. Renal tuberculosis [1%]

✔ C. Drug reaction [95%]

D. Nephrolithiasis [0%]

E. Glomerulopathy [1%]

Omitted
Correct answer
C

95%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The most probable diagnosis in this patient is urine discoloration due to rifampin. It is important to remember that rifampin causes red to orange discoloration of body fluids including urine, saliva, sweat, and tears. It can also cause discoloration of soft contact lenses. Knowing this fact will help you to get an 'easy' diagnosis. Of course, more serious conditions should be ruled out, starting with simple urinalysis. Absence of significant changes on urinalysis (proteinuria, hematuria, and sterile pyuria) essentially rules out renal tuberculosis or tuberculosis-associated glomerulopathy **(Choice E)**.

(Choices A and D) Hematuria during acute cystitis or nephrolithiasis is usually accompanied by pain and dysuric symptoms.

Educational objective:
Rifampin causes red to orange discoloration of body fluids. 'Red urine' in a patient taking rifampin is usually a benign drug effect.

A 72-year-old woman with poorly controlled type 2 diabetes mellitus presents to your clinic one week after being discharged from the hospital. She had been admitted with pyelonephritis secondary to a multi-drug resistant organism, and received several days of intravenous antibiotics. Her serum creatinine on admission had been 2.1mg/dL. Today it is found to be 4.9 mg/dL. Urinalysis reveals rare epithelial casts and no white blood cells. FE_{Na} is greater than 2%. What antibiotic did she most likely receive during her hospitalization?

- A. Nafcillin
- B. Vancomycin
- C. Levofloxacin
- D. Amikacin
- E. Doxycycline
- F. Azithromycin

Submit

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- A. Nafcillin [9%]
- B. Vancomycin [25%]
- C. Levofloxacin [10%]
- ✔ D. Amikacin [45%]
- E. Doxycycline [3%]
- F. Azithromycin [4%]

Omitted
Correct answer
D

45%
Answered correctly

2 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

This patient has acute renal failure (ARF) in the setting of chronic kidney disease. Of the drugs listed, amikacin is the most likely causative agent. There are several indications that amikacin or another aminoglycoside is the cause of this patient's acute renal

This patient has acute renal failure (ARF) in the setting of chronic kidney disease. Of the drugs listed, amikacin is the most likely causative agent. There are several indications that amikacin or another aminoglycoside is the cause of this patient's acute renal failure. First, she had pyelonephritis with a multidrug-resistant organism, probably a gram-negative rod. Aminoglycosides are commonly used in this setting. Second, her urine sediment does not contain any white blood cells. If she had acute interstitial nephritis (AIN), eosinophils and white blood cell casts would be present in her urine. Third, her elevated FE_{Na} is not consistent with a pre-renal etiology of acute renal failure. Aminoglycosides may be used in patients with renal dysfunction, but their serum levels and the patient's renal function must be followed closely. Because of their adverse effects and need for monitoring, aminoglycosides are being used with decreasing frequency, particularly in older patients.

(Choice A) Nafcillin is a common cause of acute renal failure due to AIN. This drug is used to treat infections caused by methicillin-sensitive Staphylococcus aureus (MSSA). Nafcillin would not have been used to treat multidrug-resistant pyelonephritis and her urinary sediment is not consistent with AIN.

(Choice B) Vancomycin is used to treat infections with methicillin-resistant S. aureus (MRSA). MRSA pyelonephritis is uncommon but may occur in patients who are chronically ill, are institutionalized, or have indwelling bladder catheters. Vancomycin can be nephrotoxic at high doses and it must be dosed according to serum levels. This woman is more likely to have been infected with a gram-negative organism than a gram-positive one, so is unlikely to have received vancomycin.

(Choice C) Levofloxacin is a fluoroquinolone antibiotic often used to treat pyelonephritis. Levofloxacin must be renally dosed but is not a common cause of ARF.

(Choice E) Doxycycline is most often used to treat community acquired pneumonia, some zoonotic infections (e.g., Lyme disease), Chlamydia, and acne. It is not strongly associated with renal dysfunction and is not likely to have been used for multidrug-resistant pyelonephritis.

(Choice F) Azithromycin is a macrolide antibiotic typically used to treat community acquired pneumonia, sinus infections, streptococcal pharyngitis, and Chlamydia. It has approximately 20% renal clearance but is not strongly associated with acute renal

pre-renal etiology of acute renal failure. Aminoglycosides may be used in patients with renal dysfunction, but their serum levels and the patient's renal function must be followed closely. Because of their adverse effects and need for monitoring, aminoglycosides are being used with decreasing frequency, particularly in older patients.

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(Choice F) Azithromycin is a macrolide antibiotic typically used to treat community acquired pneumonia, sinus infections, streptococcal pharyngitis, and Chlamydia. It has approximately 20% renal clearance but is not strongly associated with acute renal failure.

Educational Objective:
Aminoglycosides are antibiotics used to treat serious gram-negative infections. They are potentially nephrotoxic and drugs levels and renal function must be monitored closely during therapy.

A 32-year-old woman is brought to the emergency department due to weakness, tingling, and numbness of her extremities. She takes no medications. Her blood pressure is 110/70 mm Hg, pulse is 90/min, and respirations are 14/min. The physical examination is unremarkable. Laboratory results are as follows:

Serum chemistry	
Sodium	135 mEq/L
Potassium	2.9 mEq/L
Chloride	88 mEq/L
Bicarbonate	37 mEq/L
Blood urea nitrogen	22 mg/dL
Creatinine	0.9 mg/dL
pH	7.56
Urine electrolytes	
Chloride	7 mEq/L (normal >20 mEq/L)
Sodium	16 mEq/L (normal >40 mEq/L)

Which of the following is the most likely cause of this patient's condition?

A. Hypoventilation syndrome [0%]

Bicarbonate	37 mEq/L
Blood urea nitrogen	22 mg/dL
Creatinine	0.9 mg/dL
pH	7.56

Urine electrolytes

Chloride	7 mEq/L (normal >20 mEq/L)
Sodium	16 mEq/L (normal >40 mEq/L)

Which of the following is the most likely cause of this patient's condition?

A. Hypoventilation syndrome
B. Persistent diarrhea
C. Primary hyperaldosteronism
D. Self-induced vomiting
E. Type I renal tubular acidosis

Submit

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Urine electrolytes

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Sodium	16 mEq/L (normal >40 mEq/L)

Which of the following is the most likely cause of this patient's condition?

- A. Hypoventilation syndrome [0%]
- B. Persistent diarrhea [6%]
- C. Primary hyperaldosteronism [11%]
- ✔ D. Self-induced vomiting [70%]
- E. Type I renal tubular acidosis [10%]

Omitted
Correct answer
D

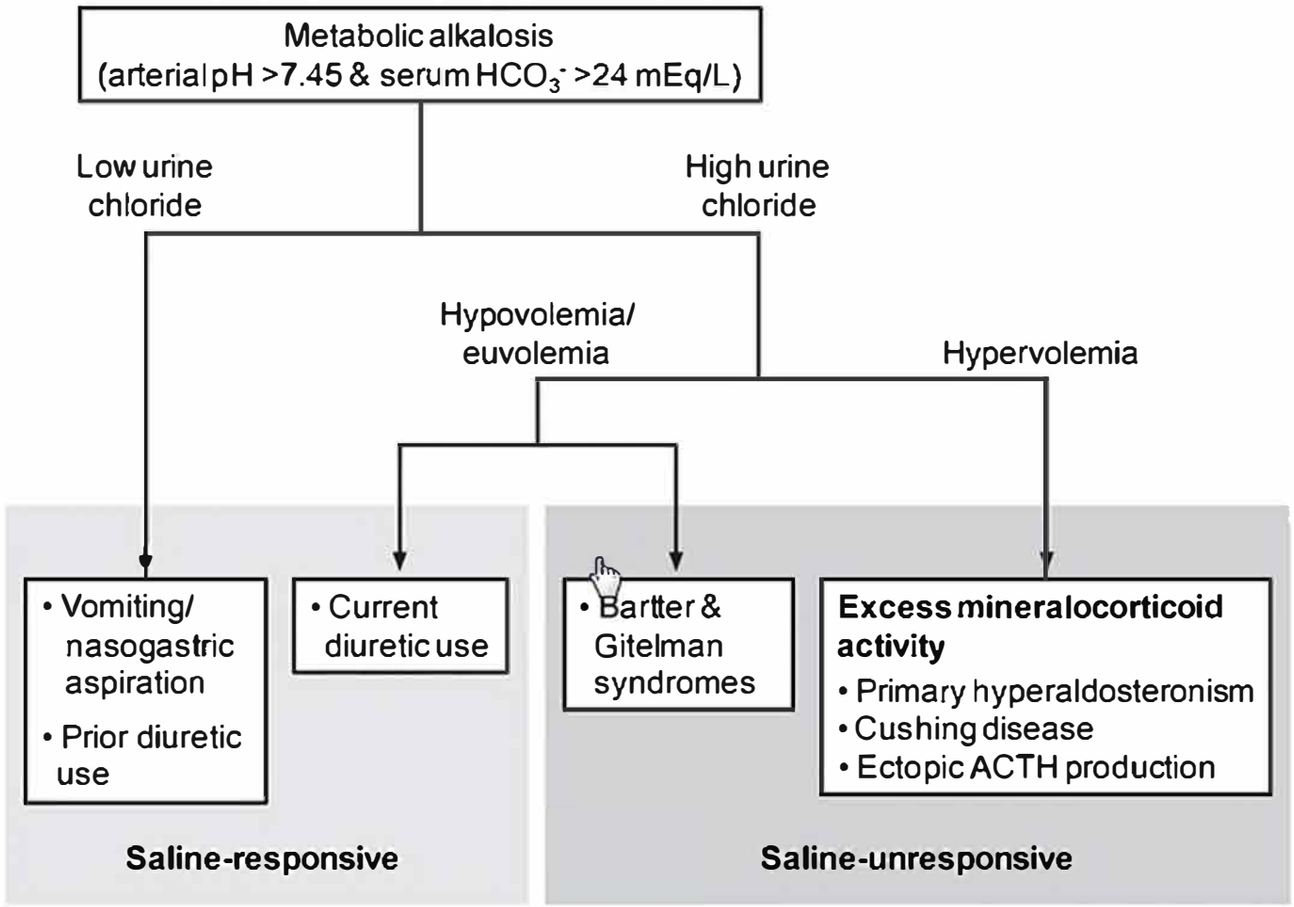
70%
Answered correctly

3 Seconds
Time Spent

08/27/2018
Last Updated

Explanation

Differential diagnosis of metabolic alkalosis



This patient's presentation is consistent with metabolic alkalosis, defined as arterial pH >7.45 and serum bicarbonate level >24 mEq/L. Metabolic alkalosis can be further classified as **saline-responsive** and **saline-unresponsive**. It can also be classified according to low (<20 mEq/L) or higher levels of urinary chloride. Saline-unresponsive metabolic alkalosis typically presents with a higher level of urinary chloride (>20 mEq/L). Patients can have expanded extracellular fluid (ECF) with hypervolemia (eg, primary hyperaldosteronism, Cushing syndrome, excessive black licorice ingestion) or appear hypo/euvolemic (eg, Bartter syndrome, Gitelman syndrome). These conditions require treatment of the underlying disorder; the metabolic alkalosis is not corrected by saline infusion alone.

Saline-responsive metabolic alkalosis is commonly due to loss of gastric secretions (eg, self-induced or spontaneous vomiting, nasogastric suctioning) that results in ECF loss. Patients typically develop volume depletion (eg, hypotension, orthostasis) and low serum Cl^- due to chloride loss in the gastric secretions. The ECF loss leads to increased renal mineralocorticoid levels, increased renal sodium and chloride reabsorption, and increased urinary H^+ and K^+ excretion. The end result is decreased urine chloride, hypokalemia, and metabolic alkalosis. Prior use of thiazide or loop diuretics will also lower urine chloride, but current diuretic use increases urine chloride. Low serum Cl^- also impairs renal HCO_3^- excretion and further worsens the alkalosis. Saline-responsive metabolic alkalosis usually corrects with isotonic saline infusion alone and restores both ECF volume and low serum Cl^-.

(Choice A) Hypoventilation causes respiratory acidosis with high pCO_2 due to CO_2 retention. The kidneys retain HCO_3^- to compensate, leading to metabolic alkalosis. However, pH is usually low (<7.35) in hypoventilation compared to >7.45 in metabolic alkalosis.

(Choices B and E) Persistent diarrhea causes excess HCO_3^- loss in the intestine and leads to non-anion gap metabolic **acidosis**. All forms of renal tubular acidosis also cause non-anion gap metabolic **acidosis**. However, this patient's elevated serum bicarbonate is consistent with metabolic alkalosis instead of acidosis.

(Choice C) Primary hyperaldosteronism increases urinary H^+ and K^+ excretion to cause metabolic alkalosis. Patients also typically develop hypertension, expanded ECF volume, and hypokalemia. However, urine chloride is usually >20 mEq/L as there is no

increases urine chloride. Low serum Cl^- also impairs renal HCO_3^- excretion and further worsens the alkalosis. Saline-responsive metabolic alkalosis usually corrects with isotonic saline infusion alone and restores both ECF volume and low serum Cl^-.

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(Choice C) Primary hyperaldosteronism increases urinary H^+ and K^+ excretion to cause metabolic alkalosis. Patients also typically develop hypertension, expanded ECF volume, and hypokalemia. However, urine chloride is usually >20 mEq/L as there is no stimulus for renal NaCl retention. The metabolic alkalosis in primary hyperaldosteronism requires aldosterone antagonists (or adrenalectomy) and does not respond to saline alone.

Educational objective:
Based on urinary chloride levels and extracellular fluid volume status, metabolic alkalosis can be classified as saline-responsive and saline-unresponsive. Saline-responsive metabolic alkalosis is associated with low urinary chloride excretion and volume contraction, and corrects with saline infusion alone. Saline-unresponsive metabolic alkalosis typically presents with urinary chloride >20 mEq/L.

References

- Metabolic alkalosis.
- Approach to metabolic alkalosis.

A 42-year-old man is found unconscious in a subway station. He is brought to the emergency department where cardiopulmonary resuscitation is performed and intravenous glucose and thiamine are administered. His laboratory profile shows:

pH	7.20
PaO_2	90 mm Hg
$PaCO_2$	28 mm Hg
HCO_3^-	12 mEq/L

Which of the following is the best next step in the diagnosis of this patient's acid-base status?

A. Calculate the plasma anion gap
B. Calculate the plasma osmolal gap
C. Calculate the urine anion gap
D. Calculate the urine osmolar gap
E. Obtain venous blood gas

Submit

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Which of the following is the best next step in the diagnosis of this patient's acid-base status?

- A. Calculate the plasma anion gap [91%]
- B. Calculate the plasma osmolal gap [3%]
- C. Calculate the urine anion gap [2%]
- D. Calculate the urine osmolar gap [1%]
- E. Obtain venous blood gas [1%]

Omitted
Correct answer
A

91%
Answered correctly

2 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

This patient has **metabolic acidosis** (pH <7.35, primary decrease in HCO_3^-). To correctly identify his acid-base status, the best next step is calculation of the **anion gap** (AG). The AG represents the unmeasured anions in the body. It can be calculated with the following formula:

$$AG = Na - (HCO_3^- + Cl)$$

The normal AG is between 10 and 14 mEq/L. An increase in the AG indicates the presence of nonchloride acids that contain inorganic, organic, exogenous or unidentified anions. Finding an **increased anion gap** clinically narrows the differential diagnosis of metabolic acidosis to a few common causes. Some of the most common causes of anion gap metabolic acidosis are:

- Lactic acidosis: Hypoxia, poor tissue perfusion, mitochondrial dysfunction
- Ketoacidosis: Type I diabetes, starvation, or alcoholism
- Methanol ingestion: Formic acid accumulation
- Ethylene glycol ingestion: Glycolic and oxalic acid accumulation
- Salicylate poisoning: Salicylate accumulation (also causes concomitant respiratory alkalosis)
- Uremia (eg, end-stage renal disease): Failure to excrete H^+ as NH_4^+

(Choice B) The osmolal gap should be calculated in selected cases of increased anion gap acidosis, such as when ethanol, methanol, or ethylene glycol toxicity is suspected. It is calculated by determining the difference between the measured and calculated serum osmolality.

(Choice C) The urine anion gap (urine Na + urine K − urine Cl) is calculated when there is a normal anion gap metabolic acidosis. The urine anion gap helps determine if such acidosis is due to the renal or intestinal bicarbonate losses. Renal losses of bicarbonate occur in distal renal tubular acidosis (positive urine anion gap) or carbonic anhydrase inhibitor use. Gastrointestinal loss of bicarbonate occurs classically in diarrhea (negative urine anion gap).

metabolic acidosis to a few common causes. Some of the most common causes of anion gap metabolic acidosis are:

- Lactic acidosis: Hypoxia, poor tissue perfusion, mitochondrial dysfunction
- Ketoacidosis: Type I diabetes, starvation, or alcoholism
- Methanol ingestion: Formic acid accumulation
- Ethylene glycol ingestion: Glycolic and oxalic acid accumulation
- Salicylate poisoning: Salicylate accumulation (also causes concomitant respiratory alkalosis)
- Uremia (eg, end-stage renal disease): Failure to excrete H^+ as NH_4^+

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(Choice C) The urine anion gap (urine Na + urine K − urine Cl) is calculated when there is a normal anion gap metabolic acidosis. The urine anion gap helps determine if such acidosis is due to the renal or intestinal bicarbonate losses. Renal losses of bicarbonate occur in distal renal tubular acidosis (positive urine anion gap) or carbonic anhydrase inhibitor use. Gastrointestinal loss of bicarbonate occurs classically in diarrhea (negative urine anion gap).

(Choice D) Calculation of the urine osmolar gap will not help determine the acid-base status of this patient.

(Choice E) A venous blood gas should differ little from an arterial blood gas except for the decrease in O_2 levels seen in venous blood compared to arterial blood. Venous blood that contains a similar oxygen content as arterial blood may indicate a disorder of respiratory metabolism such as cyanide poisoning.

Educational objective:
The anion gap must always be calculated in patients with metabolic acidosis in order to narrow the differential diagnosis.

A 56-year-old male comes to the emergency room because of a 2-day history of fever, chills, shortness of breath and productive cough. He also threw up once in the emergency room. He has been smoking for several years and occasionally drinks alcohol. On admission, his BP was 90/60, but with one liter of normal saline it improved to 120/80 mm Hg. His temperature is 38.8° C (102° F). His arterial blood gas (ABG) analysis is as follows:

Blood pH	7.53
PaO2	70 mmHg
PaCO2	30 mmHg
HCO3-	22 mEq/L

Which of the following best describes his primary acid-base status?

- A. Respiratory acidosis
- B. Respiratory alkalosis
- C. Metabolic acidosis
- D. Metabolic alkalosis
- E. Normal acid base status

Submit

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Blood pH	7.53
PaO2	70 mmHg
PaCO2	30 mmHg
HCO3-	22 mEq/L

Which of the following best describes his primary acid-base status?

- A. Respiratory acidosis [1%]
- ✔ B. Respiratory alkalosis [88%]
- C. Metabolic acidosis [1%]
- D. Metabolic alkalosis [8%]
- E. Normal acid base status [0%]

Omitted
Correct answer
B

88%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

B

Explanation

Human blood pH is normally maintained at 7.35-7.45. HCO_3^- is normally 24 meq/L, and $PaCO_2$ is normally 40 mm Hg. Any deviation from these normal values is indicative of an acid-base disorder. Metabolic acid-base disorders are due to a primary change in the concentration of HCO_3^-, whereas respiratory acid-base disorders are due to a primary change in PCO_2. A primary rise in $PaCO_2$ (respiratory acidosis) or fall in plasma HCO_3^- (metabolic acidosis) reduces the pH, whereas a primary fall in PCO_2 (respiratory alkalosis) or a rise in plasma HCO_3^- (metabolic alkalosis) increases the pH. In this case, the patient has a respiratory alkalosis because his pH is greater than 7.45 and he has a primary decrease in his $PaCO_2$. He is suffering from pneumonia, which has caused tachypnea due to hypoxia. Hyperventilation results in CO_2 loss and a consequent decrease in $PaCO_2$. Because CO_2 is an acid, CO_2 loss causes alkalosis. The slightly decreased bicarbonate level is due to early renal compensation.

(Choice A) Respiratory acidosis is most commonly caused by respiratory suppression. Potential causes include narcotic overdose, neurologic diseases causing airway muscle weakness and COPD.

(Choice C) Classic causes of metabolic acidosis can be recalled using the mnemonic MUDPILES (anion-gap): Methanol, Uremia (renal failure), DKA, Paraldehyde, INH, Lactic acidosis, Ethylene glycol, Salicylates. Diarrhea and renal tubular acidosis are other causes (non-anion gap).

(Choice D) Metabolic alkalosis is most commonly caused by vomiting. Hyperaldosteronism and volume contraction are other causes.

Educational Objective:
Respiratory alkalosis is characterized by an increased pH and a primary decrease in the $PaCO_2$. Typical causes include hyperventilation due to pneumonia, high altitude or salicylate intoxication.

An 18-year-old African American man comes to the office due to several months of excessive urination. The patient voids every few hours during the day and wakes 2 or 3 times per night to urinate despite restricting his evening fluid intake. He says that his urine appears clear and has no visible blood. Medical history is significant for seasonal allergies, for which he frequently takes an antihistamine. He takes no other medications and does not use tobacco, alcohol, or illicit drugs. The patient is sexually active and does not use condoms. He was adopted at a young age; records indicate that his birth mother had sickle cell disease and died from a stroke at age 32. The patient is afebrile and normotensive. Hematocrit is 41% and serum sodium is 138 mEq/L. Urinalysis of a first-morning specimen reveals the following:

Specific gravity	1.001	(normal: 1.010-1.030)
pH	6.6	(normal: 4.5-8)
Protein	none	
Blood	negative	
Glucose	negative	
Ketones	negative	
Leukocyte esterase	negative	
Nitrites	negative	

pH	6.0	(normal: 4.5-8)
Protein	none	
Blood	negative	
Glucose	negative	
Ketones	negative	
Leukocyte esterase	negative	
Nitrites	negative	

This patient's polyuria is most likely caused by which of the following?

- A. Central diabetes insipidus
- B. Chlamydia infection
- C. Hyposthenuria
- D. Medication adverse effect
- E. Primary polydipsia
- F. Type 1 diabetes mellitus
- G. Type 2 diabetes mellitus

Submit

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pH	6.6	(normal: 4.5-8)
Protein	none	
Blood	negative	
Glucose	negative	
Ketones	negative	
Leukocyte esterase	negative	
Nitrites	negative	

This patient's polyuria is most likely caused by which of the following?

A. Central diabetes insipidus [34%]
B. Chlamydia infection [2%]
C. Hyposthenuria [43%]

Blood	negative
Glucose	negative
Ketones	negative
Leukocyte esterase	negative
Nitrites	negative

This patient's polyuria is most likely caused by which of the following?

- A. Central diabetes insipidus [34%]
- B. Chlamydia infection [2%]
- ✔ C. Hyposthenuria [43%]
- D. Medication adverse effect [5%]
- E. Primary polydipsia [9%]
- F. Type 1 diabetes mellitus [4%]
- G. Type 2 diabetes mellitus [0%]

Omitted
Correct answer
C

43%
Answered correctly

4 Seconds
Time Spent

12/19/2018
Last Updated

Explanation

Sickle cell trait	
Clinical features	• Usually asymptomatic • No change in overall life expectancy
Laboratory findings	• Normal hemoglobin, reticulocyte count, RBC indices & morphology • Hemoglobin electrophoresis: Hb A > Hb S
Complications	• Hematuria/papillary necrosis, hyposthenuria • Splenic infarction (especially at higher altitudes), venous thromboembolism, priapism • Exertional rhabdomyolysis

Hb A = hemoglobin A; **Hb S** = hemoglobin S; **RBC** = red blood cell.

This patient has a **low urine specific gravity** consistent with **hyposthenuria**, the inability of the kidneys to concentrate urine. His family history is significant for early-onset stroke, which can be a severe complication of sickle cell disease (SCD) due to cerebrovascular occlusion from sickling; however, he has a normal hematocrit level and no other symptoms. Therefore, **sickle cell trait** (SCT) is the most likely cause of this patient's hyposthenuria.

Hyposthenuria is common in patients with SCD and may also develop in those with SCT. In response to hypoxic, hyperosmolar conditions of the renal medulla, red blood cells sickle in the vasa recta, impairing free water reabsorption and countercurrent exchange. Patients typically have **polyuria** and nocturia despite fluid restriction. Urine osmolality is low; however, **normal serum sodium** is maintained due to intact antidiuretic hormone (ADH). Urinary diluting capacity is also intact as it is a function of the superficial loop of Henle, which is not supplied by the vasa recta.

Typically, mild hyposthenuria due to SCT requires no treatment. In patients with SCD, red blood cell transfusions often improve urine-

sodium is maintained due to intact antidiuretic hormone (ADH). Urinary diluting capacity is also intact as it is a function of the superficial loop of Henle, which is not supplied by the vasa recta.

Typically, mild hyposthenuria due to SCT requires no treatment. In patients with SCD, red blood cell transfusions often improve urine-concentrating ability and provide relief of symptoms.

(Choice A) Central diabetes insipidus causes polyuria and hyposthenuria due to insufficient ADH production. However, thirst mechanism is impaired and serum sodium is elevated in patients with central diabetes insipidus.

(Choice B) Chlamydia infection in men can cause dysuria and urethral discharge. Impaired urine-concentrating ability is not associated with chlamydia infection, and urinalysis would likely be positive for leukocyte esterase.

(Choice D) Antihistamines (eg, diphenhydramine) treat allergic rhinitis and can cause anticholinergic adverse effects such as dry mouth, tachycardia, and urinary retention, none of which is consistent with this patient's presentation.

(Choice E) Primary (psychogenic) polydipsia causes hyposthenuria due to excessive water intake. However, serum sodium is typically low, and this condition is unlikely in a patient restricting evening fluid intake.

(Choices F and G) Diabetes mellitus is characterized by elevated serum glucose due to lack of insulin (type 1) or insulin resistance (type 2). When serum glucose exceeds the capacity of kidney resorption, glucose draws water into the collecting system, causing polyuria and glucosuria. Negative urine glucose makes this diagnosis unlikely.

Educational objective:
Hyposthenuria is the inability of the kidneys to concentrate urine and can occur in patients with sickle cell disease and sickle cell trait. Patients have polyuria, low urine specific gravity, and normal serum sodium.

References

- Sickle cell disease: renal manifestations and mechanisms.

A 72-year-old man comes to the emergency department due to a 24-hour history of progressive lower abdominal discomfort and difficulty voiding. The patient has never had urinary difficulty in the past. He has a history of hypertension, ischemic stroke with mild left-sided residual weakness, and a recent episode of abdominal shingles. The patient also reports several days of non-productive cough and has been taking over-the-counter diphenhydramine for 2 days. He is a former smoker and does not drink alcohol. Temperature is 36.7 C (98 F), blood pressure is 150/80 mm Hg, and pulse is 105/min. The patient appears restless. Bilateral breath sounds are normal with no added sounds. Cardiac examination reveals regular rhythm. Previous area of shingles on the abdominal wall has no active lesions but the area is hyperesthetic. There is suprapubic fullness and tenderness. A mildly enlarged, nontender prostate is palpated on the rectal examination. Which of the following is the most likely cause of this patient's current condition?

- A. Abdominal muscle weakness
- B. Bladder motor nerve injury
- C. Detrusor hypocontractility
- D. Internal sphincter spasm
- E. Urethral extrinsic compression

Submit

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- A. Abdominal muscle weakness [1%]
- B. Bladder motor nerve injury [7%]
- ✔ C. Detrusor hypocontractility [60%]
- D. Internal sphincter spasm [12%]
- E. Urethral extrinsic compression [17%]

Omitted
Correct answer
C

60%
Answered correctly

3 Seconds
Time Spent

10/20/2018
Last Updated

Explanation

Explanation

This elderly patient with difficulty voiding and a palpable lower abdominal mass (likely representing an overfilled bladder) has **acute urinary retention** due to an adverse effect of diphenhydramine. **First-generation H1-antihistamines** (eg, diphenhydramine, chlorpheniramine, hydroxyzine) also have significant **anticholinergic** effects (eg, at muscarinic receptors of the parasympathetic nervous system), which can manifest as dryness of the eyes, dryness of the oral mucosa and respiratory passages, or urinary retention.

Urinary retention caused by anticholinergic agents results from impaired **detrusor muscle contraction** and, to a lesser extent, impaired internal sphincter relaxation, both of which are controlled by parasympathetic input from the pelvic splanchnic nerves. Elderly men, who are likely to have some degree of underlying urinary obstruction due to **benign prostatic hyperplasia (BPH)**, are at increased risk of developing urinary retention due to anticholinergic agents.

(Choice A) The abdominal musculature may be used in an accessory fashion to increase intraabdominal pressure and facilitate urination or defecation; however, a solitary defect in the strength of the abdominal musculature would be insufficient to cause urinary retention.

(Choices B and D) Spinal cord injury or stroke can lead to urinary retention due to motor nerve injury (neurogenic bladder) or due to spasm of the internal urethral sphincter. This patient's acute-onset urinary retention following diphenhydramine ingestion makes anticholinergic-induced impairment of detrusor contraction more likely. Shingles (varicella-zoster virus reactivation) typically involves sensory nerves and motor nerve involvement would be unusual.

(Choice E) Extrinsic compression of the urethra commonly occurs in older men due to BPH. Common symptoms include urinary urgency, difficulty with initiating and maintaining urinary stream, and feeling of incomplete bladder emptying. This patient likely has some component of BPH-related obstruction; however, he had not been having difficulty voiding until taking diphenhydramine.

Educational objective:

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Educational objective:
First-generation H1-antihistamines have potent anticholinergic effects and may cause eye and oropharyngeal dryness as well as urinary retention. Due to a high prevalence of benign prostatic hyperplasia, elderly men are at increased risk of urinary retention due to anticholinergic activity.

References

- Adverse effects of medications on urinary symptoms and flow rate: a community-based study.

A 69-year-old man is brought to the emergency department by his family for increasing confusion over the last 2 days. He also has had nausea, vomiting, and back and abdominal pain. At baseline, the patient is interactive and pleasant. His medical history is notable for type 2 diabetes mellitus controlled with metformin and hypertension treated with amlodipine. Temperature is 37.2 C (99 F), blood pressure is 112/70 mm Hg, and pulse is 102/min and regular. The patient appears disheveled and confused. Examination shows clear lung fields and normal heart sounds. There are no neck masses or enlarged lymph nodes. Neurologic examination shows no focal muscle weakness. Laboratory results are as follows:

Leukocytes	3200/mm^3
Hematocrit	32%
Platelets	87,000/mm^3
Sodium	139 mEq/dL
Potassium	4.2 mEq/dL
Chloride	111 mEq/dL
Bicarbonate	26 mEq/dL
Calcium	14.1 mg/dL
Blood urea nitrogen	36 mg/dL
Creatinine	1.8 mg/dL
Glucose	190 mg/dL

Which of the following is the most appropriate next step for this patient?

Chloride	111 mEq/dL
Bicarbonate	26 mEq/dL
Calcium	14.1 mg/dL
Blood urea nitrogen	36 mg/dL
Creatinine	1.8 mg/dL
Glucose	190 mg/dL

Which of the following is the most appropriate next step for this patient?

- A. Bone marrow biopsy
- B. Furosemide
- C. Hemodialysis
- D. Insulin
- E. Methylprednisolone
- F. Normal saline infusion
- G. Pamidronate

Submit

Calcium	14.1 mg/dL
Blood urea nitrogen	36 mg/dL
Creatinine	1.8 mg/dL
Glucose	190 mg/dL

Which of the following is the most appropriate next step for this patient?

- A. Bone marrow biopsy [12%]
- B. Furosemide [5%]
- C. Hemodialysis [11%]
- D. Insulin [3%]
- E. Methylprednisolone [1%]
- ✔ F. Normal saline infusion [59%]
- G. Pamidronate [6%]

Omitted
Correct answer
F

59%
Answered correctly

3 Seconds
Time Spent

12/14/2018
Last Updated

Explanation

Management of hypercalcemia	
Severe (calcium >14 mg/dL) or symptomatic	**Short-term (immediate) treatment** • Normal saline hydration plus calcitonin • Avoid loop diuretics unless volume overload (heart failure) exists **Long-term treatment** • Bisphosphonate (zoledronic acid)
Moderate (calcium 12-14 mg/dL)	• Usually no immediate treatment required unless symptomatic • Treatment is similar to that for severe hypercalcemia
Asymptomatic or mild (calcium <12 mg/dL)	• No immediate treatment required • Avoid thiazide diuretics, lithium, volume depletion & prolonged bed rest

This patient has severe, symptomatic **hypercalcemia**, likely due to an undiagnosed malignancy. Severe hypercalcemia (ie, serum calcium >14 mg/dL) can cause weakness, gastrointestinal distress, and neuropsychiatric symptoms (eg, confusion, stupor, coma), especially with a rapid rise in serum calcium. Patients are typically volume-depleted due to polyuria and decreased oral intake.

Patients with severe hypercalcemia require aggressive **saline hydration** to restore intravascular volume and promote urinary calcium excretion. **Calcitonin**, by inhibiting osteoclast-mediated bone resorption, quickly reduces serum calcium concentrations and can be administered concurrently with saline. **Bisphosphonates** (eg, pamidronate, zoledronic acid) also inhibit bone resorption and provide a sustained reduction in calcium levels. However, the calcium-lowering effect of bisphosphonates is delayed, usually occurring over 2-4 days, and they are typically given after initial administration of saline and calcitonin **(Choice G)**.

(Choice A) Severe hypercalcemia with pancytopenia is a typical presentation of multiple myeloma, which can be confirmed with bone

4 days, and they are typically given after initial administration of saline and calcitonin **(Choice G)**.

(Choice A) Severe hypercalcemia with pancytopenia is a typical presentation of multiple myeloma, which can be confirmed with bone marrow biopsy. However, this patient's acute hypercalcemia should first be corrected with saline hydration.

(Choice B) Routine use of loop diuretics (eg, furosemide) is not recommended in hypercalcemic patients as it can worsen the associated volume depletion.

(Choice C) Hemodialysis is an effective treatment for hypercalcemia, but is typically reserved for patients with renal insufficiency or heart failure in whom aggressive hydration cannot be administered safely. This patient likely has confusion due to hypercalcemia not uremia.

(Choice D) Insulin can be used to drive potassium (and glucose) into cells and is an effective treatment for hyperkalemia. It does not have a significant effect on acute hypercalcemia.

(Choice E) Glucocorticoids (eg, methylprednisolone) inhibit the formation of 1,25-dihydroxyvitamin D by activated mononuclear cells in the lungs and lymph nodes. They can be used to treat hypercalcemia due to excessive vitamin D intake, granulomatous diseases (eg, sarcoidosis), and certain lymphomas. However, their calcium-lowering effects can take 2-5 days to occur.

Educational objective:
Severe hypercalcemia can cause weakness, gastrointestinal distress, and neuropsychiatric symptoms. Patients are typically volume-depleted due to polyuria and decreased oral intake. Initial treatment includes saline hydration to restore intravascular volume and calcitonin to inhibit bone resorption. Bisphosphonates further reduce calcium levels and are given after initial administration of saline.

References

- Treatment of acute hypercalcemia.

A 34-year-old woman comes to the physician's office because of occasional headaches and palpitations. She has no other medical problems. She takes no medications. She smokes one and a half packs of cigarettes daily. Her blood pressure is 170/100 mm Hg in both arms, and heart rate is 80/min. Physical examination shows bilateral flank masses. Laboratory studies show:

Serum sodium	140 mEq/L
Serum potassium	4.4 mEq/L
BUN	26 mg/dL
Serum creatinine	1.3 mg/dL

Urinalysis shows 10-12 red blood cells/hpf, but otherwise shows no abnormalities. The most likely complication that can occur in this patient is which of the following?

- A. Liver necrosis
- B. Intracranial bleeding
- C. Restrictive cardiomyopathy
- D. Pancreatic cancer
- E. Aortic dissection

Submit

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Urinalysis shows 10-12 red blood cells/hpf, but otherwise shows no abnormalities. The most likely complication that can occur in this patient is which of the following?

- A. Liver necrosis [2%]
- ✔ B. Intracranial bleeding [77%]
- C. Restrictive cardiomyopathy [3%]
- D. Pancreatic cancer [2%]
- E. Aortic dissection [13%]

Omitted
Correct answer
B

77%
Answered correctly

3 Seconds
Time Spent

12/16/2018
Last Updated

B

Explanation

This patient most likely has autosomal dominant polycystic kidney disease (ADPKD). The clues to the correct diagnosis are hypertension, palpable bilateral abdominal masses and microhematuria. Intracranial berry aneurysm is a common complication, and is seen in 5 to 10% of the cases. Although such aneurysms are common and dangerous when coupled with hypertension, routine screening for intracranial aneurysms is not recommended.

The other major extra-renal complications of ADPKD are:

1. Hepatic cysts - most common extrarenal manifestations of ADPKD
2. Valvular heart disease - most often mitral valve prolapse and aortic regurgitation
3. Colonic diverticula
4. Abdominal wall and inguinal hernia

(Choice A) Liver necrosis is a rare complication of the disease.

(Choice E) Aortic dissection can occur as a rare complication of severe hypertension, not polycystic kidney disease itself.

(Choices C and D) Restrictive cardiomyopathy and pancreatic cancer are not associated with polycystic kidney disease.

Educational objective:
Autosomal dominant polycystic kidney disease is a potential cause of hypertension. Hepatic cysts are the most common extrarenal manifestations. Intracranial berry aneurysms are seen in 5 to 10 % of the cases. Although such aneurysms are common and dangerous when coupled with hypertension, routine screening for intracranial aneurysms is not recommended.

A 50-year-old man comes to the emergency department due to sudden onset of severe, colicky pain in the right flank. He was admitted twice previously for similar symptoms. On both occasions, the patient was managed conservatively and sent home. He has no other medical problems and no history of urinary infections. He usually eats precooked food from a local store and has a soda with each meal. The patient does not use tobacco, alcohol, or illicit drugs. His vital signs are stable. He is given intravenous fluids and pain medications. Laboratory results are as follows:

Hemoglobin	14.5 g/dL
Leukocytes	13,000/mm^3; no bands
Platelets	300,000/mm^3
Blood urea nitrogen	16 mg/dL
Creatinine	0.8 mg/dL

CT scan of the abdomen without contrast reveals renal calculi. Urinalysis shows moderate levels of blood and calcium oxalate crystals. Which of the following is the best recommendation for the prevention of future stones in this patient?

A. Calcium supplements
B. Decreased dietary calcium intake
C. Decreased potassium intake
D. High dietary animal protein
E. High doses of vitamin C
F. High-fructose diet

Hemoglobin	14.5 g/dL
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- D. High dietary animal protein
- E. High doses of vitamin C
- F. High-fructose diet
- G. Limited sodium intake
- H. Low-fat diet

Submit

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Creatinine	0.8 mg/dL

CT scan of the abdomen without contrast reveals renal calculi. Urinalysis shows moderate levels of blood and calcium oxalate crystals. Which of the following is the best recommendation for the prevention of future stones in this patient?

A. Calcium supplements [11%]
B. Decreased dietary calcium intake [22%]
C. Decreased potassium intake [2%]
D. High dietary animal protein [1%]
E. High doses of vitamin C [16%]
F. High-fructose diet [0%]

Platelets	300,000/mm³
Blood urea nitrogen	16 mg/dL
Creatinine	0.8 mg/dL

CT scan of the abdomen without contrast reveals renal calculi. Urinalysis shows moderate levels of blood and calcium oxalate crystals. Which of the following is the best recommendation for the prevention of future stones in this patient?

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- B. Decreased dietary calcium intake [22%]
- C. Decreased potassium intake [2%]
- D. High dietary animal protein [1%]
- E. High doses of vitamin C [16%]
- F. High-fructose diet [0%]
- ✔ G. Limited sodium intake [33%]
- H. Low-fat diet [12%]

Omitted
Correct answer
G

33%
Answered correctly

9 Seconds
Time Spent

10/28/2018
Last Updated

Explanation

The most common renal stones are **calcium stones**. Increased sodium intake enhances calcium excretion (hypercalciuria), and low sodium intake promotes sodium and calcium reabsorption through its effect on the medullary concentration gradient. Reabsorption of sodium and calcium is coupled via complex mechanisms involving the calcium-sensing receptor in the thick ascending limb of the loop of Henle. Therefore, patients with recurrent renal calculi should be advised to restrict sodium intake. If these patients continue to develop renal stones, their urine sodium levels may be checked to evaluate adherence to a sodium-restricted diet.

(Choices A and F) Excessive calcium intake (>2 g/day) and increased fructose intake may worsen hypercalciuria and calcium oxalate precipitation in urine. Hydrochlorothiazide reduces urinary calcium excretion and may be used in recurrent stone formers with hypercalciuria.

(Choices B and E) Calcium binds oxalate to form inabsorbable calcium oxalate in the gastrointestinal tract. Calcium restriction may increase free oxalate absorption, leading to hyperoxaluria and urinary calcium oxalate stone formation. Increased vitamin C intake also promotes hyperoxaluria. Patients should be advised to restrict oxalate-containing foods (eg, chocolate, tea, peanuts).

(Choice C) A high-potassium diet decreases urinary calcium excretion. Foods rich in potassium enhance urinary citrate excretion (likely from urinary alkalization), forming soluble calcium citrate and thereby preventing stone formation.

(Choice D) Diets rich in animal protein are associated with an increased risk of nephrolithiasis in men. Increased acid resulting from protein metabolism is excreted in urine, leading to calcium salt precipitation.

(Choice H) Patients with malabsorption syndromes (eg, Crohn disease, cystic fibrosis) have increased amounts of intestinal fatty acids that form salts with calcium. Less calcium is available to bind oxalate, resulting in hyperoxaluria. This patient has no evidence of malabsorption.

Educational objective:
The dietary recommendations for patients with renal calculi are:

oxalate precipitation in urine. Hydrochlorothiazide reduces urinary calcium excretion and may be used in recurrent stone formers with hypercalciuria.

(Choices B and E) Calcium binds oxalate to form inabsorbable calcium oxalate in the gastrointestinal tract. Calcium restriction may increase free oxalate absorption, leading to hyperoxaluria and urinary calcium oxalate stone formation. Increased vitamin C intake also promotes hyperoxaluria. Patients should be advised to restrict oxalate-containing foods (eg, chocolate, tea, peanuts).

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(Choice H) Patients with malabsorption syndromes (eg, Crohn disease, cystic fibrosis) have increased amounts of intestinal fatty acids that form salts with calcium. Less calcium is available to bind oxalate, resulting in hyperoxaluria. This patient has no evidence of malabsorption.

Educational objective:
The dietary recommendations for patients with renal calculi are:

1. Increased fluid intake
2. Decreased sodium intake
3. Normal dietary calcium intake

References

- Diet and fluid prescription in stone disease.

A 24-year-old man comes to the urgent care clinic due to blood in his urine. He has a 1-day history of mild midline back discomfort, associated with a single episode of red urine. The hematuria subsequently resolved and his urine is now a normal yellow color. The patient also has had a sore throat for the past 4 days, for which he took 3 tablets of over-the-counter acetaminophen. He has had no fever, abdominal pain, or burning during urination. He has smoked 1 pack of cigarettes daily for 5 years. Physical examination is unremarkable. Urinalysis shows:

Color	Yellow
Glucose	Negative
Protein	3+
Ketones	Negative
Leukocyte esterase	Negative
Nitrites	Negative
White blood cells	2-3/hpf
Red blood cells	50-75/hpf

Which of the following is the most likely source of this patient's urinary complaints?

A. Glomerulus
B. Prostate gland
C. Renal interstitium
D. Renal papilla

Glucose	Negative
Protein	3+
Ketones	Negative
Leukocyte esterase	Negative
Nitrites	Negative
White blood cells	2-3/hpf
Red blood cells	50-75/hpf

Which of the following is the most likely source of this patient's urinary complaints?

- A. Glomerulus
- B. Prostate gland
- C. Renal interstitium
- D. Renal papilla
- E. Renal tubule
- F. Urinary bladder epithelium

Submit

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Leukocyte esterase	Negative
Nitrites	Negative
White blood cells	2-3/hpf
Red blood cells	50-75/hpf

Which of the following is the most likely source of this patient's urinary complaints?

- A. Glomerulus [67%]
- B. Prostate gland [0%]
- C. Renal interstitium [9%]
- D. Renal papilla [12%]

Protein	3+
Ketones	Negative
Leukocyte esterase	Negative
Nitrites	Negative
White blood cells	2-3/hpf
Red blood cells	50-75/hpf

Which of the following is the most likely source of this patient's urinary complaints?

- ✔ A. Glomerulus [67%]
- B. Prostate gland [0%]
- C. Renal interstitium [9%]
- D. Renal papilla [12%]
- E. Renal tubule [6%]
- F. Urinary bladder epithelium [3%]

Omitted
Correct answer
A

67%
Answered correctly

4 Seconds
Time Spent

12/03/2018
Last Updated

Explanation

	Glomerular hematuria	Nonglomerular hematuria
Type of hematuria	• Microscopic > gross	• Gross > microscopic

Common etiologies	• Glomerulonephritis
	  ◦ Poststreptococcal
	  ◦ IgA nephropathy
	• Basement membrane disorder
	  ◦ Alport syndrome
		• Nephrolithiasis
		• Cancer (eg, renal, prostate)
		• Polycystic kidney disease
		• Infection (eg, cystitis)
		• Papillary necrosis

Clinical presentation	• Nonspecific or no symptoms • Nephritic syndrome	• Dysuria • Urinary obstruction
Urinalysis	• Blood & protein • RBC casts, dysmorphic RBCs	• Blood but no protein • Normal-appearing RBCs

RBC = red blood cell.

Hematuria can be due to glomerular or non-glomerular causes. **Non-glomerular hematuria** is more common; it is characterized by gross hematuria with normal-appearing red blood cells (RBCs) but without significant proteinuria. Etiologies include cancer (eg, renal cell, prostate), infections (eg, cystitis), polycystic kidney disease, and nephrolithiasis. Patients frequently have specific urinary symptoms (eg, dysuria, flank pain, renal colic) that help to clarify the diagnosis.

In contrast, **glomerular hematuria** usually causes microscopic hematuria, though gross hematuria may be present in some cases. It is usually due to glomerulonephritis (GN) or basement membrane disease. Symptoms are usually absent or nonspecific (eg, low back pain), and patients may present after developing features of nephritic syndrome (eg, hematuria, hypertension, oliguria, elevated

symptoms (eg, dysuria, flank pain, renal colic) that help to clarify the diagnosis.

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(Choice B) Prostate carcinoma, benign prostatic hypertrophy (BPH), or prostatitis can cause non-glomerular hematuria. Prostate carcinoma and BPH usually present in men age >55, often with symptoms of urinary tract obstruction. However, prostatitis is unlikely as this patient had no evidence of urinary tract infection (negative leukocyte esterase, nitrite, and white blood cells).

(Choice C) Renal tumors or malignancies (eg, renal cell cancer) involving the interstitium can present as non-glomerular hematuria. However, proteinuria would not be typically seen with this condition.

(Choice D) Papillary necrosis with sloughing of the renal papilla is a rare cause of non-glomerular hematuria. It may be seen with long-term acetaminophen abuse but not with light-intermittent use (mnemonic **NSAID**: **N**onsteroidal antiinflammatory drugs, **S**ickle cell disease, **A**nalgesic abuse, **I**nfection (pyelonephritis), and **D**iabetes mellitus).

(Choice E) Acute tubular necrosis is characterized by acute azotemia following a hypotensive or nephrotoxic injury. Patients may have dark or cola-colored urine, but significant hematuria is uncommon. Tubulointerstitial nephritis causes microscopic hematuria associated with elevated creatinine and variable proteinuria. It is most commonly due to certain medications including antibiotics, NSAIDs, and diuretics.

(Choice F) Urinary bladder tumor, carcinoma, or cystitis can present as non-glomerular hematuria. While smoking is a risk factor for bladder carcinoma, this malignancy is rarely seen in younger adults (age <55). And cystitis is unlikely as this patient had no evidence of urinary tract infection.

Educational objective:

carcinoma and BPH usually present in men age >55, often with symptoms of urinary tract obstruction. However, prostatitis is unlikely as this patient had no evidence of urinary tract infection (negative leukocyte esterase, nitrite, and white blood cells).

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Educational objective:
Hematuria (gross or microscopic) can be due to glomerular or non-glomerular causes. The source of hematuria is more likely to be glomerular if the urine studies reveal proteinuria, dysmorphic red blood cells (RBCs) or RBC casts.

References

- Assessment of microscopic hematuria in adults.

A 36-year-old man recently diagnosed with Hodgkin lymphoma comes to the physician due to fatigue and generalized edema.

Laboratory results are shown below:

Sodium	145 mEq/L
Potassium	3.8 mEq/L
Creatinine	0.9 mg/dL
Albumin	2.0 g/dL
Total bilirubin	0.9 mg/dL
Globulin	4.6 g/dL

Urinalysis shows 4+ proteinuria with no hematuria. Vital signs are within normal limits. Which of the following glomerular diseases is most likely to be present in this patient?

A. Amyloidosis
B. Crescentic glomerulonephritis
C. Focal segmental glomerulosclerosis
D. Membranoproliferative glomerulonephritis
E. Minimal change disease

Submit

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Globulin	4.6 g/dL

Urinalysis shows 4+ proteinuria with no hematuria. Vital signs are within normal limits. Which of the following glomerular diseases is most likely to be present in this patient?

A. Amyloidosis [25%]
B. Crescentic glomerulonephritis [4%]
C. Focal segmental glomerulosclerosis [20%]
D. Membranoproliferative glomerulonephritis [21%]
✔ E. Minimal change disease [27%]

Omitted
Correct answer
E

27%
Answered correctly

4 Seconds
Time Spent

07/12/2018
Last Updated

Explanation

Primary renal causes of nephrotic syndrome	
Etiology	**Clinical associations**
Focal segmental glomerulosclerosis	African American & Hispanic ethnicity; obesity; HIV & heroin use
Membranous nephropathy	Adenocarcinoma (eg, breast, lung); nonsteroidal antiinflammatory drugs (NSAIDs); hepatitis B; systemic lupus erythematosus
Membranoproliferative glomerulonephritis	Hepatitis B & C; lipodystrophy
Minimal change disease	NSAIDs; lymphoma
IgA nephropathy	Upper respiratory tract infection

A variety of glomerular diseases may be seen in the setting of malignancy. This patient has **nephrotic syndrome** (indicated by the high degree of proteinuria, hypoalbuminemia, and edema) following a recent diagnosis of Hodgkin lymphoma. **Minimal change disease** (MCD) is the most common form of nephrotic syndrome in patients with **Hodgkin lymphoma**. Nephrotic syndrome will usually resolve with successful treatment of the lymphoma.

Overall, **membranous glomerulopathy** is the most common form of nephrotic syndrome associated with **malignancies**. However, these are **usually solid** cancers (lung, colon, prostate, breast), unlike in the case of the association between MCD and Hodgkin lymphoma. Awareness of these disease associations may help guide the screening and detection of underlying conditions in patients with nephrotic syndrome.

(Choice A) Nephrotic syndrome due to amyloidosis is usually seen in multiple myeloma (deposition of immunoglobulin light chains), not Hodgkin lymphoma. Weight gain and lower extremity edema can occur at a slow pace and may initially go unnoticed by the patient or physician.

(Choice B) This patient is unlikely to have crescentic glomerulonephritis as he does not have acute kidney injury (present in almost all cases), hematuria, or hypertension. In addition, crescentic glomerulonephritis is usually associated with autoimmune disorders and not with malignancies.

(Choice C) Focal segmental glomerulosclerosis is associated with HIV, heroin, obesity, and some medications; it is not classically associated with malignancies.

(Choice D) Membranoproliferative glomerulonephritis is associated with hepatitis B and C infections, chronic bacterial infections (eg, endocarditis), and some autoimmune conditions. It is uncommonly associated with leukemias (chronic lymphocytic leukemia) or lymphomas (non-Hodgkin lymphoma, B-cell lymphoma) and is not typically seen in Hodgkin lymphoma.

Educational objective:
Membranous nephropathy is the most common form of nephrotic syndrome associated with carcinoma. However, minimal change disease is usually seen in patients with Hodgkin lymphoma.

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Educational objective:
Membranous nephropathy is the most common form of nephrotic syndrome associated with carcinoma. However, minimal change disease is usually seen in patients with Hodgkin lymphoma.

References

- Minimal change nephrotic syndrome and classical Hodgkin's lymphoma: report of 21 cases and review of the literature.
- Glomerular lesions in lymphomas and leukemias.

A 50-year-old man comes to the physician for a routine follow-up visit. He has hypertension, diabetes mellitus, secondary hyperparathyroidism, and end-stage renal disease. He has been on hemodialysis for the past three years. He was admitted three months ago for line sepsis, which was treated with antibiotics. He had a right below-the-knee amputation two years ago following a non-healing foot ulcer. Physical examination shows a right carotid bruit. If this patient dies within the next five years, what would be the most likely cause of his death?

- A. Cardiovascular disease
- B. Stroke
- C. Infection
- D. Cancer
- E. Withdrawal from dialysis

Submit

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- ✔ A. Cardiovascular disease [56%]
- B. Stroke [12%]
- C. Infection [18%]
- D. Cancer [0%]
- E. Withdrawal from dialysis [12%]

Omitted
Correct answer
A

56%
Answered correctly

3 Seconds
Time Spent

10/10/2018
Last Updated

Explanation

Cardiovascular disease is the most common cause of death in the general population, but the rates are declining. Interestingly, this recent trend has not been observed in the dialysis population. Cardiovascular disease remains as the most common cause of death in dialysis patients. It accounts for approximately 50% of deaths in this group of patients. Of these deaths, 20% are attributed to

Explanation

Cardiovascular disease is the most common cause of death in the general population, but the rates are declining. Interestingly, this recent trend has not been observed in the dialysis population. Cardiovascular disease remains as the most common cause of death in dialysis patients. It accounts for approximately 50% of deaths in this group of patients. Of these deaths, 20% are attributed to acute myocardial infarction and approximately 60% to sudden cardiac deaths.

The following risk factors are associated with cardiovascular disease in dialysis patients:

- Risk factors not related to dialysis: A large number of patients on dialysis already have multiple risk factors for cardiovascular disease. These are:
  - Hypertension (96%)
  - Diabetes (54%)
  - Low serum HDL cholesterol (33%)
  - Left ventricular hypertrophy by ECG criteria (22%)
  - Coronary artery disease: Approximately 75% of patients with total end-stage renal disease have at least a 50% narrowing of at least one coronary artery.
  - Increased age: The average age of patients at the start of dialysis is about 60 years.
- Additional risk factors due to end stage renal disease and dialysis are:
  - End stage renal disease: This, by itself, is an independent risk factor for cardiovascular disease.
  - Anemia.
  - Metabolic abnormality, particularly hyperphosphatemia, and increased PTH levels.
  - Increased homocysteine levels: These are due to impaired metabolism and decreased removal.
  - Accelerated atherogenesis in dialysis patients: This is due to enhanced oxidant stress due to uremia and bio-incompatible renal replacement therapies.
  - Increased calcium intake (calcium is given to correct hyperphosphatemia in dialysis patients): This enhances coronary artery calcification.

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  - Increased homocysteine levels: These are due to impaired metabolism and decreased removal.
  - Accelerated atherogenesis in dialysis patients: This is due to enhanced oxidant stress due to uremia and bio-incompatible renal replacement therapies.
  - Increased calcium intake (calcium is given to correct hyperphosphatemia in dialysis patients): This enhances coronary artery calcification.
  - Inhibition of NO: This is a common finding in dialysis patients, and can cause vasoconstriction and hypertension.

(Choice C) Infection is also a common cause of death in dialysis patients. It accounts for approximately 15-20% of deaths, and is most commonly related to vascular access (line sepsis).

(Choice E) Withdrawal from dialysis accounts for 20% of deaths in dialysis patients.

(Choice D) Patients on dialysis are not at increased risk for any cancer.

(Choice B) Stroke can occur secondary to cardiovascular disease; thus, cardiovascular disease remains as the most common cause of death in dialysis patients.

Educational Objective:
Cardiovascular disease is the most common cause of death in dialysis patients. It accounts for approximately 50% of all deaths in this population. Cardiovascular disease is also the most common cause of death in renal transplant patients.

A 34-year-old male is recovering from head trauma sustained in a motor vehicle accident. He is currently in the intensive care unit on mechanical ventilation. His most recent arterial blood gas analysis shows:

pH	7.54
PaO_2	124 mmHg
$PaCO_2$	20 mmHg
Bicarbonate	17 mEq/L

Which of the following additional findings do you most expect in this patient?

A. Low urine bicarbonate excretion
B. High urine pH
C. High serum aldosterone level
D. High serum anion gap
E. High serum albumin level

Submit

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pH	7.54
PaO_2	124 mmHg
$PaCO_2$	20 mmHg
Bicarbonate	17 mEq/L

Which of the following additional findings do you most expect in this patient?

- A. Low urine bicarbonate excretion [14%]
- ✔ B. High urine pH [65%]
- C. High serum aldosterone level [8%]
- D. High serum anion gap [10%]
- E. High serum albumin level [2%]

Omitted
Correct answer
B

65%
Answered correctly

3 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

While being mechanically ventilated this patient is vulnerable to forced hyper- or hypoventilation. Ventilation is defined as the tidal volume multiplied by the respiratory rate. Hyperventilation (caused by increased tidal volume and/or respiratory rate) results in excessive CO_2 losses and respiratory alkalosis, while hypoventilation (caused by decreased tidal volume and/or respiratory rate) results in CO_2 retention and respiratory acidosis. The patient described is experiencing respiratory alkalosis as evidenced by his increased arterial blood pH (normal = 7.4) and his decreased $PaCO_2$ (normal = 40 mmHg). His serum bicarbonate level is decreased due to attempted renal compensation for the respiratory alkalosis. Specifically, the kidneys retain increased amounts of H^+ (protons) and excrete increased amounts of bicarbonate (HCO_3-) in an attempt to normalize the serum pH. The increased amount of HCO_3- in the urine alkalinizes the urine.

(Choice A) Renal compensation for respiratory or metabolic acidosis involves low urine bicarbonate excretion.

(Choice C) Increased serum aldosterone would result in increased H^+ and K^+ losses in the urine and serum alkalosis. "Contraction alkalosis" occurs in states of intracellular volume contraction, where increased levels of aldosterone function to restore intravascular volume but also cause increased urinary proton loss.

(Choice D) A high serum anion gap occurs in metabolic acidoses resulting from diabetic ketoacidosis, renal failure, lactic acidosis, and methanol, ethylene glycol or salicylate poisoning.

(Choice E) A high serum albumin level may indicate hemoconcentration, as occurs in dehydration.

Educational objective:
The kidney compensates for respiratory alkalosis by preferentially excreting bicarbonate in the urine. The result is an alkalinized urine

A newborn boy is evaluated shortly after vaginal delivery for respiratory distress. He was born at 37 weeks gestation to a primigravid mother who had no medical problems. Second-trimester prenatal ultrasound showed bilateral hydronephrosis and oligohydramnios. His temperature is 36.7 C (98 F), pulse is 144/min, and respirations are 50/min. Pulse oximetry is 84% on room air. Examination shows decreased aeration of both lungs and intercostal retractions. The child has flattened facies, abdominal distension, and clubfeet. What is the most likely underlying diagnosis in this patient?

- A. Bilateral cryptorchidism
- B. Congenital diaphragmatic hernia
- C. Duodenal atresia
- D. Hypospadias
- E. Posterior urethral valves
- F. Prostatic hyperplasia
- G. Wilms tumor

Submit

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- A. Bilateral cryptorchidism [1%]
- B. Congenital diaphragmatic hernia [8%]
- C. Duodenal atresia [7%]
- D. Hypospadias [2%]
- ✔ E. Posterior urethral valves [70%]
- F. Prostatic hyperplasia [1%]
- G. Wilms tumor [8%]

Omitted
Correct answer
E

70%
Answered correctly

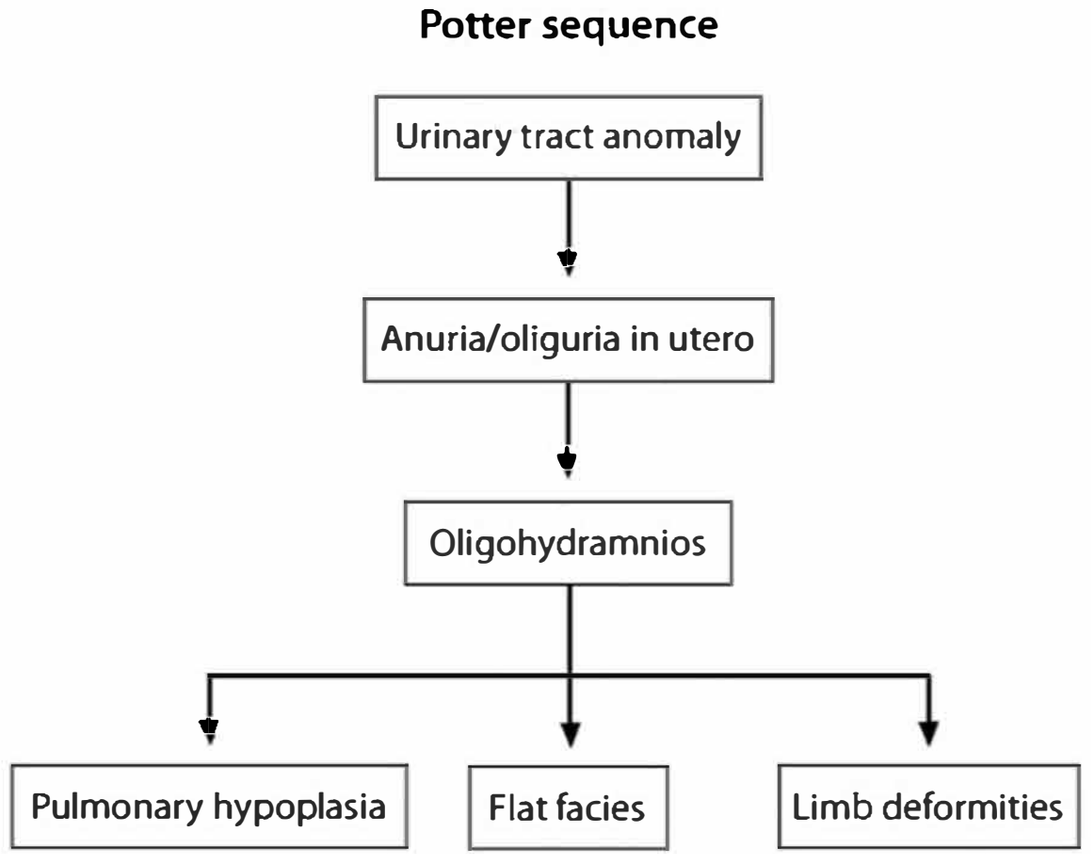
2 Seconds
Time Spent

09/25/2018
Last Updated

Explanation

Explanation

Exhibit Display



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Posterior urethral valves (PUV) are the **most common cause of urinary tract obstruction** in newborn boys. Abnormal folds in the distal prostatic urethra obstruct urinary flow, resulting in **progressive dilation of the bladder, ureters and kidneys**. Prenatal ultrasonography findings of **bladder distention, bilateral hydroureters**, and **bilateral hydronephrosis** are highly suggestive of PUV. Poor urine output in utero results in oligohydramnios as fetal urine is a major source of amniotic fluid. **Oligohydramnios** in the second trimester is ominous and associated with high perinatal mortality because normal amniotic fluid levels are required for lung development. Low amniotic fluid also restricts fetal movement, leading to a cascade of physical anomalies including flat facies and limb deformities **(Potter sequence)**.

Other affected infants can present with poor urinary stream, straining with voiding, urosepsis, failure to thrive, and **renal failure**. Diagnosis is confirmed by voiding cystourethrogram and cystoscopy. Treatment options include PUV ablation and urinary diversion. Despite prenatal diagnosis and early surgical intervention, patients are at high risk for permanent kidney damage.

(Choice A) Cryptorchidism is the failure of one or both testes to descend from the abdomen through the inguinal canal(s) into the scrotum. Examination shows a hypoplastic or poorly rugated scrotum. However, cryptorchidism would not cause obstructive uropathy and oligohydramnios.

(Choice B) Congenital diaphragmatic hernia is a life-threatening defect of the diaphragm that allows abdominal viscera to herniate into the chest, resulting in restriction of lung development. Patients can present with respiratory distress, but on examination the abdomen appears scaphoid due to loss of the abdominal contents into the chest. Prenatal ultrasound demonstrates a mass in the chest (abdominal contents) and sometimes polyhydramnios from esophageal compression, making this diagnosis unlikely.

(Choice C) Fetal swallowing is responsible for amniotic fluid removal. An atretic duodenum can obstruct amniotic fluid clearance, resulting in polyhydramnios, in contrast to the oligohydramnios seen in this patient. Another significant finding on prenatal ultrasound includes the characteristic "double bubble" sign due to dilation of the stomach and the first part of the duodenum.

(Choice D) Hypospadias is a congenital abnormality in which the penile urethra opens on the ventral surface of the penis rather than the tip. These patients are able to urinate but may have difficulty controlling the urinary stream.

(Choice E) Benign prostatic hyperplasia can cause obstructive uropathy at the same level as posterior urethral valves. However, this

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(Choice F) Benign prostatic hyperplasia can cause obstructive uropathy at the same level as posterior urethral valves. However, this condition is common in men age >50 and does not affect children.

(Choice G) Wilms tumor (nephroblastoma) is the most common renal malignancy in childhood. Most children present at age 2-5 years with a large, palpable flank mass but no other symptoms. It is extremely rare for Wilms tumor to present in utero.

Educational objective:
Posterior urethral valves are the most common cause of urinary tract obstruction in newborn boys. Classic findings on prenatal ultrasonography include bladder distension, bilateral hydroureters, and bilateral hydronephrosis. Oligohydramnios from low urine production in utero can cause pulmonary hypoplasia and postnatal respiratory distress.

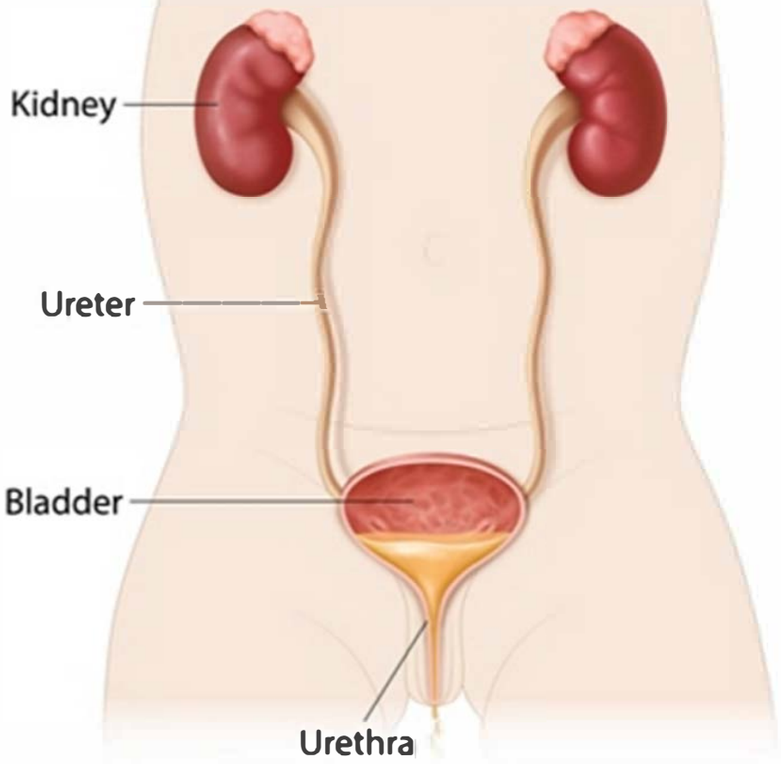
References

- Congenital urethral anomalies in boys. Part I: posterior urethral valves.
- Risk factors for end stage renal disease in children with posterior urethral valves.

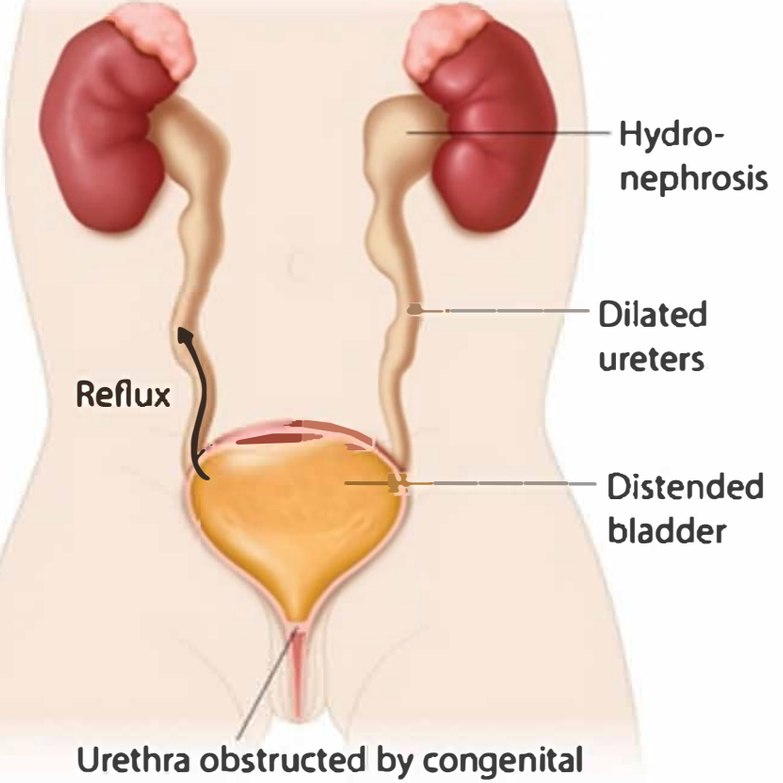
Exhibit Display

Posterior urethral valves

Normal urinary system



Posterior urethral valves



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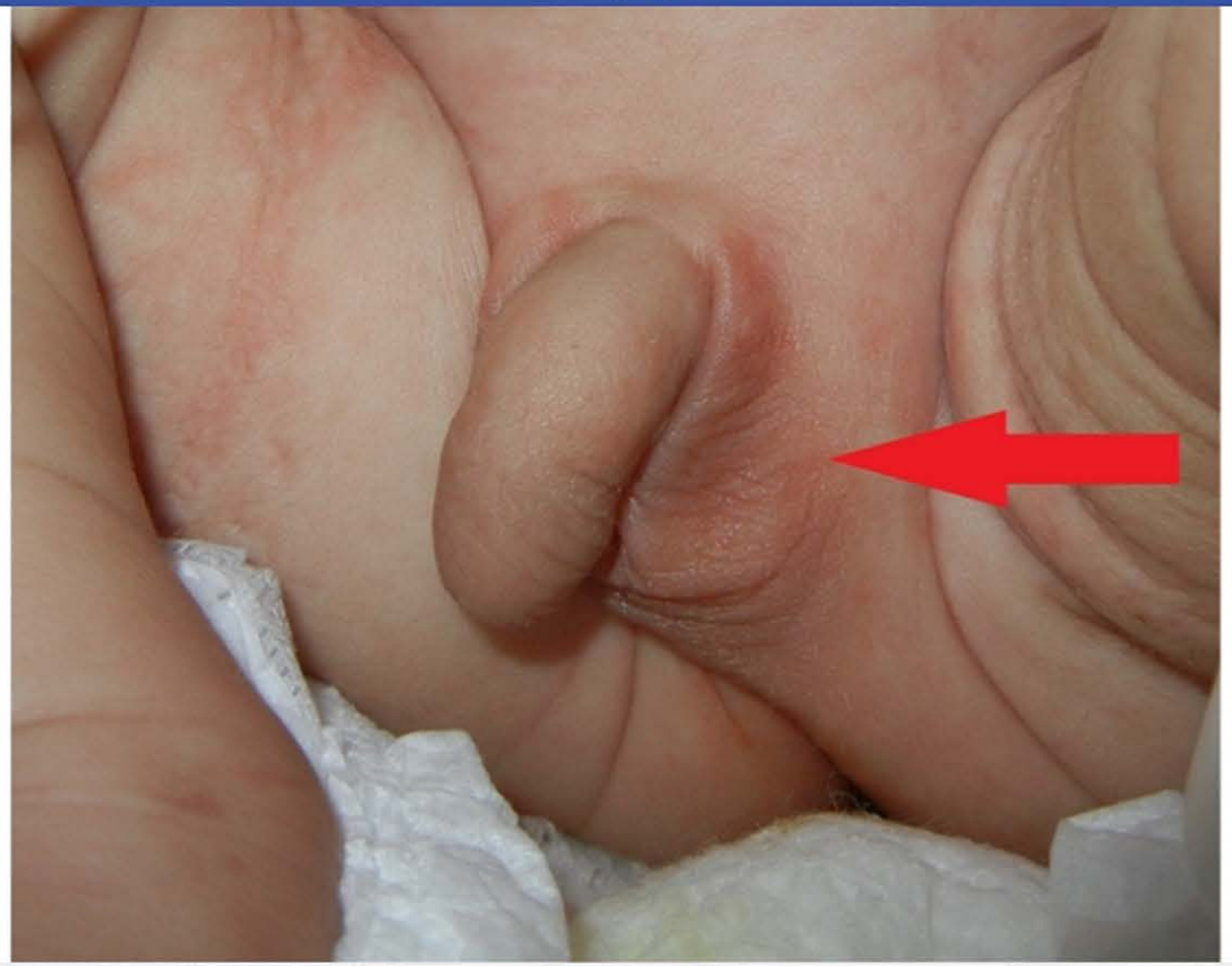

Zoom In | Zoom Out | Reset | Add To Flash Card

A 28-year-old man is brought to the hospital after a high-speed, single motor vehicle collision. The patient has pain in the right pelvis and lower abdomen. Blood pressure is 120/80 mm Hg and pulse is 104/min. Cardiopulmonary examination is normal. Abdominal palpation shows fullness and mild tenderness in the suprapubic region without rigidity or rebound tenderness. No blood is noted at the urethral meatus and digital rectal examination is normal. Pelvic x-rays reveal a right pubic ramus fracture. A Foley catheter is placed without resistance and there is immediate return of frank blood. A pelvic binder is applied, and the patient is taken for an emergency CT scan of the abdomen and pelvis. Which of the following injuries is most likely to be seen on CT scan in this patient?

- A. Extraperitoneal bladder injury
- B. Intraperitoneal bladder rupture
- C. Renal laceration
- D. Right ureteral injury
- E. Urethral injury

Submit

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- ✔ A. Extraperitoneal bladder injury [50%]
- B. Intraperitoneal bladder rupture [23%]
- C. Renal laceration [3%]
- D. Right ureteral injury [10%]
- E. Urethral injury [12%]

Omitted
Correct answer
A

50%
Answered correctly

3 Seconds
Time Spent

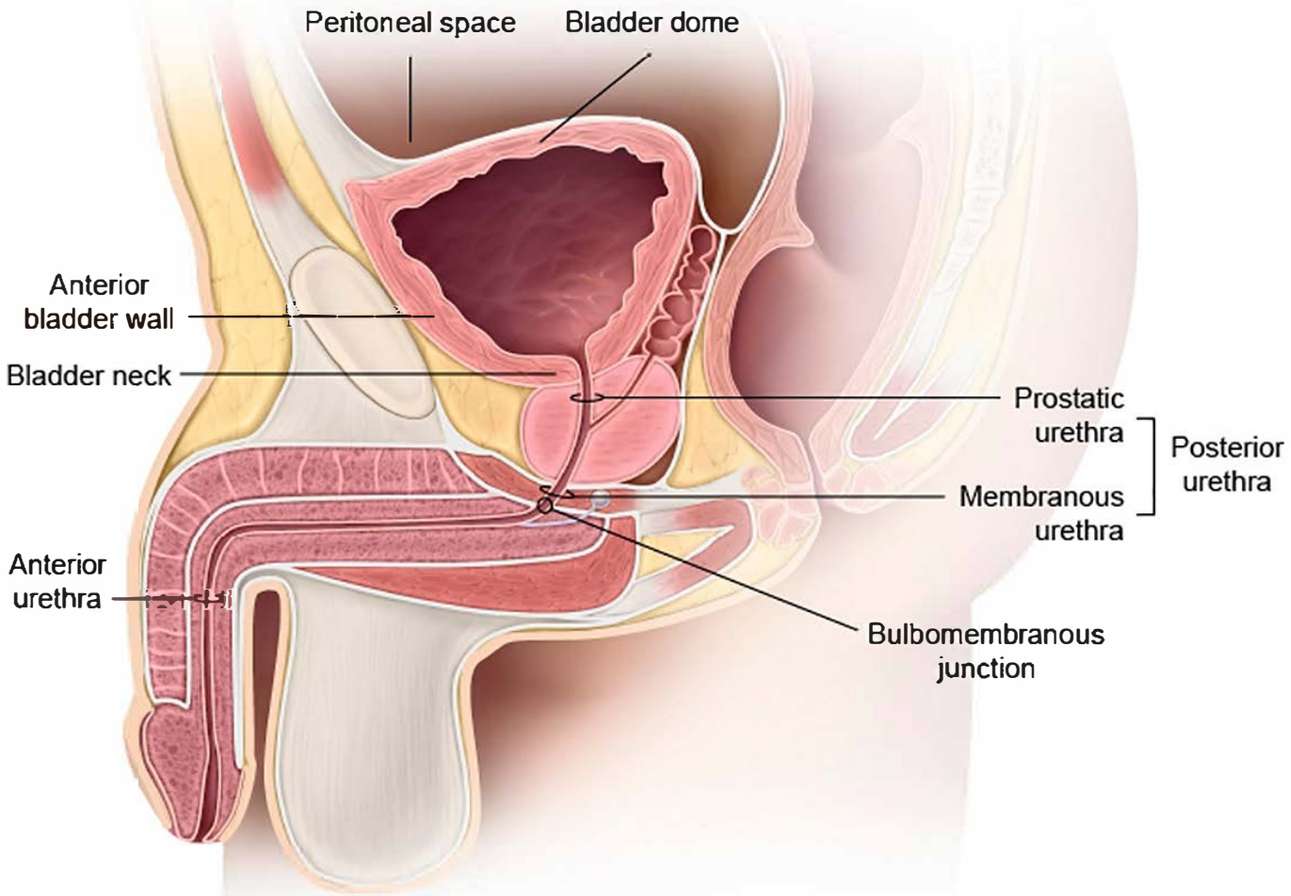
09/23/2018
Last Updated

Explanation

Male urogenital anatomy

Explanation

Male urogenital anatomy



This patient likely has an **extraperitoneal bladder injury (EPBI)**, which may consist of either **contusion** or **rupture** of the neck, anterior wall, or anterolateral wall of the bladder. In the case of rupture, extravasation of urine into adjacent tissues causes **localized pain** in the lower abdomen and pelvis. **Pelvic fracture** is almost always present in EPBI, and sometimes a bony fragment can directly puncture and rupture the bladder. **Gross hematuria** is also usually present, and **urinary retention** (evidenced by suprapubic fullness in this patient) may occur, especially in the case of injury to the bladder neck.

(Choice B) Intraperitoneal bladder rupture describes rupture of the dome of the bladder; the dome is composed of the superior and lateral bladder walls and directly abuts the peritoneal space. Rupture of this area results in intraperitoneal urine leakage and typically presents with signs of chemical peritonitis (eg, diffuse abdominal tenderness, guarding, rebound), which are absent in this patient. Pelvic fracture is often present but less commonly than in EPBI.

(Choice C) Renal laceration may present with hematuria, but urinary retention would be unusual. Patients with this injury usually have flank pain and hemorrhage into the retroperitoneal space.

(Choice D) The most common cause of ureteral injury is iatrogenic trauma during abdominal surgery. Injury due to blunt trauma is relatively rare. When it does occur, the most common site is the ureteropelvic junction. In such cases, hematuria may be present, and fever, flank pain, and a renal mass (from hydronephrosis) may develop several hours after injury.

(Choice E) Urethral injury is more common in men and occurs in approximately 25% of male pelvic fractures. The ability to pass a Foley catheter into the bladder makes urethral injury unlikely. In addition, this patient lacks physical examination findings suggestive of urethral injury (eg, blood at the urethral meatus, high-riding prostate).

Educational objective:
Extraperitoneal bladder injury can occur as contusion or rupture of the neck, anterior wall, or anterolateral wall of the bladder. Localized pain, gross hematuria, and associated pelvic fracture are typically present. Signs of peritonitis (eg, diffuse abdominal tenderness, guarding, rebound) should not be present.

References

A 65-year-old man comes to the emergency department due to fever, chills, and a productive cough for the past 3 days. He also has shortness of breath and right-sided chest pain that is worse with deep breathing. His other medical problems include coronary artery disease, type 2 diabetes mellitus, and hyperlipidemia. The patient's medications include low-dose aspirin, atorvastatin, metformin, and sitagliptin. He does not use tobacco, alcohol, or illicit drugs. His temperature is 39.2 C (102.5 F), blood pressure is 95/60 mm Hg, pulse is 112/min, and respirations are 24/min. Oxygen saturation is 96% on room air. Examination shows dry mucous membranes. Laboratory results are as follows:

Hemoglobin	17.0 g/dL
Platelets	250,000/μL
Leukocytes	16,500/μL
Sodium	135 mEq/L
Potassium	4.9 mEq/L
Blood urea nitrogen	48 mg/dL
Creatinine	2.0 mg/dL
Calcium	10.3 mg/dL
Blood glucose	128 mg/dL

Chest x-ray reveals a right lower-lobe infiltrate. Blood cultures are obtained and intravenous fluids and antibiotics are administered. Which of the following is the most appropriate next step in management of this patient?

A. Discontinue aspirin

Sodium	135 mEq/L
Potassium	4.9 mEq/L
Blood urea nitrogen	48 mg/dL
Creatinine	2.0 mg/dL
Calcium	10.3 mg/dL
Blood glucose	128 mg/dL

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Which of the following is the most appropriate next step in management of this patient?

- A. Discontinue aspirin
- B. Discontinue atorvastatin
- C. Discontinue metformin
- D. Start intravenous insulin infusion
- E. Start lisinopril
- F. Start zoledronic acid

Submit

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Blood glucose	128 mg/dL

Chest x-ray reveals a right lower-lobe infiltrate. Blood cultures are obtained and intravenous fluids and antibiotics are administered. Which of the following is the most appropriate next step in management of this patient?

A. Discontinue aspirin [8%]

Potassium	4.9 mEq/L
Blood urea nitrogen	48 mg/dL
Creatinine	2.0 mg/dL
Calcium	10.3 mg/dL
Blood glucose	128 mg/dL

Chest x-ray reveals a right lower-lobe infiltrate. Blood cultures are obtained and intravenous fluids and antibiotics are administered. Which of the following is the most appropriate next step in management of this patient?

- A. Discontinue aspirin [8%]
- B. Discontinue atorvastatin [3%]
- ✔ C. Discontinue metformin [59%]
- D. Start intravenous insulin infusion [9%]
- E. Start lisinopril [11%]
- F. Start zoledronic acid [6%]

Omitted
Correct answer
C

59%
Answered correctly

5 Seconds
Time Spent

11/07/2018
Last Updated

Explanation

This patient's clinical presentation – fever, cough, hypotension, tachycardia, and lung infiltrate on imaging – suggests sepsis due to community-acquired pneumonia. The patient also has dry mucous membranes and laboratory findings (high hemoglobin, blood urea nitrogen [BUN], and creatinine levels) suggestive of volume depletion. A BUN/creatinine ratio >20 is consistent with a prerenal cause of azotemia.

Hypotension and volume depletion due to sepsis are typically treated with intravenous fluids (isotonic saline), which generally results in improvement of prerenal azotemia. However, this patient's clinical condition may temporarily worsen before improving if he develops acute tubular necrosis or septic shock. Therefore, any nephrotoxins (eg, nonsteroidal anti-inflammatory drugs) that can further worsen kidney function must be avoided. Metformin can cause lactic acidosis in acute kidney injury and sepsis and should be withheld until renal function improves.

(Choice A) Low-dose aspirin use does not affect renal blood flow and has not been shown to precipitate or worsen renal failure in patients with otherwise normally functioning kidneys. Therefore, in this patient with coronary artery disease, it does not need to be stopped at this time.

(Choice B) Statins are not known to cause acute kidney injury. Instead, myopathy is the most frequent side effect. There is no indication for stopping atorvastatin in this patient.

(Choice D) Target blood glucose level for acutely ill patients with hyperglycemia is 140-180 mg/dL. Short-acting insulin regimens are typically recommended. This patient's blood sugar is currently controlled; intravenous insulin therapy is unnecessary and would put him at risk for hypoglycemia and adverse clinical outcomes.

(Choice E) Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers can temporarily worsen renal function and hypotension, and should not be started in the setting of acute kidney injury.

(Choice F) Hypercalcemia in this patient is most likely due to volume depletion and hemoconcentration. It should correct with

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(Choice E) Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers can temporarily worsen renal function and hypotension, and should not be started in the setting of acute kidney injury.

(Choice F) Hypercalcemia in this patient is most likely due to volume depletion and hemoconcentration. It should correct with hydration and does not require bisphosphonate therapy.

Educational objective:
Metformin should not be given to acutely ill patients with acute renal failure, liver failure, or sepsis as these conditions increase the risk of lactic acidosis.

References

- Inpatient treatment of type 2 diabetes.

A 31-year-old woman comes to the office with a 6-week history of periorbital edema and abdominal distention. Her temperature is 37.1 C (98.9 F), blood pressure is 125/75 mm Hg, pulse is 80/min, and respirations are 14/min. Examination shows moderate ascites and lower extremity edema. Urinalysis shows proteinuria and 24-hour urinary protein excretion is 4 g/day, total serum protein is 5 g/dL, and serum albumin is 2.5 g/dL. Renal ultrasound is unremarkable. A renal biopsy is performed. The patient is started on diuretics and salt and protein intake is restricted. The edema begins to improve. However, the patient suddenly develops severe right-sided abdominal pain, fever, and gross hematuria. Which of the following is the most likely diagnosis revealed by renal biopsy?

- A. Focal segmental glomerulosclerosis
- B. IgA nephropathy
- C. Membranous glomerulopathy
- D. Minimal change disease
- E. Systemic amyloidosis

Submit

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- A. Focal segmental glomerulosclerosis [23%]
- B. IgA nephropathy [12%]
- ✔ C. Membranous glomerulopathy [49%]
- D. Minimal change disease [4%]
- E. Systemic amyloidosis [9%]

Omitted
Correct answer
C

49%
Answered correctly

4 Seconds
Time Spent

10/04/2018
Last Updated

Explanation

Renal vein thrombosis (RVT) and other thromboembolism are important complications of nephrotic syndrome. Loss of antithrombin III (an inhibitor of multiple coagulation factors) in the urine increases the risk of venous and arterial thrombosis. Thrombosis of the

Correct answer
C

Answered correctly

Time Spent

Last Updated

Explanation

Renal vein thrombosis (RVT) and other thromboembolism are important complications of nephrotic syndrome. Loss of antithrombin III (an inhibitor of multiple coagulation factors) in the urine increases the risk of venous and arterial thrombosis. Thrombosis of the renal vein can be acute and present with abdominal pain, fever, and hematuria, as in this patient. However, RVT is more commonly progressive, causing gradual worsening of renal function and proteinuria in an asymptomatic patient. RVT can occur in any etiology of nephrotic syndrome, but it is most commonly seen with membranous glomerulopathy.

(Choices A, D, and E) Focal segmental glomerulosclerosis, minimal change disease, and systemic amyloidosis can all cause nephrotic syndrome and subsequent renal vein thrombosis; however, RVT is less frequent with these disorders.

(Choice B) IgA nephropathy usually presents with gross or microscopic hematuria with minimal proteinuria after an upper respiratory tract infection. Patients rarely develop rapidly progressive glomerulonephritis or nephrotic syndrome. This patient's presentation of nephrotic syndrome and symptoms of renal infarction is not consistent with IgA nephropathy.

Educational objective:
Renal vein thrombosis is an important complication of all causes of nephrotic syndrome. However, it is most commonly associated with membranous glomerulopathy.

References

- Disease-specific risk of venous thromboembolic events is increased in idiopathic glomerulonephritis.

Answered correctly
Time Spent
Last Updated

C

Explanation

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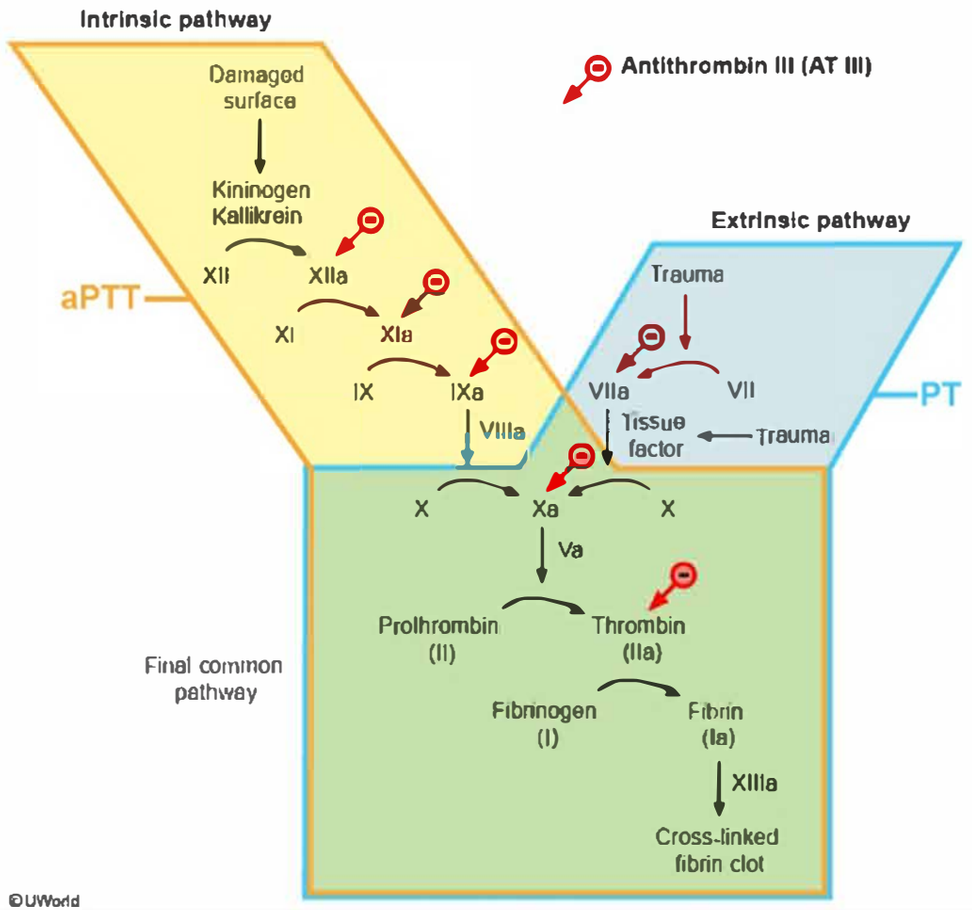
Exhibit Display

Antithrombin III & coagulation cascade



Zoom In | Zoom Out | Reset | Add To Flash Card

A 65-year-old man comes to the hospital with a 1-day history of increasing shortness of breath and cough. He has been using albuterol occasionally without relief of symptoms. The patient is noncompliant with medications and has not seen a doctor in the past year. He smoked 1 pack of cigarettes daily for 40 years. Temperature is 37.5 C (99.5 F), blood pressure is 110/70 mm Hg, pulse is 98/min, and respirations are 20/min. His pulse oximetry shows 86% on room air. Examination shows jugular venous distension, and lung auscultation reveals diffuse wheezing with no crackles. His heart sounds are distant. Mild hepatomegaly is present, and he has bilateral pitting edema halfway to the knees. Chest x-ray reveals hyperinflated lungs and a flattened diaphragm with no infiltrates. He is started on systemic steroids, bronchodilator nebulization, and furosemide. Laboratory studies at the time of admission and 5 days later show the following:

	Admission	Day 5
pH	7.32	7.33
$PaCO_2$	65	45
Bicarbonate (mEq/L)	32	23
Serum sodium (mEq/L)	136	139
Serum potassium (mEq/L)	4.6	4.5
Serum chloride (mEq/L)	101	98
Blood urea nitrogen (mg/dL)	28	60
Serum creatinine (mg/dL)	1.1	2.4
Blood glucose (mg/dL)	110	144

Which of the following is the most likely explanation for the changes in acid-base balance and serum chemistry values between day 1

$PaCO_2$	65	45
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Blood glucose (mg/dL)	110	144

Which of the following is the most likely explanation for the changes in acid-base balance and serum chemistry values between day 1 and day 5 in this patient?

A. Acute kidney injury from diuretic therapy
B. Development of diabetic ketoacidosis
C. Glucocorticoid treatment
D. Obstructive sleep apnea
E. Worsening respiratory failure

Submit

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Which of the following is the most likely explanation for the changes in acid-base balance and serum chemistry values between day 1 and day 5 in this patient?

- A. Acute kidney injury from diuretic therapy [70%]
- B. Development of diabetic ketoacidosis [2%]
- C. Glucocorticoid treatment [17%]
- D. Obstructive sleep apnea [0%]
- E. Worsening respiratory failure [8%]

Omitted
Correct answer
A

70%
Answered correctly

4 Seconds
Time Spent

09/23/2018
Last Updated

Explanation

This patient with an extensive smoking history has a chronic obstructive pulmonary disease (COPD) exacerbation (dyspnea, cough, wheezing, hypoxia), which was treated with glucocorticoids and bronchodilators. In addition, his elevated jugular venous pressure, hepatomegaly, and peripheral edema in the presence of clear lungs suggest pulmonary hypertension and cor pulmonale, likely due to COPD.

In cor pulmonale, loop **diuretics** (eg, furosemide) are often used to lower right ventricular filling volumes and reduce peripheral edema. However, they can lead to hypovolemia, low cardiac output, and renal hypoperfusion. **Prerenal azotemia/acute kidney injury** (AKI) can develop, as reflected by this patient's laboratory values on the fifth day of hospitalization:

- Elevated creatinine (doubled from admission)
- Elevated blood urea nitrogen (BUN) with **BUN:creatinine ratio >20**
- Elevated anion gap **metabolic acidosis** [$(Na) – (Cl + HCO_3) = 139 – (98 + 23) = 18$] likely from **uremia**

Although diuretic use can cause hypokalemia, AKI negates that effect, explaining this patient's stable potassium levels. The other laboratory values are related to his underlying COPD. At baseline, patients with COPD have chronic respiratory acidosis (high $PaCO_2$ from CO_2 retention) with compensatory (renal) metabolic alkalosis (high bicarbonate) and a near normal pH. In COPD exacerbation, there is an acute rise in $PaCO_2$ (acute-on-chronic respiratory acidosis) with a low pH, as seen on admission of this patient. With appropriate treatment, these values should return to their baseline. However, despite having a lowered $PaCO_2$ (likely close to his baseline), this patient continues to have low pH and a serum bicarbonate level below what would be expected in chronic COPD. This suggests development of metabolic acidosis due to renal failure and uremia.

(Choices B and C) Diabetic ketoacidosis is typically accompanied by much higher glucose levels (eg, >400 mg/dL). Glucocorticoids can increase serum BUN due to their catabolic effect on body proteins and can cause hyperglycemia; however, they are unlikely to cause creatinine elevation.

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(Choices D and E) Obstructive sleep apnea can lead to chronic respiratory acidosis with compensatory metabolic alkalosis but would not cause acute metabolic acidosis. Worsening respiratory failure would lead to an increase (not a decrease) in $PaCO_2$.

Educational objective:
Patients with chronic obstructive pulmonary disease often have chronic CO_2 retention, resulting in respiratory acidosis and compensatory metabolic alkalosis. Diuretics are often administered to treat cor pulmonale symptoms but must be used cautiously as they can lead to a reduction in cardiac output and subsequent development of prerenal acute renal injury.

edema. However, they can lead to hypovolemia, low cardiac output, and renal hypoperfusion. **Prerenal azotemia/acute kidney injury** (AKI) ca

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Exhibit Display

Anion gap metabolic acidosis	
Calculation	Anion gap = Sodium – (Chloride + Bicarbonate) (Normal = 10-14)
Common causes Mnemonic: MUDPILES	• Methanol • Uremia • Diabetic ketoacidosis • Propylene glycol/paraldehyde • Isoniazid/iron • Lactic acidosis • Ethylene glycol (antifreeze) • Salicylates (aspirin)

Add To Flash Card

A 32-year-old man with a known history of recreational drug abuse is found by a friend on the floor of his apartment. There is a pool of urine around him. He is confused, not oriented to time or place, and does not recall recent events. His blood pressure is 110/70 mm Hg and pulse is 120/min. He talks and moves all his extremities. His laboratory findings are the following:

Hemoglobin	15.2 mg/dL
Leukocytes	12,500/mm^3
Platelets	160,000/mm^3
Sodium	136 mEq/L
Potassium	5.1 mEq/L
Creatinine	1.1 mg/dL
Aspartate aminotransferase	42 units/L
Alanine aminotransferase	40 units/L
Alkaline phosphatase	70 units/L
Creatine phosphokinase	26,000 units/L

His urine toxicology screen is positive for cocaine and cannabinoids. The patient is at the greatest risk of which of the following?

A. Aseptic meningitis
B. Acute renal failure

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His urine toxicology screen is positive for cocaine and cannabinoids. The patient is at the greatest risk of which of the following?

- A. Aseptic meningitis
- B. Acute renal failure
- C. Reye syndrome
- D. Dermatomyositis
- E. Splenic rupture

Submit

A 32-year-old man with a known history of recreational drug abuse is found by a friend on the floor of his apartment. There is a pool of urine around him. He is confused, not oriented to time or place, and does not recall recent events. His blood pressure is 110/70 mm Hg and pulse is 120/min. He talks and moves all his extremities. His laboratory findings are the following:

Hemoglobin	15.2 mg/dL
Leukocytes	12,500/mm^3
Platelets	160,000/mm^3
Sodium	136 mEq/L
Potassium	5.1 mEq/L
Creatinine	1.1 mg/dL
Aspartate aminotransferase	42 units/L
Alanine aminotransferase	40 units/L
Alkaline phosphatase	70 units/L
Creatine phosphokinase	26,000 units/L

His urine toxicology screen is positive for cocaine and cannabinoids. The patient is at the greatest risk of which of the following?

A. Aseptic meningitis [4%]
✔ B. Acute renal failure [88%]

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Alanine aminotransferase	40 units/L
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Creatine phosphokinase	26,000 units/L

His urine toxicology screen is positive for cocaine and cannabinoids. The patient is at the greatest risk of which of the following?

- A. Aseptic meningitis [4%]
- ✔ B. Acute renal failure [88%]
- C. Reye syndrome [1%]
- D. Dermatomyositis [3%]
- E. Splenic rupture [1%]

Omitted
Correct answer
B

88%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

The greatest risk for this patient is acute renal failure due to **rhabdomyolysis**. There is ample evidence that this patient has

Explanation

The greatest risk for this patient is acute renal failure due to **rhabdomyolysis**. There is ample evidence that this patient has rhabdomyolysis. First, he has 2 strong risk factors for rhabdomyolysis: immobilization and cocaine abuse. Prolonged **immobilization**, as suggested by his presence on the floor, causes direct muscle damage and the release of creatine phosphokinase (CPK), also known as creatine kinase. Similarly, **cocaine** is a potent vasoconstrictor that causes diffuse ischemia, seizures, agitation, incidental trauma, hyperpyrexia and a direct toxic effect on myocytes. All of these may contribute to increased muscle breakdown. Second, the **elevated potassium and CPK concentrations** are strongly suggestive of rhabdomyolysis. As **muscle cells break down**, potassium, CPK, and myoglobin are released into the blood stream. In fact, 20% of cocaine overdoses are complicated by these laboratory abnormalities. Elevations in aspartate and alanine aminotransferase levels can also be seen with muscle breakdown.

Renal failure in rhabdomyolysis is caused by acute tubular necrosis from excessive filtered myoglobin. The risk of **myoglobin-induced renal failure** is significant when the CPK concentration is >20,000 units/L. Aside from an elevated CPK, rhabdomyolysis is suggested when a **urine dipstick tests positive for blood, but no red blood cells** are seen on microscopy. This finding is caused by **myoglobin in the urine**. The risk of rhabdomyolysis-induced renal failure may be decreased with aggressive hydration. Mannitol and urine alkalinization may also be beneficial.

(Choice A) Aseptic meningitis is meningeal infection and/or inflammation in which no causative organisms are found on routine CSF stains and cultures. It is most commonly caused by Coxsackie virus or Echovirus. Intravenous drug abuse can predispose to bacterial meningitis.

(Choice C) Reye syndrome is an often fatal childhood hepatoencephalopathy associated with a viral infection and treatment with aspirin. It is not associated with rhabdomyolysis.

(Choice D) Dermatomyositis is an inflammatory autoimmune myopathy that causes direct destruction of skeletal myocytes. It is, therefore, associated with elevated CPK levels, but it does not fit the context of this otherwise healthy patient who recently used

breakdown.

Renal failure in rhabdomyolysis is caused by acute tubular necrosis from excessive filtered myoglobin. The risk of **myoglobin-induced renal failure** is significant when the CPK concentration is >20,000 units/L. Aside from an elevated CPK, rhabdomyolysis is suggested when a **urine dipstick tests positive for blood, but no red blood cells** are seen on microscopy. This finding is caused by **myoglobin in the urine**. The risk of rhabdomyolysis-induced renal failure may be decreased with aggressive hydration. Mannitol and urine alkalinization may also be beneficial.

(Choice A) Aseptic meningitis is meningeal infection and/or inflammation in which no causative organisms are found on routine CSF stains and cultures. It is most commonly caused by Coxsackie virus or Echovirus. Intravenous drug abuse can predispose to bacterial meningitis.

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(Choice D) Dermatomyositis is an inflammatory autoimmune myopathy that causes direct destruction of skeletal myocytes. It is, therefore, associated with elevated CPK levels, but it does not fit the context of this otherwise healthy patient who recently used cocaine.

(Choice E) Splenic rupture generally results from blunt abdominal trauma. Neither cocaine nor marijuana use are risk factors if trauma is not involved. Infectious mononucleosis and post transplant Epstein-Barr virus-mediated pseudolymphoma can also predispose to splenic rupture.

Educational objective:
Nearly 20% of cocaine overdoses are complicated by rhabdomyolysis, as indicated by marked elevations in serum creatine phosphokinase (CPK). The main danger associated with CPK levels >20,000 U/L is acute renal tubular necrosis due to myoglobinuria.

A 54-year-old man comes to the emergency department due to a cough. He has a 44-pack-year smoking history but quit a month ago. The patient has also experienced weight loss, anorexia, constipation, increased thirst, and fatigue, which he attributes to depression. He lost his wife to breast cancer 4 months ago and says that "life hasn't felt the same since then." Blood pressure is 104/68 mm Hg, pulse is 95/min, and respirations are 24/min. Oxygen saturation is 92% on room air. The patient appears thin, pale, and short of breath. Laboratory results are as follows:

Sodium	144 mEq/dL
Potassium	4.3 mEq/dL
Chloride	98 mEq/dL
Bicarbonate	21 mEq/dL
Calcium	14.5 mg/dL
Blood urea nitrogen	48 mg/dL
Creatinine	2.0 mg/dL

Chest x-ray reveals a hilar mass in the left lung. What is the best next step in management of this patient?

A. CT scan of the head [5%]
B. Emergency hemodialysis [8%]
C. Glucocorticoids [3%]

Sodium	mEq/dL
Potassium	4.3 mEq/dL
Chloride	98 mEq/dL
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- B. Emergency hemodialysis
- C. Glucocorticoids
- D. Loop diuretics
- E. Saline hydration

Submit

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- A. CT scan of the head [5%]
- B. Emergency hemodialysis [8%]
- C. Glucocorticoids [3%]
- D. Loop diuretics [9%]
- ✔ E. Saline hydration [72%]

Omitted
Correct answer
E

72%
Answered correctly

222 Seconds
Time Spent

12/22/2018
Last Updated

Explanation

Management of hypercalcemia	
Severe (calcium >14 mg/dL) or symptomatic	**Short-term (immediate) treatment** • Normal saline hydration plus calcitonin • Avoid loop diuretics unless volume overload (heart failure) exists **Long-term treatment** • Bisphosphonate (zoledronic acid)

Omitted
Correct answer
E

72%
Answered correctly

222 Seconds
Time Spent

12/22/2018
Last Updated

Explanation

Management of hypercalcemia	
Severe (calcium >14 mg/dL) or symptomatic	**Short-term (immediate) treatment** • Normal saline hydration plus calcitonin • Avoid loop diuretics unless volume overload (heart failure) exists **Long-term treatment** • Bisphosphonate (zoledronic acid)
Moderate (calcium 12-14 mg/dL)	• Usually no immediate treatment required unless symptomatic • Treatment is similar to that for severe hypercalcemia
Asymptomatic or mild (calcium <12 mg/dL)	• No immediate treatment required • Avoid thiazide diuretics, lithium, volume depletion & prolonged bed rest

This patient has severe symptomatic hypercalcemia. In light of his smoking history and associated lung mass, this likely represents **humoral hypercalcemia of malignancy** due to secretion of parathyroid hormone-related protein. **Severe hypercalcemia** (serum calcium >14 mg/dL), especially with a rapid rise in serum calcium, can cause weakness, gastrointestinal distress, and neuropsychiatric symptoms (eg, confusion, stupor, coma). Patients are typically **volume depleted** (as suggested by this patient's blood urea nitrogen to creatinine ratio >20) due to both polyuria from hypercalcemia-induced nephrogenic diabetes insipidus and decreased oral intake.

Patients with severe hypercalcemia require **aggressive saline hydration** (ie, several liters of normal saline) to restore intravascular volume and promote urinary calcium excretion. **Calcitonin**, by inhibiting osteoclast-mediated bone resorption, quickly reduces serum calcium concentrations and may be administered concurrently with saline. **Bisphosphonates** (eg, pamidronate, zoledronic acid) also inhibit bone resorption and provide a sustained reduction in calcium levels. However, the calcium-lowering effect of bisphosphonates is delayed, usually occurring over 2-4 days, and they are typically given after initial administration of saline and calcitonin.

(Choice A) This patient may have metastatic carcinoma of the lung, which warrants additional imaging. However, the hypercalcemia and hypovolemia must be corrected urgently, especially before administration of radiographic contrast agents.

(Choice B) Hemodialysis is an effective treatment for hypercalcemia. However, it is typically reserved for patients with renal insufficiency or heart failure in whom aggressive hydration cannot be administered safely.

(Choice C) Glucocorticoids inhibit the formation of 1,25-dihydroxyvitamin D by activated mononuclear cells in the lungs and lymph nodes. Glucocorticoids can be used to treat hypercalcemia due to excessive vitamin D intake, granulomatous diseases (eg, sarcoidosis), and certain lymphomas. However, most cases of malignancy-related hypercalcemia are not due to vitamin D excess.

(Choice D) Although loop diuretics can promote diuresis and reduce calcium levels, their routine use in hypercalcemic patients is not recommended as they can worsen the volume depletion.

Educational objective:
Severe hypercalcemia can cause weakness, gastrointestinal distress, and neuropsychiatric symptoms. Patients are typically volume depleted due to polyuria and decreased oral intake. Initial treatment includes saline hydration to restore intravascular volume and calcitonin to inhibit bone resorption. Bisphosphonates further reduce calcium levels and are given after initial administration of saline.

References

- Treatment of acute hypercalcemia.

A 32-year-old man comes to the emergency room (ER) because of acute onset left flank pain, hematuria and vomiting. His pain is relieved with analgesics in the ER. He has a history of abdominal pain due to Crohn disease, but that pain was always in the right lower quadrant and was never this severe. His temperature is 36.8°C (98.2°F), blood pressure is 120/65 mm Hg, pulse is 110/min and respirations are 16/min. Chest auscultation is clear. Abdomen is soft and mildly tender over the left flank. He has no rebound or rigidity. Bowel sounds are decreased. A laparotomy scar is present in right lower quadrant. Which of the following is the most likely cause of his symptoms?

A. Increased recycling of bile salts and fatty acids
B. Increased absorption of oxalate
C. Increased absorption of calcium
D. Increased parathyroid hormone activity
E. Recurrent bacteria infection in the kidney

Submit

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- A. Increased recycling of bile salts and fatty acids [5%]
- ✔ B. Increased absorption of oxalate [64%]
- C. Increased absorption of calcium [14%]
- D. Increased parathyroid hormone activity [9%]
- E. Recurrent bacteria infection in the kidney [6%]

Omitted
Correct answer
B

64%
Answered correctly

5 Seconds
Time Spent

09/22/2018
Last Updated

Explanation

The patient described is experiencing symptoms consistent with nephrolithiasis, which classically presents with flank pain and hematuria frequently accompanied by nausea and vomiting. Patients with Crohn disease, or any other small intestinal disorder

Correct answer
B

64%
Answered correctly

5 Seconds
Time Spent

09/22/2018
Last Updated

Explanation

The patient described is experiencing symptoms consistent with nephrolithiasis, which classically presents with flank pain and hematuria frequently accompanied by nausea and vomiting. Patients with Crohn disease, or any other small intestinal disorder resulting in fat malabsorption, are predisposed to hyperoxaluria. Oxalate is obtained from the diet and is a normal product of human metabolism. Symptomatic hyperoxaluria is classically the result of increased oxalate absorption in the gut. Under normal circumstances, calcium binds oxalate in the gut and prevents its absorption. In patients with fat malabsorption, calcium is preferentially bound by fat leaving oxalate unbound and free to be absorbed into the bloodstream. Failure to adequately absorb bile salts in states of fat malabsorption also cause decreased bile salt reabsorption in the small intestine. Excess bile salts may damage the colonic mucosa and contribute to increased oxalate absorption.

(Choice A) Bile salt recycling and fatty acid absorption are decreased in Crohn disease.

(Choices C and D) Hypercalciuria, which may be idiopathic or may result from hyperparathyroidism, excessive calcium and vitamin D ingestion, dehydration or prolonged immobilization, predisposes to calcium stone formation.

(Choice E) Recurrent urinary tract infections, particularly by Proteus species, predispose to struvite stone formation. Struvite stones may eventually grow to fill the entire renal pelvis, at which point they are known as "staghorn" calculi.

Educational objective:
Oxalate absorption is increased in Crohn disease and all other intestinal diseases causing fat malabsorption. Increased absorption is the most common cause of symptomatic hyperoxaluria and oxalate stone formation.

A 65-year-old man comes to the physician after noticing blood in his urine. He has had 2 episodes of bloody urine over the last month that cleared spontaneously. He has no abdominal pain or dysuria or any recent illness. The patient has a 90-pack-year smoking history and quit 5 years ago. There are no family members with kidney disease or cancer. He has a history of hypertension and a transient ischemic attack. His medications include a daily aspirin and hydrochlorothiazide. Temperature is 36.7 C (98 F), blood pressure is 130/86 mm Hg, pulse is 80/min, and respirations are 16/min. Physical examination, including digital rectal examination, is unremarkable. Laboratory results are as follows:

Urinalysis	
Specific gravity	1.016
pH	6.4
Protein	None
Blood	Moderate
Glucose	Negative
Leukocyte esterase	Negative
Nitrites	Negative
Bacteria	None
Red blood cells	20-30/high power field
Casts	None
Crystals	None

Glucose	Negative
Leukocyte esterase	Negative
Nitrites	Negative
Bacteria	None
Red blood cells	20-30/high power field
Casts	None
Crystals	None

Which of the following is the most likely cause of his symptoms?

- A. Acute glomerulonephritis
- B. Bladder cancer
- C. Polycystic kidney disease
- D. Prostate cancer
- E. Urinary tract infection
- F. Urolithiasis

Submit

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Nitrites	Negative
Bacteria	None
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Casts	None
Crystals	None

Which of the following is the most likely cause of his symptoms?

- A. Acute glomerulonephritis [2%]
- ✔ B. Bladder cancer [94%]
- C. Polycystic kidney disease [0%]
- D. Prostate cancer [0%]
- E. Urinary tract infection [0%]
- F. Urolithiasis [2%]

Omitted
Correct answer
B

94%
Answered correctly

5 Seconds
Time Spent

11/25/2018
Last Updated

Explanation

This patient has gross painless hematuria. Common causes of hematuria in the United States include neoplasms, infections, trauma, nephrolithiasis, glomerulonephritis, and prostatic disease (eg, benign prostatic hyperplasia). Initial assessment includes urinalysis to rule out urinary tract infection and confirm microhematuria (≥3 red blood cells [RBC]/hpf) as red urine can occur in other conditions (eg, myoglobinuria, beet ingestion, rifampin use). Renal disease (eg, glomerulonephritis) is usually associated with RBC casts, proteinuria, hypertension, peripheral edema, or elevated creatinine. This patient's absence of RBC casts, bacteria, and proteinuria make glomerulonephritis and urinary tract infection unlikely **(Choices A and E)**.

Nearly 80% of malignancies of the kidney, ureter, and bladder in adults present with gross hematuria. Risk factors include age >35, smoking history, occupational history (eg, chemicals, dyes), and drug exposure (eg, cyclophosphamide). These patients should have imaging (eg, contrast CT scan) and cystoscopy to evaluate the bladder and urethra. Of these malignancies, bladder cancer is the most common cause of gross hematuria in patients age >35 with a smoking history.

(Choice C) Autosomal dominant polycystic kidney disease (ADPKD) is a progressive kidney disease that usually leads to end-stage renal disease by age 60. ADPKD usually presents with gross hematuria, recurrent flank pain, kidney cysts, urinary tract infection/pyelonephritis, and nephrolithiasis. This patient's absence of family history of ADPKD makes this less likely.

(Choice D) Prostate cancer can be asymptomatic and associated with elevated *prostate-specific antigen* or firm nodules on prostate examination. Symptomatic patients can have lower urinary tract symptoms (eg, hesitancy, frequency, weak urinary stream). However, gross hematuria is more commonly associated with benign prostatic hyperplasia than prostate cancer.

(Choice F) Urolithiasis can cause hematuria but is usually associated with flank pain radiating to the groin.

Educational objective:
Bladder tumors are the most common malignancy associated with painless hematuria in adults. Patients age >35 with gross hematuria should be evaluated for urological neoplasms with diagnostic imaging (CT urogram) and cystoscopy.

proteinuria, hypertension, peripheral edema, or elevated creatinine. This patient's absence of RBC casts, bacteria, and proteinuria make glomerulonephritis and urinary tract infection unlikely **(Choices A and E)**.

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Educational objective:
Bladder tumors are the most common malignancy associated with painless hematuria in adults. Patients age >35 with gross hematuria should be evaluated for urological neoplasms with diagnostic imaging (CT urogram) and cystoscopy.

References

- Stratifying risk of urinary tract malignant tumors in patients with asymptomatic microscopic hematuria.
- A prospective analysis of 1,930 patients with hematuria to evaluate current diagnostic practice.

A 7-year-old boy is brought to the office due to persistent bedwetting. Since age 3, he has been able to stay dry during the day but continues to have "accidents" 3 nights a week. The child drinks fluids primarily in the morning and early afternoon and does not drink juice or caffeinated beverages. For the past 4 months, his parents have tried enuresis alarms and awarded "gold stars" for dry nights without improvement in the frequency of bedwetting. His parents are frustrated, and the patient is sad about missing activities such as summer camp. He was recently invited to a sleepover party but is nervous about wetting the bed. He has otherwise been in good health, has met all other developmental milestones, and takes no medications. The father had a history of nocturnal enuresis until age 8. Physical examination and urinalysis are normal. Which of the following is the best next step in management of this patient?

- A. Desmopressin
- B. Imipramine
- C. Oxybutynin
- D. Serum glucose
- E. Urine culture
- F. Vesicoureterogram

Submit

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- ✔ A. Desmopressin [65%]
- B. Imipramine [15%]
- C. Oxybutynin [5%]
- D. Serum glucose [4%]
- E. Urine culture [3%]
- F. Vesicoureterogram [5%]

Omitted
Correct answer
A

65%
Answered correctly

3 Seconds
Time Spent

09/25/2018
Last Updated

Explanation

Explanation

Primary nocturnal enuresis	
Definition	• Urinary incontinence age ≥5

Management	• Urinalysis to rule out secondary causes
	• Lifestyle changes:
	  ◦ Minimize fluid intake before bedtime
	  ◦ Avoid sugary/caffeinated beverages
	  ◦ Void before bedtime
	  ◦ Institute reward system (eg, "gold star" chart)
	• Enuresis alarm
	• Desmopressin therapy

Primary nocturnal enuresis is defined by urinary incontinence in children **age ≥5** who have never achieved nighttime dryness and have no underlying medical condition explaining enuresis. Boys with a family history of delayed bladder control, as in this case, are predisposed to prolonged bedwetting.

Although enuresis has a high rate of spontaneous resolution over time, many families seek active intervention to improve quality of life due to embarrassment or anxiety. Initial steps in management include **behavioral modifications** such as voiding before bedtime and minimizing evening fluid intake.

For children who do not respond to lifestyle changes, **enuresis alarms** are the most effective long-term intervention but can take 3-4 months to be effective. Pharmacotherapy with **desmopressin** is also considered first-line therapy for those who desire immediate improvement. Oral desmopressin, an antidiuretic hormone analogue, decreases urine production during sleep. Patients should be counseled on minimizing fluid intake in the evening to prevent hyponatremia with therapy. In addition, the rate of relapse on

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(Choice B) Tricyclic antidepressants (eg, imipramine) are as effective as desmopressin. However, they are reserved for patients who do not respond to desmopressin due to their rare, but serious, side effects (eg, increased suicidality, cardiotoxicity).

(Choice C) Oxybutynin is an anticholinergic medication that causes urinary retention. In children with nighttime and daytime incontinence, it can be used in conjunction with desmopressin to increase bladder capacity. This child has only nocturnal enuresis.

(Choice D) Polyuria and enuresis are symptoms of diabetes mellitus. Screening for diabetes mellitus includes urinalysis for glucosuria or serum glucose for hyperglycemia. This patient's urinalysis is normal, and no further testing is required.

(Choice E) Urine culture should be considered in a patient with signs of a urinary tract infection (eg, dysuria, hematuria) or an abnormal urinalysis (eg, positive nitrites).

(Choice F) A voiding cystourethrogram is used to diagnose vesicoureteral reflux, which can present with recurrent urinary tract infections. This patient has isolated nocturnal enuresis with no history of infection.

Educational objective:
Desmopressin is the first-line pharmacotherapy for nocturnal enuresis. It can provide immediate improvement when behavioral modifications and alarm therapy have failed. When used as monotherapy, it has a high rate of relapse on discontinuation of therapy.

References

- Evaluation of and treatment for monosymptomatic enuresis: a standardization document from the International Children's Continence Society

A 5-year-old boy is brought to the physician due to a 1-week history of generalized edema, fatigue, and abdominal pain. Otherwise, he has been well and his medical history is unremarkable. The patient takes a daily multivitamin and no other medications. Blood pressure is 92/55 mm Hg and pulse is 90/min. Periorbital edema and 1+ pretibial edema are found on examination. The scrotum is mildly swollen but nontender. Abdominal examination is unremarkable. Urinalysis results are as follows:

Specific gravity	1.028
pH	5
Protein	4+
Blood	negative
Casts	none
Crystals	none

Which of the following light microscopy findings would be expected if a kidney biopsy were performed?

A. Crescent formation
B. Diffuse thickening of basement membrane
C. Mesangial hypercellularity
D. Normal findings
E. Subepithelial spikes

A 5-year-old boy is brought to the physician due to a 1-week history of generalized edema, fatigue, and abdominal pain. Otherwise, he has been well and his medical history is unremarkable. The patient takes a daily multivitamin and no other medications. Blood pressure is 92/55 mm Hg and pulse is 90/min. Periorbital edema and 1+ pretibial edema are found on examination. The scrotum is mildly swollen but nontender. Abdominal examination is unremarkable. Urinalysis results are as follows:

Specific gravity	1.028
pH	5
Protein	4+
Blood	negative
Casts	none
Crystals	none

Which of the following light microscopy findings would be expected if a kidney biopsy were performed?

- A. Crescent formation [2%]
- B. Diffuse thickening of basement membrane [6%]
- C. Mesangial hypercellularity [3%]
- ✔ D. Normal findings [84%]
- E. Subepithelial spikes [3%]

Omitted
Correct answer
D

84%
Answered correctly

6 Seconds
Time Spent

08/23/2018
Last Updated

Explanation

Minimal change disease	
Epidemiology	• Most common cause of nephrotic syndrome in children • Median age 2-3
Pathogenesis	• T cell-mediated injury to podocytes
Clinical features	• Edema • Fatigue
Diagnosis	• Proteinuria • Renal biopsy without microscopic changes
Treatment	Corticosteroids

This child's generalized **edema, fatigue, abdominal pain** (from rapid fluid accumulation), and **proteinuria** are consistent with nephrotic syndrome. The most likely diagnosis is **minimal change disease (MCD)** as it accounts for 80% of pediatric nephrotic syndrome cases. Children age <10 with isolated nephrotic syndrome usually do not require biopsy for diagnosis as MCD is highly likely. If performed, light microscopy of tissue obtained from renal biopsy usually demonstrates **normal renal architecture**;

syndrome cases. Children age <10 with isolated nephrotic syndrome usually do not require biopsy for diagnosis as MCD is highly likely. If performed, light microscopy of tissue obtained from renal biopsy usually demonstrates **normal renal architecture**; immunofluorescence staining of the glomeruli also shows no abnormality. Diffuse **effacement of foot processes** of podocytes (renal epithelial cells) on electron microscopy is confirmatory for MCD.

The majority of patients respond rapidly to **corticosteroid** therapy; >90% of children have **complete remission** with the disappearance of proteinuria. Therefore, biopsy is reserved for steroid-resistant or progressive disease. Older children or adolescents should undergo renal biopsy to exclude other causes of nephrotic syndrome.

(Choice A) Crescent formation is a typical finding of rapidly progressive glomerulonephritis, which is a severe condition associated with a rapid decline in renal function and crescent formation in the majority of glomeruli.

(Choices B and E) Thickened basement membrane and subepithelial "spikes" are pathognomonic for membranous glomerulonephritis, which is one of the most common causes of nephrotic syndrome in adults. However, it is rare in young children.

(Choice C) Mesangial hypercellularity is suggestive of membranoproliferative glomerulonephritis, which most commonly presents with nephritic syndrome rather than nephrotic syndrome. It is uncommon in young children.

Educational objective:
Minimal change disease is the most common cause of nephrotic syndrome in preadolescent children. Renal biopsy shows normal kidney architecture but is not routinely obtained in patients age <10. Steroids are the treatment of choice.

References

- Minimal change (steroid sensitive) nephrotic syndrome in children: new aspects on pathogenesis and treatment.
- The nephrotic syndrome.

A 43-year-old woman with hepatitis C, alcohol use, and liver cirrhosis is admitted to the hospital for severe vomiting over the last 2 days. The vomitus has appeared clear with no blood or coffee-ground material. She feels very weak but has no abdominal pain, fever, or diarrhea. The patient appears lethargic on examination. Her temperature is 36.8 C (98.2 F), blood pressure is 100/70 mm Hg (while supine), pulse is 90/min, and respirations are 14/min. Lungs are clear to auscultation. There is mild epigastric tenderness but no abdominal distension. Extremities have no edema. Laboratory results are as follows:

Serum chemistry	
Sodium	138 mEq/L
Potassium	3.0 mEq/L
Chloride	95 mEq/L
Bicarbonate	30 mEq/L
Arterial blood gases	
pH	7.49
PaO_2	100 mm Hg
$PaCO_2$	41 mm Hg

Which of the following is the most appropriate initial treatment for this patient?

A. Acetazolamide

B. Intravenous normal saline and potassium

Potassium	3.0 mEq/L
Chloride	95 mEq/L
Bicarbonate	30 mEq/L

Arterial blood gases

pH	7.49
PaO_2	100 mm Hg
$PaCO_2$	41 mm Hg

Which of the following is the most appropriate initial treatment for this patient?

- A. Acetazolamide
- B. Intravenous normal saline and potassium
- C. Intravenous sodium bicarbonate and potassium
- D. Oral lactulose and oral potassium
- E. Propranolol
- F. Spironolactone

Submit

A 43-year-old woman with hepatitis C, alcohol use, and liver cirrhosis is admitted to the hospital for severe vomiting over the last 2 days. The vomitus has appeared clear with no blood or coffee-ground material. She feels very weak but has no abdominal pain, fever, or diarrhea. The patient appears lethargic on examination. Her temperature is 36.8 C (98.2 F), blood pressure is 100/70 mm Hg (while supine), pulse is 90/min, and respirations are 14/min. Lungs are clear to auscultation. There is mild epigastric tenderness but no abdominal distension. Extremities have no edema. Laboratory results are as follows:

Serum chemistry

Sodium	138 mEq/L
Potassium	3.0 mEq/L
Chloride	95 mEq/L
Bicarbonate	30 mEq/L

Arterial blood gases

pH	7.49
PaO_2	100 mm Hg
$PaCO_2$	41 mm Hg

Which of the following is the most appropriate initial treatment for this patient?

A. Acetazolamide [2%]

✔ B. Intravenous normal saline and potassium [87%]

Chloride	95 mEq/L
Bicarbonate	30 mEq/L

Arterial blood gases

pH	7.49
PaO_2	100 mm Hg
$PaCO_2$	41 mm Hg

Which of the following is the most appropriate initial treatment for this patient?

A. Acetazolamide [2%]
B. Intravenous normal saline and potassium [87%]
C. Intravenous sodium bicarbonate and potassium [1%]
D. Oral lactulose and oral potassium [6%]
E. Propranolol [0%]
F. Spironolactone [1%]

Omitted
Correct answer
B

87%
Answered correctly

6 Seconds
Time Spent

10/31/2018
Last Updated

Explanation

Repeated **vomiting** causes hypokalemic, hypochloremic, **metabolic alkalosis**. The pathogenesis can be separated into 2 phases:

- Generation phase: Repeated vomiting leads to depletion of total body acid (gastric contents are rich in hydrochloric acid) as well as loss of fluid, sodium, and chloride. Normally, acidic gastric secretions stimulate the pancreas and liver to excrete bicarbonate into the duodenum. However, in patients with recurrent vomiting, this bicarbonate excretion does not occur due to loss of gastric acid, leading to buildup of alkali (metabolic alkalosis).
- Maintenance phase: Although excess alkali are filtered in the glomerulus, they are also reabsorbed as a result of an activated renin-angiotensin-aldosterone system (RAAS). Fluid depletion from vomiting triggers RAAS in an attempt to conserve sodium and water. Aldosterone functions to retain water at the expense of excreting both potassium and acid in the urine, despite total body acid depletion **(contraction alkalosis)**.

Restoration of the extracellular volume with **intravenous fluids** (such as normal saline) removes the stimulus for RAAS activation, restoring the kidney's ability to excrete excess bicarbonate. Potassium supplementation should also be administered to treat hypokalemia (which should improve with normalization of the acid-base status).

(Choices A and F) This patient is volume depleted, and diuretic therapy would worsen the contraction alkalosis. Acetazolamide, a diuretic that inhibits proximal renal bicarbonate reabsorption, may be used in patients with hypervolemia and metabolic alkalosis. Spironolactone, a potassium-sparing diuretic, is used in combination with furosemide for treatment of ascites.

(Choice C) Intravenous sodium bicarbonate is used to treat severe metabolic acidosis and could worsen this patient's alkalosis.

(Choice D) Oral lactulose is used to treat hepatic encephalopathy in patients with cirrhosis. This patient has no evidence of encephalopathy.

(Choice E) Propranolol is used for both primary and secondary prophylaxis of variceal hemorrhage in patients with liver cirrhosis. This patient has no evidence of a variceal bleed.

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(Choice E) Propranolol is used for both primary and secondary prophylaxis of variceal hemorrhage in patients with liver cirrhosis. This patient has no evidence of a variceal bleed.

Educational objective:
Recurrent vomiting causes depletion of fluid, acid, and sodium chloride, leading to metabolic alkalosis, activation of the renin-angiotensin-aldosterone system, and increased urinary potassium loss. Volume resuscitation with normal saline corrects contraction alkalosis. Hypokalemia should be treated as well.

A 45-year-old man is brought to the emergency department after being involved in a motor vehicle collision. He has pain in the lower abdomen and right groin. The patient is unable to move his right lower extremity due to pain. Temperature is 37 C (98.6 F), blood pressure is 130/82 mm Hg, pulse is 100/min, and respirations are 16/min. There is fullness in the suprapubic region. The right lower extremity is adducted, flexed, and internally rotated. Bruising is present around the scrotum and perineum and blood is seen at the urethral meatus. The testicles are not tender on palpation. Digital rectal examination reveals a high-riding prostate. Which of the following is the most appropriate next step in management of this patient?

A. Foley catheterization
B. Immediate surgical repair of urethra
C. Retrograde cystogram
D. Retrograde urethrogram
E. Testicular and scrotal ultrasound

Submit

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- A. Foley catheterization [4%]
- B. Immediate surgical repair of urethra [14%]
- C. Retrograde cystogram [7%]
- ✔ D. Retrograde urethrogram [70%]
- E. Testicular and scrotal ultrasound [3%]

Omitted
Correct answer
D

70%
Answered correctly

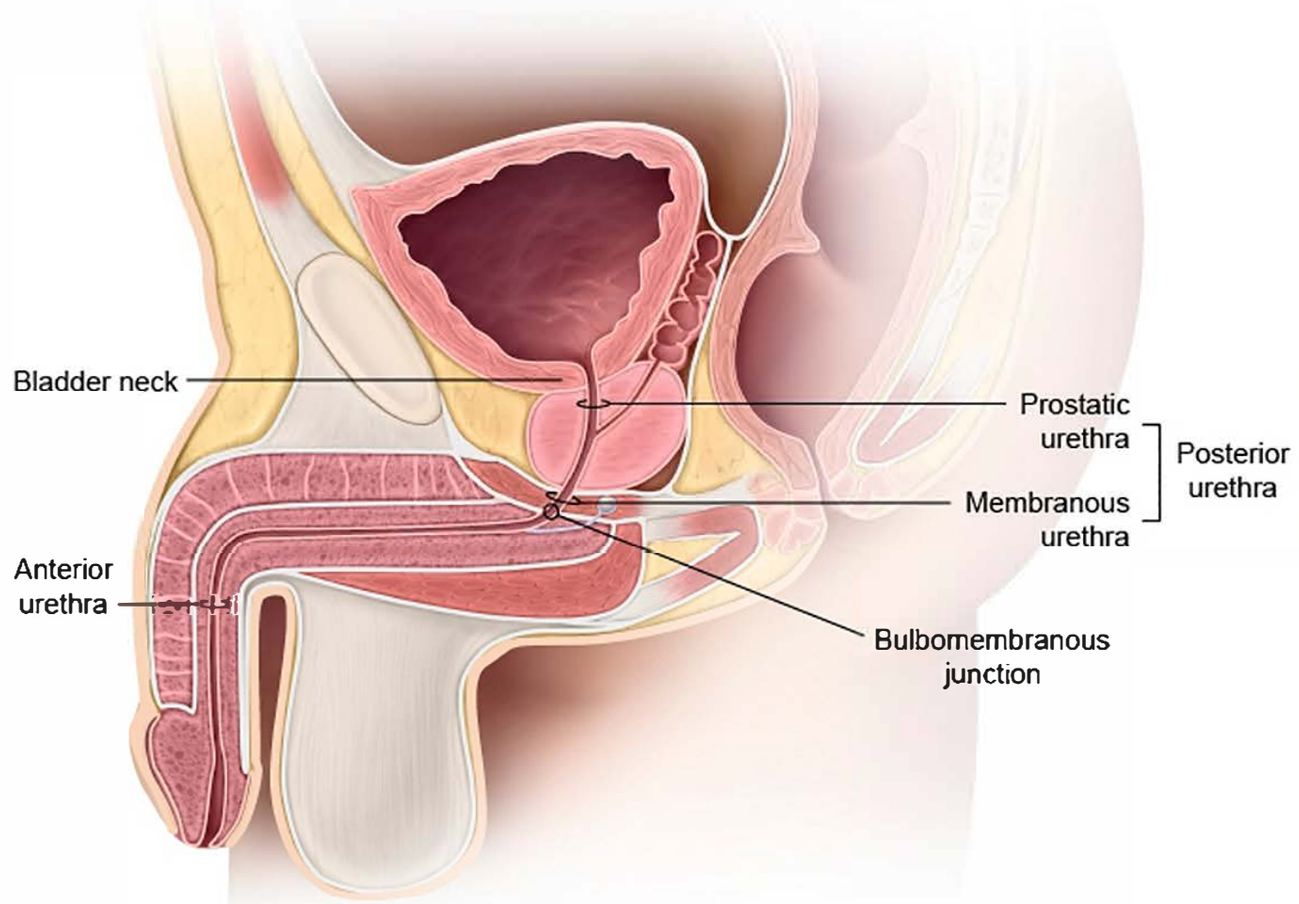
3 Seconds
Time Spent

09/23/2018
Last Updated

Explanation

Male urogenital anatomy

Male urogenital anatomy



This patient's adducted and internally rotated right lower extremity (suggestive of acetabular fracture with posterior hip dislocation).

This patient's adducted and internally rotated right lower extremity (suggestive of acetabular fracture with posterior hip dislocation), pain in the lower abdomen and groin, and bruising along the scrotum and perineum are consistent with **pelvic fracture**. Due to the length and positioning of the male urethra, men with pelvic fractures are at significant risk for **posterior urethral injury (PUI)**. Abrupt upward shifting of the bladder and prostate can lead to urethral tearing, which most commonly affects the membranous urethra at the **bulbomembranous junction** (dividing point between the anterior and posterior urethra). Findings consistent with PUI include **blood at the urethral meatus**, inability to void, perineal or scrotal hematoma, and a **high-riding prostate** on digital rectal examination.

In patients with suspected urethral injury, a **retrograde urethrogram** should be performed. This diagnostic test involves an x-ray of the lower genitourinary tract obtained during the injection of radiopaque contrast into the urethra. A normal study demonstrates contrast entering the bladder uninterrupted. **Extravasation of contrast** from the urethra or inability of contrast to reach the bladder is diagnostic of urethral injury.

(Choice A) Attempting to pass a Foley catheter should generally be avoided in patients with suspected urethral injury as it can worsen periurethral or perivesical hematomas and can convert a partial into a complete urethral laceration.

(Choice B) Immediate surgical repair is occasionally done in cases of anterior urethral injury, which often occurs with straddle injury and is not commonly associated with pelvic fracture. Most cases of urethral injury are treated with temporary urinary diversion by suprapubic catheter followed by delayed repair.

(Choice C) A retrograde cystogram uses radiopaque contrast to diagnose bladder rupture, which, like PUI, is commonly associated with pelvic fracture. Patients typically have gross hematuria and possibly difficulty voiding. However, blood at the urethral meatus, ecchymosis of the scrotum, and a high-riding prostate are more consistent with PUI, as in this patient.

(Choice E) Ultrasound is appropriate for evaluating for testicular pathology (eg, torsion, rupture); however, lack of testicular tenderness in this patient suggests that the scrotal ecchymosis is due to nontesticular injury.

Educational objective:

Pelvic fractures in men are commonly complicated by posterior urethral injury. Patients with suspected urethral injury should undergo

the lower genitourinary tract obtained during the injection of radiopaque contrast into the urethra. A normal study demonstrates contrast entering the bladder uninterrupted. **Extravasation of contrast** from the urethra or inability of contrast to reach the bladder is diagnostic of urethral injury.

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Educational objective:
Pelvic fractures in men are commonly complicated by posterior urethral injury. Patients with suspected urethral injury should undergo retrograde urethrogram to evaluate for the diagnosis.

References

- Management of posterior urethral disruption injuries.
- Diagnosis and classification of urethral injuries.

An 80-year-old woman is brought to the emergency department from a nursing home due to progressive lethargy. The patient has a history of Alzheimer disease and at baseline is conversant and able to indicate her needs. For the past several days, she has been somnolent and lying in bed most of the time. She has had no fever, vomiting, or diarrhea. Two weeks ago, the patient was treated with ciprofloxacin for a urinary tract infection. Her other medical problems include hypertension, type 2 diabetes mellitus, and coronary artery disease. Blood pressure is 100/60 mm Hg, pulse is 100/min, and respirations are 20/min. Mucous membranes are dry. Lungs are clear to auscultation, and heart sounds are normal. The abdomen is soft and nontender. The patient has no skin rash. Laboratory results are as follows:

Complete blood count	
Hematocrit	44%
Leukocytes	8,200/mm^3
Serum chemistry	
Blood urea nitrogen	61 mg/dL
Creatinine	2.1 mg/dL
Glucose	140 mg/dL
Urinalysis	
Protein	trace
White blood cells	1-2 /hpf
Casts	none

A month ago, her serum creatinine was 0.9 mg/dL. Which of the following is the most likely cause of this patient's current renal

Urinalysis

Protein	trace
White blood cells	1-2 /hpf
Casts	none

A month ago, her serum creatinine was 0.9 mg/dL. Which of the following is the most likely cause of this patient's current renal abnormality?

A. Age-related renal functional decline
B. Atherosclerotic renovascular disease
C. Drug hypersensitivity reaction
D. Increased central venous pressure
E. Intravascular volume depletion
F. Nodular glomerulosclerosis
G. Obstructive uropathy
H. Suppression of antidiuretic hormone release
I. Toxin-mediated renal tubular damage

Submit

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Casts	none

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White blood cells	1-2 /hpf
Casts	none

A month ago, her serum creatinine was 0.9 mg/dL. Which of the following is the most likely cause of this patient's current renal abnormality?

- A. Age-related renal functional decline [1%]
- B. Atherosclerotic renovascular disease [1%]
- C. Drug hypersensitivity reaction [11%]
- D. Increased central venous pressure [0%]
- ✔ E. Intravascular volume depletion [72%]
- F. Nodular glomerulosclerosis [1%]
- G. Obstructive uropathy [2%]
- H. Suppression of antidiuretic hormone release [0%]
- I. Toxin-mediated renal tubular damage [8%]

Omitted
Correct answer
E

72%
Answered correctly

5 Seconds
Time Spent

12/28/2018
Last Updated

Explanation

Prerenal acute kidney injury	

Etiology	• Decreased renal perfusion
	  ◦ True volume depletion
	  ◦ Decreased EABV (eg, heart failure, cirrhosis)
	  ◦ Displacement of intravascular fluid (eg, sepsis, pancreatitis)
	  ◦ Renal artery stenosis
	  ◦ Afferent arteriole vasoconstriction (eg, NSAIDs)

Clinical features	• Increase in serum creatinine (eg, 50% from baseline) • Decreased urine output • Blood urea nitrogen/creatinine ratio >20:1 • Fractional excretion of sodium <1% • Unremarkable ("bland") urine sediment
Treatment	• Restoration of renal perfusion

EABV = effective arterial blood volume; **NSAIDs** = nonsteroidal anti-inflammatory drugs.

This patient with tachycardia, mild hypotension, and dry mucous membranes most likely has **intravascular volume depletion** leading to **prerenal acute kidney injury** (AKI). Elderly patients can be especially susceptible to volume depletion due to an impaired thirst response and often an inability to obtain food and water without assistance (eg, due to dementia).

Hypovolemia leads to decreased renal blood flow and causes activation of the renin-angiotensin-aldosterone system. Increased resorption of salt and water occurs, leading to an increase in the passive resorption of urea and resulting in an elevated **blood urea**

response and often an inability to obtain food and water without assistance (eg, due to dementia).

Hypovolemia leads to decreased renal blood flow and causes activation of the **renin-angiotensin-aldosterone system**. Increased resorption of salt and water occurs, leading to an increase in the passive resorption of urea and resulting in an elevated **blood urea nitrogen/creatinine ratio >20:1**. The term "injury" is somewhat inaccurate in the prerenal state as the kidneys do not experience actual injury unless decreased renal perfusion is prolonged (leading to acute tubular necrosis). Therefore, an **unremarkable urinalysis** (absence of significant protein, cells, or casts) is typical.

(Choice A) Age-related decline in renal function occurs due to loss of renal mass and thickening of the glomerular basement membrane. However, the process is gradual and cannot account for this patient's relatively abrupt decrease in renal function.

(Choice B) Atherosclerotic renovascular disease leads to renal artery stenosis. Significant bilateral disease is required to result in serum creatinine elevation and typically presents with hypertension rather than hypotension.

(Choice C) Drug hypersensitivity reaction can cause intrinsic AKI due to acute interstitial nephritis (AIN). Ciprofloxacin can cause AIN; however, absence of leukocyte casts on urinalysis and no skin rash make AIN unlikely.

(Choice D) Increased central venous pressure occurs in volume overload due to heart failure, which can cause prerenal AKI due to impaired forward blood flow. However, dry mucous membranes make volume overload unlikely.

(Choice F) Nodular glomerulosclerosis occurs in diabetic nephropathy; renal dysfunction typically develops gradually (rather than over 1 month), and significant proteinuria is expected.

(Choice G) Obstructive uropathy (postrenal AKI) is generally uncommon in women as benign prostatic hyperplasia is the most common cause.

(Choice H) Suppression of antidiuretic hormone occurs in the setting of low blood osmolality. However, antidiuretic hormone release will be stimulated in this patient due to **nonosmotic stimulation** from hypovolemia.

(Choice I) Aminoglycosides (eg, gentamicin) can cause intrinsic AKI due to renal tubular toxicity; however, ciprofloxacin is not associated with direct renal tubular damage.

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Educational objective:
The elderly can be especially susceptible to intravascular volume depletion leading to prerenal acute kidney injury. There is often a history of poor oral intake or excessive volume loss (eg, diarrhea). Laboratory results typically demonstrate a blood urea nitrogen/creatinine ratio >20:1 and an unremarkable urine sediment.

References

- Management of acute renal failure in the elderly patient: a clinician's guide.

Clinical features	• Blood urea nitrogen/creatinine ratio >20:1
Treatment	

EABV = effective art

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to **prerenal acute kidney injury** (AKI). El
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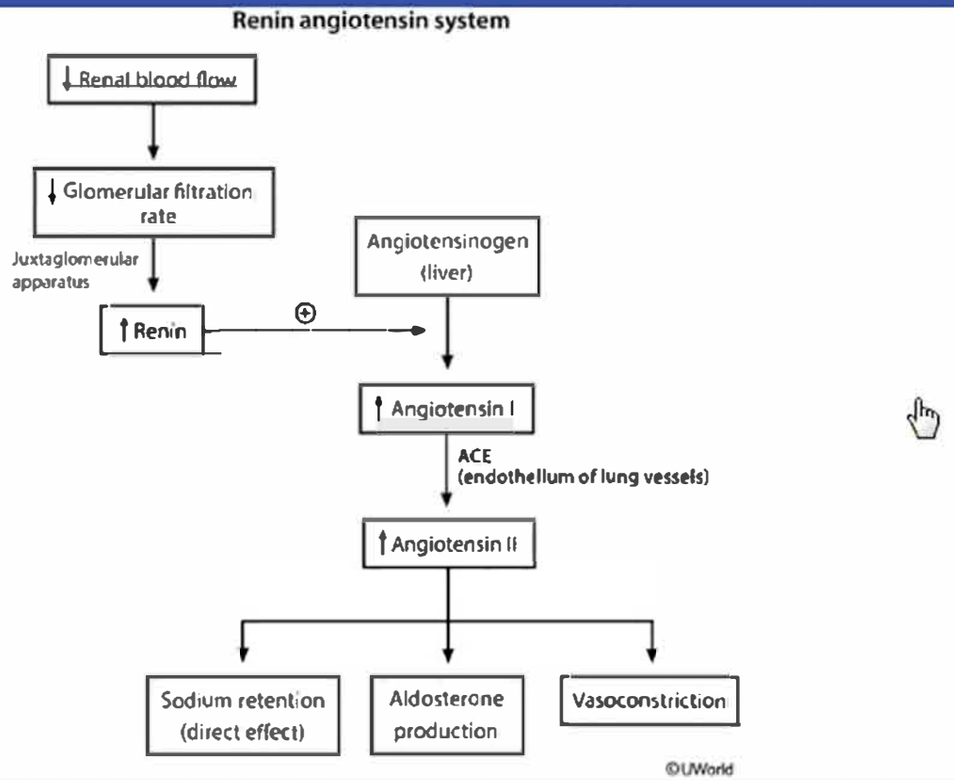

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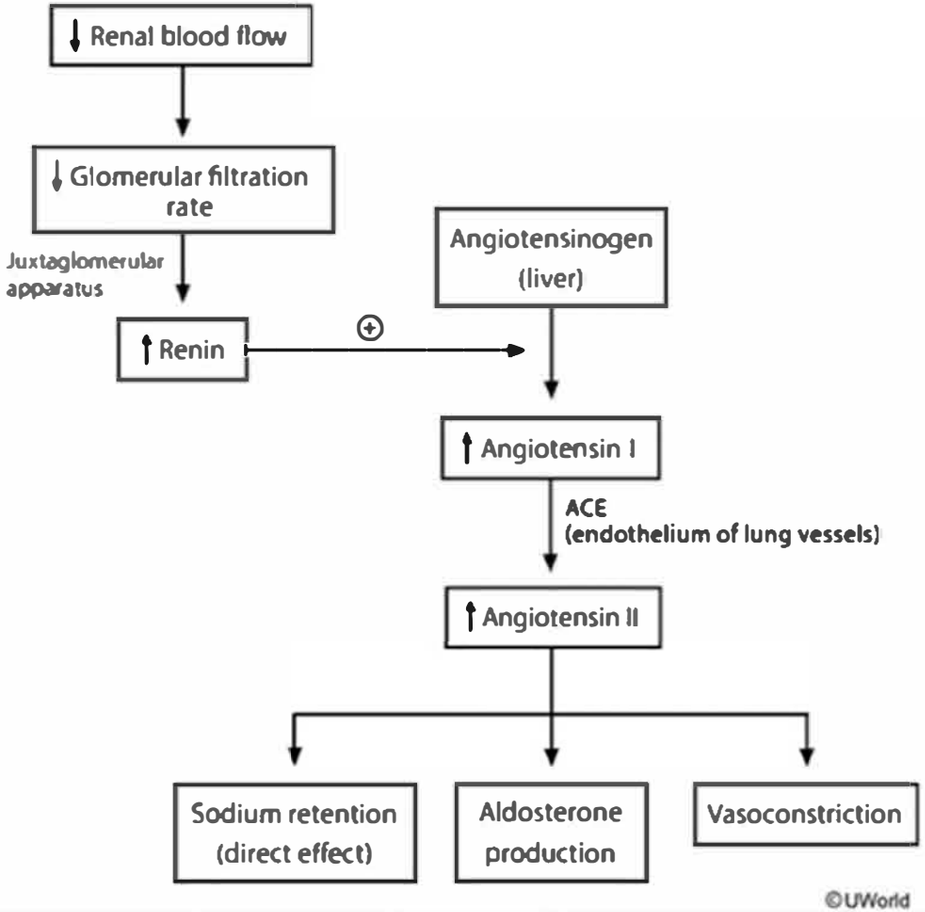

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References

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Stimuli for secretion of antidiuretic hormone	
Osmotic	• Serum osmolality > ~285 mOsm/kg H_2O
Nonosmotic	• Nausea • Pain • Physical or emotional stress • Hypotension • Hypovolemia • Hypoxia • Hypoglycemia

Add To Flash Card

A 4-week-old full-term boy is brought to the emergency department due to vomiting. His parents describe the emesis as undigested formula without blood or bile. The vomiting occurs after feeds and has increased in frequency and force over the past 6 days. However, the infant continues to bottle-feed every 1-2 hours. He previously had soft, yellow stools daily but has had no stool for the past 2 days. The patient was born at 39 weeks gestation through meconium-stained amniotic fluid with Apgar scores of 8 and 9. Temperature is 37.2 C (99 F), blood pressure is 70/30 mm Hg, pulse is 182/min, and respirations are 10/min; pulse oximetry is 98% on room air. Examination shows a thin, sleepy infant with a sunken anterior fontanelle and dry mucous membranes. Cardiac examination reveals tachycardia but no murmurs or gallops. The abdomen is soft, nontender, and nondistended. Which of the following laboratory findings would be expected in this patient?

	pH	$PaCO_2$	HCO_3	K	Cl
A.	↓	↓	↓	↓	↓
B.	↓	↑	↑	Normal	Normal
C.	Normal	Normal	Normal	Normal	Normal
D.	↑	↑	↑	↓	↓
E.	↑	↑	↑	↑	↑
F.	↑	↓	↓	↓	↓

Submit

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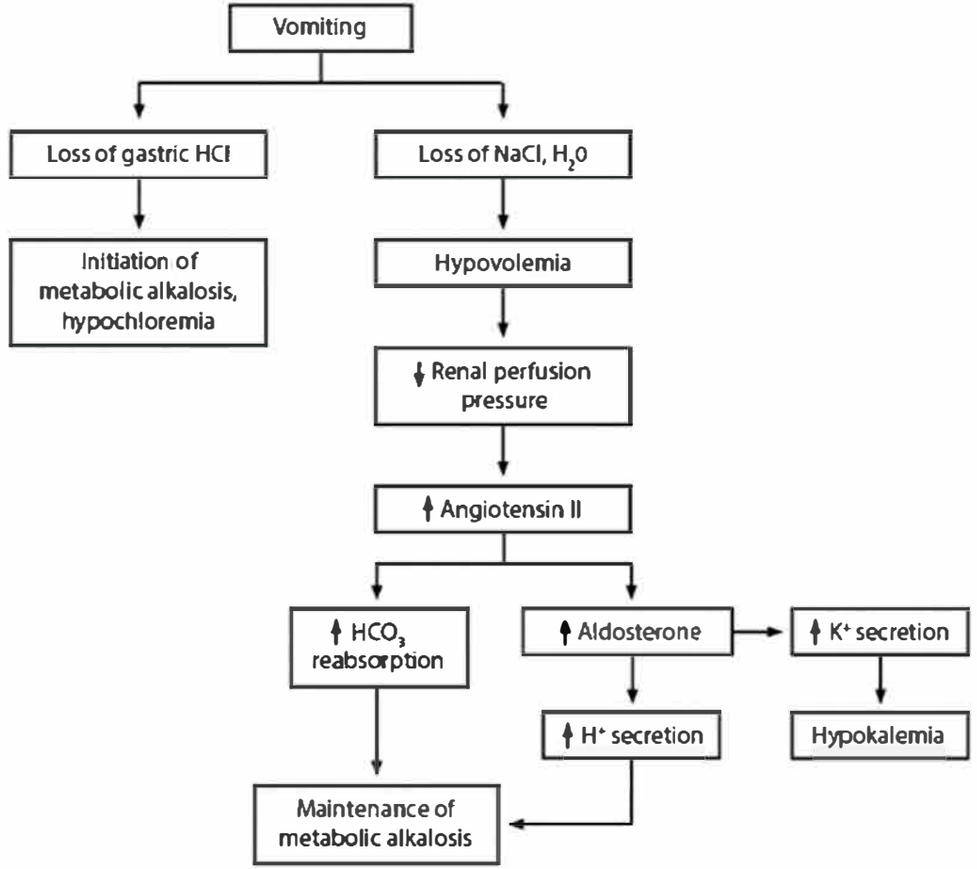
	pH	$PaCO_2$	HCO_3	K	Cl
A. [4%]	↓	↓	↓	↓	↓
B. [1%]	↓	↑	↑	Normal	Normal
C. [0%]	Normal	Normal	Normal	Normal	Normal
✔ D. [83%]	↑	↑	↑	↓	↓
E. [1%]	↑	↑	↑	↑	↑
F. [8%]	↑	↓	↓	↓	↓

Exhibit Display

Laboratory derangements in pyloric stenosis



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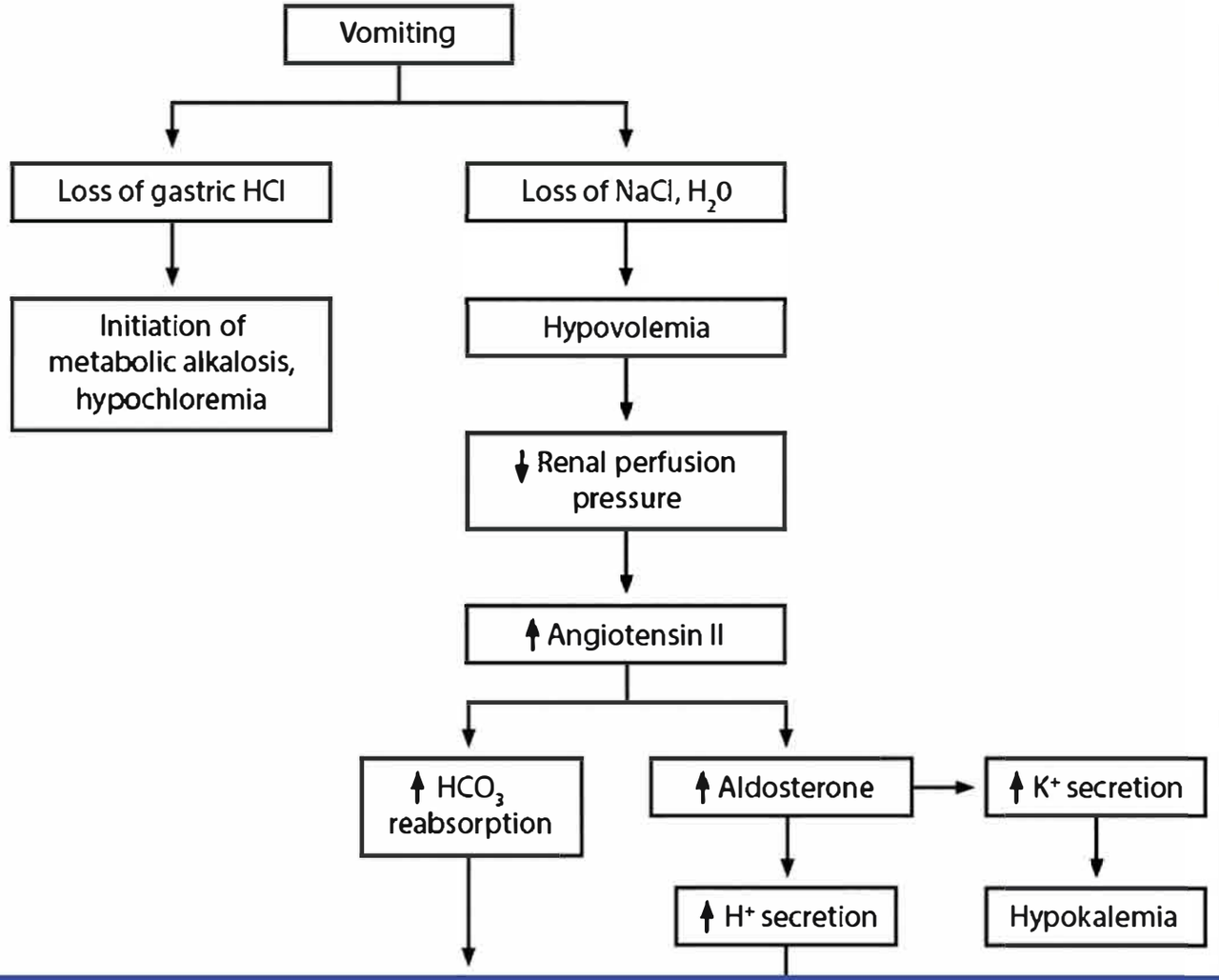
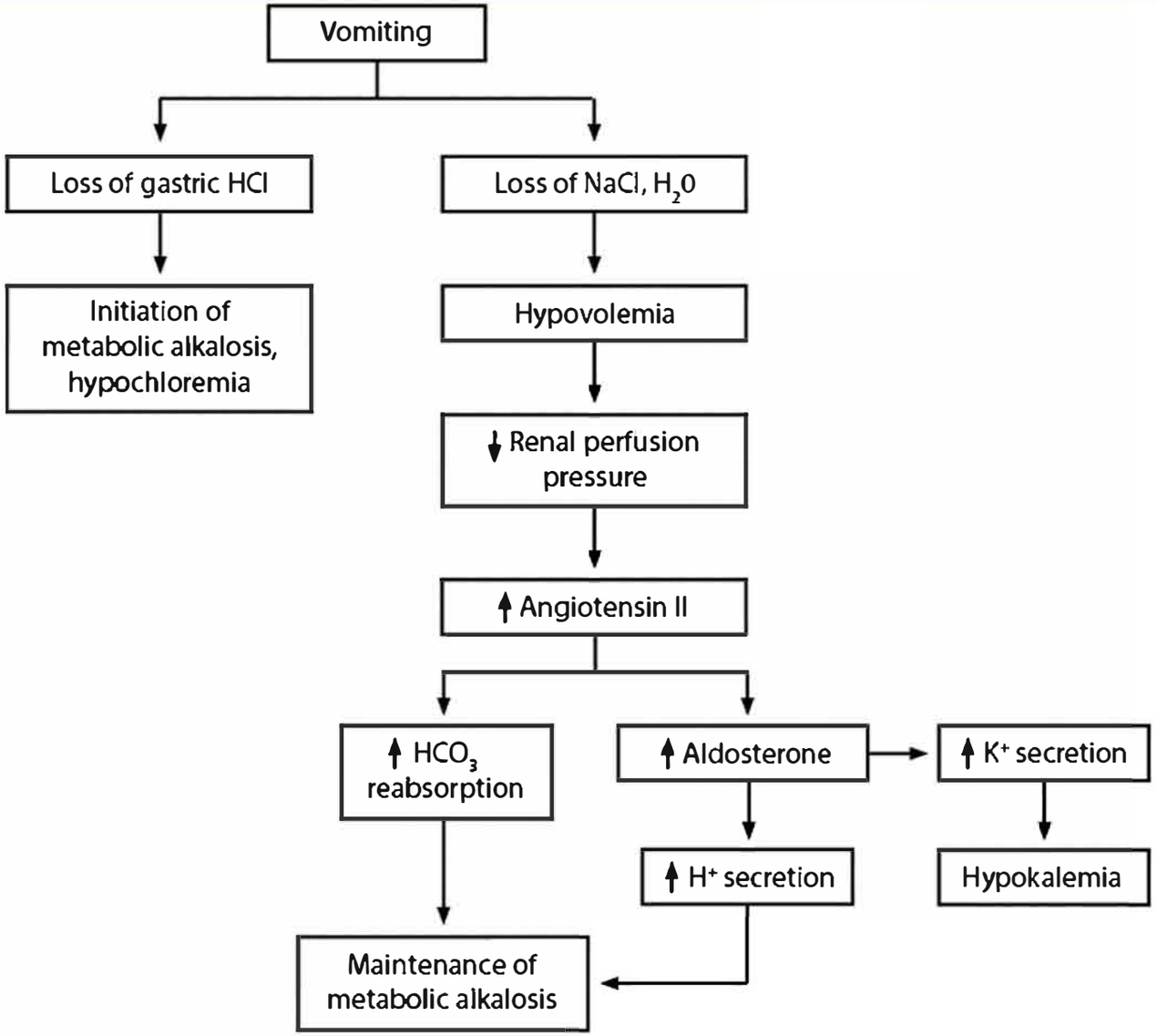
Laboratory derangements in pyloric stenosis





This infant has vomiting with feeds and signs of **dehydration** (ie, sunken fontanelle, dry mucous membranes) concerning for **infantile hypertrophic pyloric stenosis**. This condition is most common in first-born boys **age 3-5 weeks**. The **hypertrophied pylorus muscle** obstructs the gastric outlet, resulting in **nonbilious**, **projectile emesis**. Although many patients have a palpable, olive-shaped abdominal mass, its absence does not exclude the diagnosis.

If diagnosis is delayed, protracted vomiting can result in **hypochloremic**, **hypokalemic metabolic alkalosis** due to hydrochloric acid lost in the emesis. Hypovolemia also activates the renin-angiotensin-aldosterone system and potassium is excreted by the kidneys in response to aldosterone. The respiratory system responds to alkalosis with hypoventilation, which results in increased $PaCO_2$ and a compensatory respiratory acidosis.

A **thick, elongated pylorus** on abdominal ultrasonography is diagnostic, and **pyloromyotomy** is the treatment of choice.

(Choice A) Primary metabolic acidosis is characterized by decreased pH, $PaCO_2$, and bicarbonate. Metabolic acidosis can occur when vomiting is accompanied by diarrhea as significant amounts of bicarbonate are lost in the stool. Potassium and chloride are also lost in diarrhea.

(Choice B) Primary respiratory acidosis is characterized by decreased pH with elevated $PaCO_2$ and bicarbonate. This occurs in respiratory depression (eg, narcotic overdose) and hypoventilation syndromes (eg, neuromuscular disease). $PaCO_2$ retention in pyloric stenosis is compensatory to the primary metabolic alkalosis.

(Choice C) A normal acid-base status is characterized by pH of 7.35-7.45, $PaCO_2$ of 35-45 mm Hg, and bicarbonate of 22-26 mEq/L. Laboratory values may be normal initially in patients with pyloric stenosis but are unlikely in this patient with prolonged vomiting, dehydration, and abnormal vital signs.

(Choice E) Hyperkalemia and hyperchloremia are not seen in pyloric stenosis.

(Choice F) Primary respiratory alkalosis is characterized by increased pH with decreased $PaCO_2$ and bicarbonate. This is typically caused by hyperventilation and can be seen in patients with pain or anxiety

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(Choice F) Primary respiratory alkalosis is characterized by increased pH with decreased $PaCO_2$ and bicarbonate. This is typically caused by hyperventilation and can be seen in patients with pain or anxiety.

Educational objective:
Pyloric stenosis presents at age 3-5 weeks with nonbilious, projectile vomiting after each feed. Protracted vomiting produces a hypochloremic, hypokalemic metabolic alkalosis.

References

- PMID
- Title of Publication
- 22595707
- Pyloric stenosis in pediatric surgery: an evidence-based review.
- 23528507
- Electrolyte profile of pediatric patients with hypertrophic pyloric stenosis.
- 26944185
- Formula-feeding and hypertrophic pyloric stenosis: is there an association? A case-control study.

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(Choice C) A

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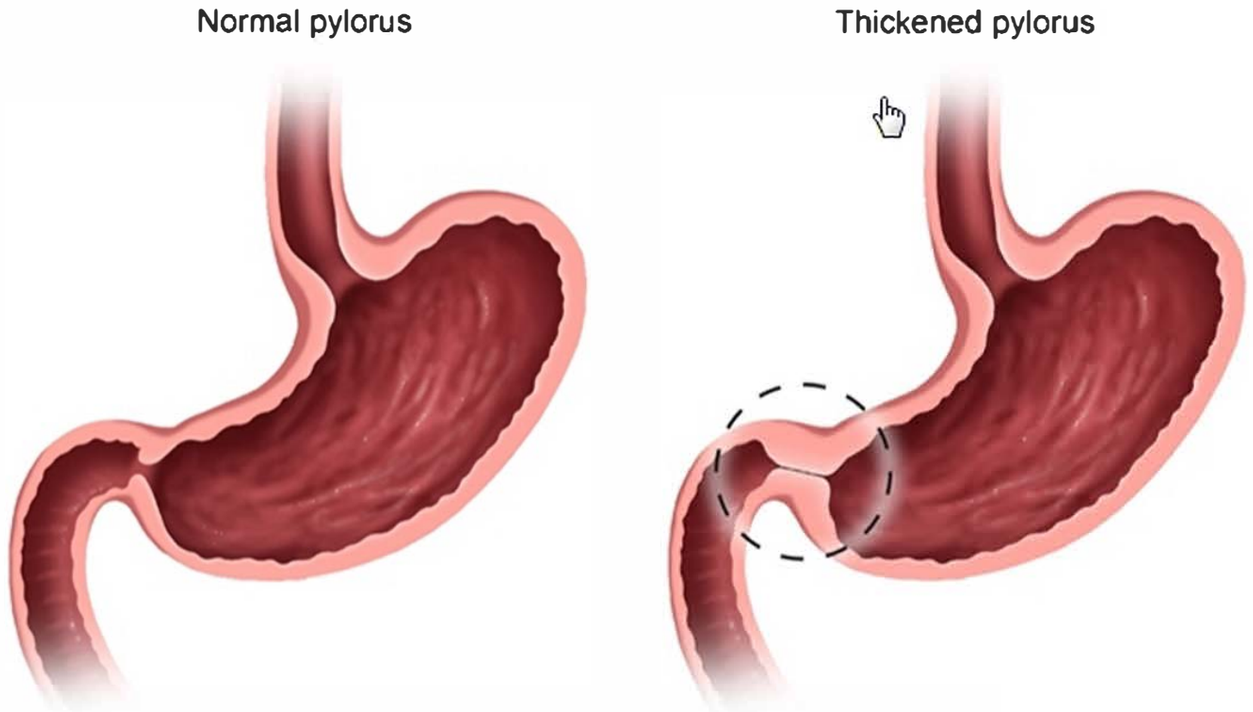
Hypertrophic pyloric stenosis



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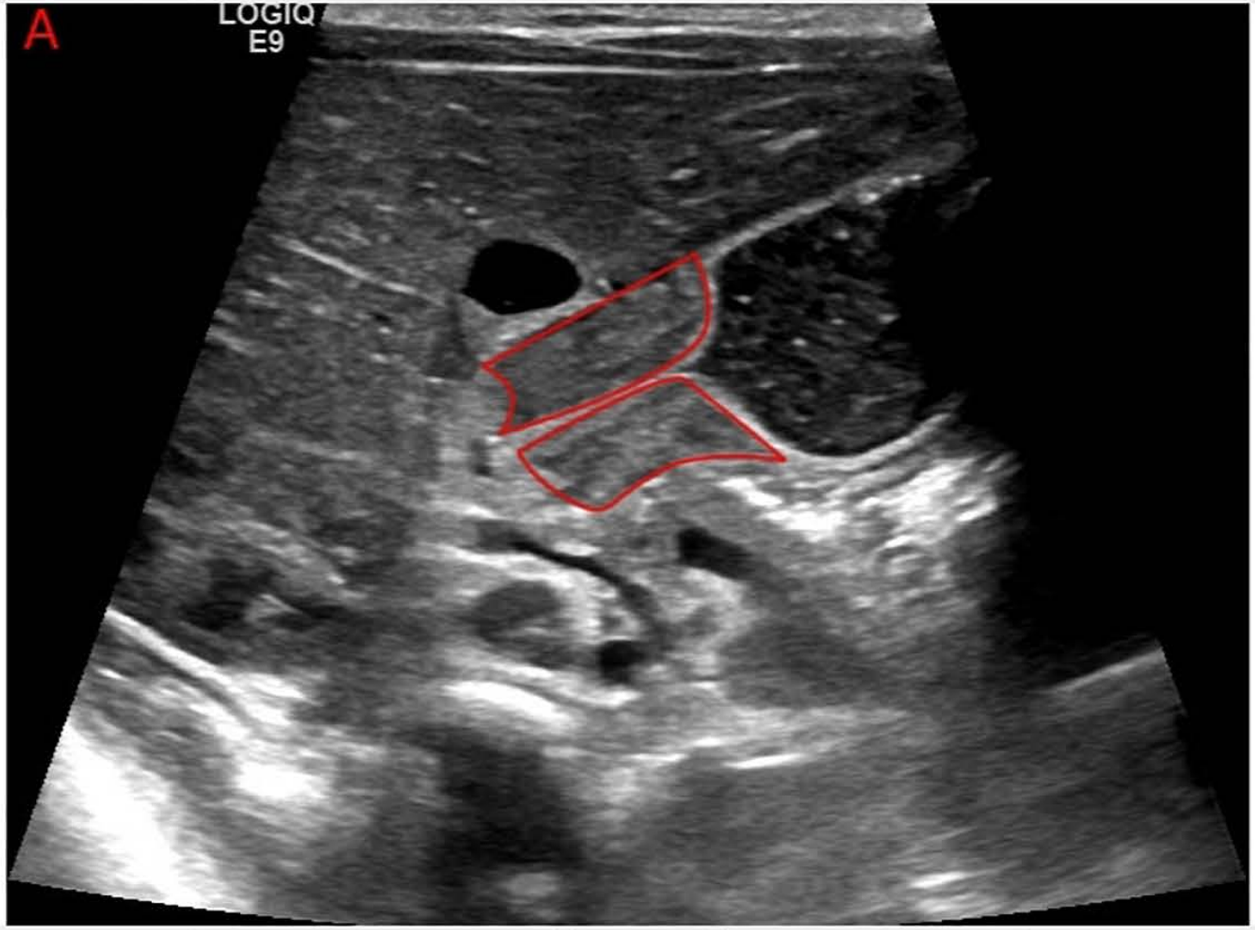

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A 45-year-old man comes to the emergency department complaining of dyspnea, fatigue, poor appetite, and weight gain over the past 2 weeks. His symptoms started with worsening shortness of breath with exertion. He has been waking up at night breathless for the past few days. He also finds it difficult to open his eyes in the morning due to facial edema. The patient has no other medical problems and takes no medications. His blood pressure is 200/120 mm Hg and pulse is 100/min. Physical examination shows anasarca with distended jugular veins while sitting upright. Lung auscultation shows bibasilar crackles. Urinalysis shows 1+ protein, no nitrite, trace leukocyte esterase, >50 red blood cells, red blood cell casts, and occasional neutrophils. Which of the following is the most likely mechanism of this patient's edema?

- A. Hyperthyroidism
- B. Hypoalbuminemia
- C. Portal hypertension
- D. Primary glomerular damage
- E. Renal hypoperfusion
- F. Right ventricular failure

Submit

A 45-year-old man comes to the emergency department complaining of dyspnea, fatigue, poor appetite, and weight gain over the past 2 weeks. His symptoms started with worsening shortness of breath with exertion. He has been waking up at night breathless for the past few days. He also finds it difficult to open his eyes in the morning due to facial edema. The patient has no other medical problems and takes no medications. His blood pressure is 200/120 mm Hg and pulse is 100/min. Physical examination shows anasarca with distended jugular veins while sitting upright. Lung auscultation shows bibasilar crackles. Urinalysis shows 1+ protein, no nitrite, trace leukocyte esterase, >50 red blood cells, red blood cell casts, and occasional neutrophils. Which of the following is the most likely mechanism of this patient's edema?

- A. Hyperthyroidism [0%]
- B. Hypoalbuminemia [17%]
- C. Portal hypertension [3%]
- ✔ D. Primary glomerular damage [52%]
- E. Renal hypoperfusion [10%]
- F. Right ventricular failure [16%]

Omitted
Correct answer
D

52%
Answered correctly

2 Seconds
Time Spent

08/14/2018
Last Updated

Explanation

Causes of peripheral edema	
Primary mechanism	**Clinical examples**
Increased capillary hydrostatic pressure	• Heart failure (left ventricular & cor pulmonale) • Primary renal sodium retention (renal disease & drugs) • Venous obstruction (eg, cirrhosis & venous insufficiency)
Decreased capillary oncotic pressure (hypoalbuminemia)	• Protein loss (eg, nephrotic syndrome & protein-losing enteropathy) • Decreased albumin synthesis (eg, cirrhosis & malnutrition)
Increased capillary permeability	• Burns, trauma & sepsis • Allergic reactions • Acute respiratory distress syndrome • Malignant ascites
Lymphatic obstruction/increased interstitial oncotic pressure	• Malignant ascites • Hypothyroidism • Lymph node dissection

This patient's presentation (anasarca, pulmonary and facial edema, hypertension, and abnormal urinalysis with proteinuria and microscopic hematuria) suggests acute nephritic syndrome with fluid overload. The table summarizes the primary mechanisms of the most common causes of peripheral edema. Acute nephritic syndrome is primary glomerular damage due to causes such as poststreptococcal glomerulonephritis, IgA nephropathy, lupus nephritis, membranoproliferative glomerulonephritis, and rapidly progressive glomerulonephritis.

Primary glomerular damage leads to decreased *glomerular filtration rate* with eventual development of significant volume overload (eg, pulmonary edema, distended neck veins, anasarca). Abnormal urinary sediment (red blood cells, red blood cell casts) and variable degrees of proteinuria are present on urinalysis. Serum creatinine can also be elevated. The increased volume also leads to hypertension. Significant proteinuria (3+) eventually leads to hypoalbuminemia, which further contributes to the edema. Decreased *glomerular filtration rate* is also the cause of edema in patients with end-stage renal disease.

(Choice A) Pretibial myxedema is a localized nonpitting thickening and induration of the skin over the lower legs, pretibial area, or the dorsum of the feet in patients with Graves' disease. However, anasarca and abnormal urinalysis are usually not seen in patients with hyperthyroidism.

(Choice B) Hypoalbuminemia is usually due to excessive albumin loss (eg, nephrotic syndrome, protein-losing enteropathy) or decreased albumin synthesis (eg, cirrhosis or severe malnutrition). It can cause significant peripheral edema but usually does not cause pulmonary edema. Alveolar capillaries have a higher permeability to albumin at baseline (reducing oncotic pressure difference) and greater lymphatic flow than skeletal muscle, protecting the lungs from edema.

(Choice C) Cirrhosis can cause portal hypertension as the scarred liver limits blood flow through the sinusoidal network. Cirrhotic patients develop ascites and edema in the lower extremities due to increased venous pressure below the liver. However, the venous pressure above the hepatic veins (eg, jugular venous pressure) is usually reduced or normal. Pulmonary edema typically does not occur in patients with cirrhosis.

(Choice E) Renal hypoperfusion can occur in heart failure (decreased cardiac output), which leads to increased renal sodium and

decreased albumin synthesis (eg, cirrhosis or severe malnutrition). It can cause significant peripheral edema but usually does not cause pulmonary edema. Alveolar capillaries have a higher permeability to albumin at baseline (reducing oncotic pressure difference) and greater lymphatic flow than skeletal muscle, protecting the lungs from edema.

(Choice C) Cirrhosis can cause portal hypertension as the scarred liver limits blood flow through the sinusoidal network. Cirrhotic patients develop ascites and edema in the lower extremities due to increased venous pressure below the liver. However, the venous pressure above the hepatic veins (eg, jugular venous pressure) is usually reduced or normal. Pulmonary edema typically does not occur in patients with cirrhosis.

(Choice E) Renal hypoperfusion can occur in heart failure (decreased cardiac output), which leads to increased renal sodium and water retention and edema. However, the urinalysis in patients with heart failure does not show red blood cells or casts, as seen in this patient.

(Choice F) Right ventricular failure is usually due to underlying left heart failure or severe pulmonary disease. It usually manifests with elevated jugular venous pressure and peripheral edema. Patients with severe cases can also develop hepatosplenomegaly, ascites, and anasarca. However, abnormal urinalysis with hematuria would not be expected in patients with right heart failure.

Educational objective:

Nephritic glomerulonephritis usually presents with urinary sediment containing red blood cells, occasional white blood cells, and red cell or mixed cellular casts. Edema in these patients is due primarily to decreased glomerular filtration rate and retention of sodium and water by the kidneys.

References

- Acute glomerulonephritis

A 66-year-old man comes to the office for a routine visit. He has no current complaints. The patient has a history of poorly controlled type 2 diabetes mellitus and hypertension. His blood work from 2 months ago showed hyperkalemia, and lisinopril was discontinued at that time. The patient's current medications include glipizide, furosemide, nifedipine, and aspirin. His blood pressure is 150/90 mm Hg and pulse is 78/min. Examination is unremarkable. Repeat blood test results today are as follows:

Sodium	136 mEq/L
Potassium	5.8 mEq/L
Chloride	108 mEq/L
Bicarbonate	18 mEq/L
Blood urea nitrogen	28 mg/dL
Creatinine	1.4 mg/dL

Which of the following is the most likely cause of this patient's electrolyte abnormalities?

A. Furosemide use
B. Laxative abuse
C. Pre-renal azotemia
D. Primary hyperaldosteronism
E. Renal artery stenosis
F. Renal tubular acidosis

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B. Laxative abuse
C. Pre-renal azotemia
D. Primary hyperaldosteronism
E. Renal artery stenosis
F. Renal tubular acidosis
G. Surreptitious vomiting

Submit

A 66-year-old man comes to the office for a routine visit. He has no current complaints. The patient has a history of poorly controlled type 2 diabetes mellitus and hypertension. His blood work from 2 months ago showed hyperkalemia, and lisinopril was discontinued at that time. The patient's current medications include glipizide, furosemide, nifedipine, and aspirin. His blood pressure is 150/90 mm Hg and pulse is 78/min. Examination is unremarkable. Repeat blood test results today are as follows:

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Which of the following is the most likely cause of this patient's electrolyte abnormalities?

A. Furosemide use [12%]
B. Laxative abuse [4%]
C. Pre-renal azotemia [13%]
D. Primary hyperaldosteronism [6%]
E. Renal artery stenosis [10%]
F. Renal tubular acidosis [51%]

Sodium	136 mEq/L
Potassium	5.8 mEq/L
Chloride	108 mEq/L
Bicarbonate	18 mEq/L
Blood urea nitrogen	28 mg/dL
Creatinine	1.4 mg/dL

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- B. Laxative abuse [4%]
- C. Pre-renal azotemia [13%]
- D. Primary hyperaldosteronism [6%]
- E. Renal artery stenosis [10%]
- ✔ F. Renal tubular acidosis [51%]
- G. Surreptitious vomiting [0%]

Omitted
Correct answer
F

51%
Answered correctly

6 Seconds
Time Spent

10/10/2018
Last Updated

Explanation

Causes of normal anion gap metabolic acidosis

- Diarrhea
- Fistulas (eg, pancreatic, ileocutaneous, etc.)
- Carbonic anhydrase inhibitors
- Renal tubular acidosis
- Ureteral diversion (e.g., ileal loop)
- Iatrogenic

This patient has a metabolic acidosis (given his low serum bicarbonate of 18 mEq/L) with hyperkalemia. He has a **non-anion gap metabolic acidosis** with a serum anion gap of 10 mEq/L (anion gap = serum Na^+ - [serum Cl^- + serum HCO_3^-]; normal anion gap = 10-14 mEq/L). He also has a mild degree of renal dysfunction consistent with chronic kidney disease. When there is a gradual loss of nephrons, the remaining nephrons maintain the kidney's ability to excrete a daily acid load by producing more NH_3 buffer that removes H^+ as NH_4^+ in the urine. As a result, metabolic acidosis in chronic kidney disease is rarely seen until there is advanced renal dysfunction (glomerular filtration rate <20 mL/min). Non-anion gap metabolic acidosis and **hyperkalemia that occur out of proportion to the renal dysfunction** indicate a renal tubular disorder.

Renal tubular acidosis (RTA) is a group of disorders characterized by **non-anion gap metabolic acidosis** in the presence of **preserved kidney function**. The cortical collecting tubule is the site for H^+ and K^+ excretion, which is regulated by aldosterone. Impaired function of the cortical collecting tubule due to aldosterone deficiency or resistance will cause retention of H^+ and K^+ and is termed **hyperkalemic RTA** (or **type 4 RTA**). Hyperkalemic RTA is commonly seen in elderly patients who have **poorly controlled diabetes** with damage to the juxtaglomerular apparatus, which causes a state of hyporeninemic hypoaldosteronism. These patients

Impaired function of the cortical collecting tubule due to aldosterone deficiency or resistance will cause retention of H^+ and K^+ and is termed **hyperkalemic RTA** (or **type 4 RTA**). Hyperkalemic RTA is commonly seen in elderly patients who have **poorly controlled diabetes** with damage to the juxtaglomerular apparatus, which causes a state of hyporeninemic hypoaldosteronism. These patients have mild hyperkalemia (K^+ 5.2-6.0 mEq/L) and mild acidosis (HCO_3^- 15-20 mEq/L) on a background of preserved or moderately diminished glomerular filtration rate (20-50 mL/min). Hypertension may or may not be present and is usually due to the accompanying atherosclerosis.

(Choice A) Loop diuretic use causes hypokalemia and metabolic alkalosis.

(Choice B) Although diarrhea most commonly causes metabolic acidosis, diarrhea from laxative abuse often causes severe hypokalemia and metabolic alkalosis. The severe hypokalemia occurs due to potassium losses in the stool and increased aldosterone secretion due to volume depletion. This leads to impaired chloride reabsorption and bicarbonate excretion, resulting in metabolic alkalosis.

(Choice C) In pre-renal azotemia (renal hypoperfusion) the blood urea nitrogen/creatinine ratio is usually more than 20:1 due to increased urea absorption. Renal hypoperfusion is due to either systemic hypovolemia or impaired cardiac output. This patient has no symptoms or signs of volume depletion.

(Choice D) In primary hyperaldosteronism, high aldosterone levels cause increased H^+ and K^+ excretion, leading to hypokalemia and metabolic alkalosis (mnemonic: **A**ldosterone **S**aves **S**odium and **P**ushes **P**otassium out).

(Choice E) Renal artery stenosis causes secondary hyperaldosteronism due to low perfusion to the kidney. Hypokalemia, metabolic alkalosis, and hypertension are characteristic findings.

(Choice G) Surreptitious vomiting causes a hypochloremic metabolic alkalosis due to loss of gastric HCl. Hypokalemia may be seen due to increased aldosterone secretion caused by volume depletion.

Educational objective:

Type 4 renal tubular acidosis (hyperkalemic renal tubular acidosis) is characterized by non-anion gap metabolic acidosis, persistent

(Choice A) Loop diuretic use causes hypokalemia and metabolic alkalosis.

(Choice B) Although diarrhea most commonly causes metabolic acidosis, diarrhea from laxative abuse often causes severe hypokalemia and metabolic alkalosis. The severe hypokalemia occurs due to potassium losses in the stool and increased aldosterone secretion due to volume depletion. This leads to impaired chloride reabsorption and bicarbonate excretion, resulting in metabolic alkalosis.

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(Choice G) Surreptitious vomiting causes a hypochloremic metabolic alkalosis due to loss of gastric HCl. Hypokalemia may be seen due to increased aldosterone secretion caused by volume depletion.

Educational objective:
Type 4 renal tubular acidosis (hyperkalemic renal tubular acidosis) is characterized by non-anion gap metabolic acidosis, persistent hyperkalemia, and mild-to-moderate renal insufficiency. It commonly occurs in patients with poorly controlled diabetes.

References

- Mechanisms in hyperkalemic renal tubular acidosis.

A 32-year-old Caucasian male presents to the ER with a 12-hour history of anorexia and vomiting. He says that he feels 'a little dizzy'. He denies abdominal pain or diarrhea. His past medical history is insignificant. His blood pressure is 110/70 mmHg while supine and 100/60 mmHg while sitting. His heart rate is 90/min. His laboratory values are:

Serum sodium	139 mEq/L
Serum potassium	3.1 mEq/L
Serum calcium	8.9 mEq/L
Serum chloride	88 mEq/L
Serum bicarbonate	33 mEq/L
Blood glucose	95 mg/dL
BUN	20 mg/dL
Serum creatinine	1.1 mg/dL

Which of the following is the most likely cause of the decreased chloride level in this patient?

Serum bicarbonate	33 mEq/L
Blood glucose	95 mg/dL
BUN	20 mg/dL
Serum creatinine	1.1 mg/dL

Which of the following is the most likely cause of the decreased chloride level in this patient?

- A. Gastrointestinal loss
- B. Bicarbonate reabsorption in the kidney
- C. Metabolic alkalosis
- D. Intracellular shift
- E. Volume depletion
- F. Hypokalemia

Submit

	mEq/L
Blood glucose	95 mg/dL
BUN	20 mg/dL
Serum creatinine	1.1 mg/dL

Which of the following is the most likely cause of the decreased chloride level in this patient?

- A. Gastrointestinal loss [74%]
- B. Bicarbonate reabsorption in the kidney [6%]
- C. Metabolic alkalosis [8%]
- D. Intracellular shift [3%]
- E. Volume depletion [4%]
- F. Hypokalemia [1%]

Omitted
Correct answer
A

74%
Answered correctly

5 Seconds
Time Spent

07/11/2018
Last Updated

Blood glucose	mg/dL
BUN	20 mg/dL
Serum creatinine	1.1 mg/dL

Which of the following is the most likely cause of the decreased chloride level in this patient?

- ✔ A. Gastrointestinal loss [74%]
- B. Bicarbonate reabsorption in the kidney [6%]
- C. Metabolic alkalosis [8%]
- D. Intracellular shift [3%]
- E. Volume depletion [4%]
- F. Hypokalemia [1%]

Omitted
Correct answer
A

74%
Answered correctly

5 Seconds
Time Spent

07/11/2018
Last Updated

Explanation

Explanation

The chemistry values obtained for this patient demonstrate hypochloremia, hypokalemia, and an elevated bicarbonate level. These findings are all consistent with gastrointestinal losses due to vomiting **(Choice A)**. Gastrointestinal fluid is rich in both hydrogen chloride and potassium chloride. Therefore, any process by which excessive gastric contents are lost (vomiting, nasogastric suctioning), may result in loss of hydrogen, chloride, and potassium ions. The results include hypochloremia and hypokalemia, as seen in this patient. Furthermore, the loss of hydrogen ions increases the concentration of bicarbonate. An arterial blood gas would be expected to confirm metabolic alkalosis in this patient by demonstrating a pH>7.4 in addition to the elevated bicarbonate.

(Choice B & E) For each hydrogen chloride ion lost due to vomiting, a bicarbonate ion is produced. In addition, the volume contraction caused by vomiting leads to activation of the renin-angiotensin system which stimulates bicarbonate reabsorption. It is important to realize that while volume contraction and bicarbonate reabsorption are increased in this patient, they are the result of hydrogen chloride loss; not the cause of it. These answer choices reverse cause and effect.

(Choice C) This answer choice again reverses cause and effect. In this patient, vomiting has caused significant loss of hydrogen chloride, resulting in metabolic alkalosis. Metabolic alkalosis is the consequence - not the cause - of vomiting.

(Choice D & F) Hypokalemia accompanies, but does not cause this patient's hypochloremia. It is the result of gastrointestinal loss of potassium, the intracellular shift of potassium caused by alkalosis, and increased renal excretion of potassium caused by aldosterone. The aldosterone is ramped up by the renin-angiotensin system as a means to retain fluid.

Educational objective:
Gastric contents are rich in hydrogen, chloride, and potassium. Therefore vomiting causes hypochloremic metabolic alkalosis and hypokalemia. Bicarbonate levels rise as a result of hydrogen loss and activation of the renin-angiotensin system. The administration of isotonic sodium chloride and potassium is used to reverse these electrolyte abnormalities.

A 56-year-old man develops oliguria three days after having a kidney transplantation. His postoperative course was uncomplicated. His blood pressure is 160/100 mm Hg and heart rate is 90/min. Palpation of the transplant reveals mild tenderness. Laboratory studies show:

Serum sodium	145 mEq/L
Serum potassium	5.5 mEq/L
Serum calcium	8.6 mg/dL
Serum creatinine	3.2 mg/dL
BUN	30 mg/dL

His serum cyclosporine level is normal. Renal ultrasonography does not detect dilatation of the calyces. Biopsy of the transplant shows heavy lymphocyte infiltration and vascular involvement with swelling of the intima. Which of the following is the most appropriate next step in management?

A. Decrease the dose of cyclosporine
B. Give IV steroids
C. Order ureterography
D. Administer IV diuretics
E. Prepare for surgery

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- A. Decrease the dose of cyclosporine [2%]
- ✔ B. Give IV steroids [77%]
- C. Order ureterography [1%]
- D. Administer IV diuretics [2%]
- E. Prepare for surgery [15%]

Omitted
Correct answer
B

77%
Answered correctly

5 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Renal transplant dysfunction in the early post-operative period manifests as oliguria, hypertension, and increased creatinine/BUN. It can be explained by a number of causes, which include ureteral obstruction, acute rejection, cyclosporine toxicity, vascular obstruction, acute tubular necrosis, etc.

In this case, the patient's clinical signs, symptoms, and laboratory findings are suggestive of acute rejection. Rapid institution of anti-rejection therapy, including high-dose IV steroids, is important.

(Choice A) Cyclosporine toxicity does not present with graft tenderness. The transplant's function is usually restored when the dose of cyclosporine is decreased. In this case, the patient's clinical features (i.e., normal serum cyclosporine level, renal biopsy findings) are more consistent with acute rejection.

(Choice C) Renal ultrasound helps to rule out ureteral obstruction.

(Choice D) Administration of IV diuretics may be employed in acute tubular necrosis, but the patient's renal biopsy findings are not consistent with this condition.

(Choice E) Surgery is not appropriate in this case.

Educational Objective:
Renal transplant dysfunction in the early post-operative period can be explained by a variety of causes, including ureteral obstruction, acute rejection, cyclosporine toxicity, vascular obstruction, and acute tubular necrosis. Radioisotope scanning, renal ultrasound, MRI,

Explanation

Renal transplant dysfunction in the early post-operative period manifests as oliguria, hypertension, and increased creatinine/BUN. It can be explained by a number of causes, which include ureteral obstruction, acute rejection, cyclosporine toxicity, vascular obstruction, acute tubular necrosis, etc.

In this case, the patient's clinical signs, symptoms, and laboratory findings are suggestive of acute rejection. Rapid institution of anti-rejection therapy, including high-dose IV steroids, is important.

(Choice A) Cyclosporine toxicity does not present with graft tenderness. The transplant's function is usually restored when the dose of cyclosporine is decreased. In this case, the patient's clinical features (i.e., normal serum cyclosporine level, renal biopsy findings) are more consistent with acute rejection.

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(Choice D) Administration of IV diuretics may be employed in acute tubular necrosis, but the patient's renal biopsy findings are not consistent with this condition.

(Choice E) Surgery is not appropriate in this case.

Educational Objective:
Renal transplant dysfunction in the early post-operative period can be explained by a variety of causes, including ureteral obstruction, acute rejection, cyclosporine toxicity, vascular obstruction, and acute tubular necrosis. Radioisotope scanning, renal ultrasound, MRI, and renal biopsy can be employed in conducting a differential diagnosis. Acute rejection is best treated with intravenous steroids.

*Extremely high yield question for the USMLE!!!

performing one of her routine 3-hour workouts. She is not doing well academically in college despite persistent attempts to improve her grades. Her blood pressure is 102/58 mm Hg and pulse is 113/min. Her body mass index is 21 kg/m². Fine hair is noted on her scalp. Examination shows dental erosions with slightly enlarged cheeks. There is no edema. Laboratory results are as follows:

Sodium	132 mEq/L
Potassium	2.4 mEq/L
Calcium	10.1 mg/dL
Chloride	90 mEq/L
Bicarbonate	40 mEq/L
Urine chloride	14 mEq/L

In addition to potassium supplementation, which of the following is the best treatment to correct the laboratory abnormalities in this patient?

A. Acetazolamide
B. Breathing in a paper bag
C. Calcium gluconate infusion
D. Hyperventilation
E. Insulin therapy
F. Loop diuretics
G. Normal saline infusion

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Calcium	10.1 mg/dL
Chloride	90 mEq/L
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- C. Calcium gluconate infusion
- D. Hyperventilation
- E. Insulin therapy
- F. Loop diuretics
- G. Normal saline infusion
- H. Sodium bicarbonate infusion

Submit

A 21-year-old woman comes to the physician with progressive weakness and loss of energy. She nearly collapsed yesterday while performing one of her routine 3-hour workouts. She is not doing well academically in college despite persistent attempts to improve her grades. Her blood pressure is 102/58 mm Hg and pulse is 113/min. Her body mass index is 21 kg/m². Fine hair is noted on her scalp. Examination shows dental erosions with slightly enlarged cheeks. There is no edema. Laboratory results are as follows:

Sodium	132 mEq/L
Potassium	2.4 mEq/L
Calcium	10.1 mg/dL
Chloride	90 mEq/L
Bicarbonate	40 mEq/L
Urine chloride	14 mEq/L

In addition to potassium supplementation, which of the following is the best treatment to correct the laboratory abnormalities in this patient?

A. Acetazolamide [12%]
B. Breathing in a paper bag [7%]
C. Calcium gluconate infusion [3%]
D. Hyperventilation [1%]
E. Insulin therapy [1%]
F. Loop diuretics [0%]

Chloride	90 mEq/L
Bicarbonate	40 mEq/L
Urine chloride	14 mEq/L

In addition to potassium supplementation, which of the following is the best treatment to correct the laboratory abnormalities in this patient?

- A. Acetazolamide [12%]
- B. Breathing in a paper bag [7%]
- C. Calcium gluconate infusion [3%]
- D. Hyperventilation [1%]
- E. Insulin therapy [1%]
- F. Loop diuretics [0%]
- ✔ G. Normal saline infusion [71%]
- H. Sodium bicarbonate infusion [1%]

Omitted
Correct answer
G

71%
Answered correctly

5 Seconds
Time Spent

08/20/2018
Last Updated

Explanation

Clinical features of metabolic alkalosis	
Causes	**Saline-responsive** • Vomiting • Gastric suctioning • Diuretics • Laxative abuse • Decreased oral fluid intake (volume depletion) **Saline-resistant** • Primary hyperaldosteronism • Cushing's syndrome • Severe hypokalemia (<2 mEq/L)
Clinical presentation	• Volume depletion: Easy fatigability, postural dizziness, muscle cramps • Hypokalemia: Muscle weakness, arrhythmias • Urine chloride: <20 mEq/L (saline-responsive), >20 mEq/L (saline-resistant)
Treatment	• Treat underlying cause to reverse generation phase in all cases • Saline-responsive: Also give normal saline to

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Treatment	• Treat underlying cause to reverse generation phase in all cases • Saline-responsive: Also give normal saline to correct maintenance phase

This patient's presentation (age, exercise, fine hair, dental erosions) is consistent with likely bulimia nervosa. Bulimia is more common in younger women and leads to binge eating followed by induced vomiting. The body mass index tends to be normal or slightly low. Patients can present with hypotension, tachycardia, dry skin, hair loss, parotid gland hypertrophy with puffy cheeks, and dental erosions due to periodic induced vomiting. The chronic vomiting can lead to volume depletion causing metabolic alkalosis (MA), hypokalemia, and hypochloremia.

MA is due to an underlying disorder producing excess bicarbonate (generation phase) combined with a process preventing renal bicarbonate excretion (maintenance phase). MA is further classified as saline responsive or resistant. The generation phase in saline-responsive MA can be due to urinary or gastrointestinal hydrogen loss (eg, diuretics, vomiting) or decreased oral intake. The maintenance phase (eg, hypovolemia) prevents the normal kidney from excreting the excess bicarbonate in the urine. The kidneys perceive a decreased effective arterial blood volume from the underlying etiology (eg, vomiting with volume depletion) and increase renin and aldosterone release. This leads to sodium reabsorption, potassium excretion, and hydrogen excretion. There is also decreased bicarbonate excretion.

If the history cannot provide the etiology, urine chloride can differentiate between saline-resistant and saline-responsive MA. Saline-resistant MA has excess mineralocorticoid causing hydrogen and potassium loss and increased sodium retention leading to increased extracellular volume. The kidneys respond by excreting both sodium and chloride to result in high urine chloride. Saline-responsive MA has low urine chloride (<20 mEq/L) due to hypovolemia and hypochloremia. Treatment for both types focuses on the underlying cause to reverse generation and increase renal bicarbonate excretion. In saline-responsive MA, normal saline administration restores arterial volume, corrects hypochloremia, and increases urinary bicarbonate excretion.

(Choice A) Acetazolamide is a diuretic that works by inhibiting carbonic anhydrase, which leads to decreased reabsorption of bicarbonate, sodium, and chloride. This may improve her alkalosis somewhat; however, this will worsen her volume depletion. Additionally, acetazolamide causes hypokalemia, an undesirable side effect in this woman who already has a low serum potassium concentration. It can be considered in edematous patients with metabolic alkalosis.

(Choices B and D) Hyperventilation decreases pCO_2 and causes respiratory alkalosis. Breathing into a bag increases pCO_2 and

cause to reverse generation and increase renal bicarbonate excretion. In saline-responsive MA, normal saline administration restores arterial volume, corrects hypochloremia, and increases urinary bicarbonate excretion.

(Choice A) Acetazolamide is a diuretic that works by inhibiting carbonic anhydrase, which leads to decreased reabsorption of bicarbonate, sodium, and chloride. This may improve her alkalosis somewhat; however, this will worsen her volume depletion. Additionally, acetazolamide causes hypokalemia, an undesirable side effect in this woman who already has a low serum potassium concentration. It can be considered in edematous patients with metabolic alkalosis.

(Choices B and D) Hyperventilation decreases pCO_2 and causes respiratory alkalosis. Breathing into a bag increases pCO_2 and decreases pH slightly, but it does not reverse the underlying metabolic alkalosis.

(Choices C and E) Calcium gluconate and insulin can be used as acute therapy for **hyperkalemia**. This patient has hypokalemia.

(Choice F) Loop diuretics increase distal sodium delivery and the secretion of aldosterone, which would further worsen metabolic alkalosis, hypokalemia and volume depletion.

(Choice H) Sodium bicarbonate infusion would not be needed in a patient who already has excess serum bicarbonate. It is used primarily in those with metabolic acidosis, hyperkalemia, and renal failure.

Educational objective:
The pathogenesis of metabolic alkalosis requires generating excess bicarbonate (generation phase) and preventing excretion of excess bicarbonate (maintenance phase). Treatment involves addressing the underlying cause and promoting urinary bicarbonate excretion.

References

- Rational treatment of acid-base disorders.

A 3-year-old boy is brought to the physician with a 1-week history of generalized edema that is gradually worsening. He has not been ill recently and his past medical history is unremarkable. He is not taking any medications and his vaccinations are up to date. His temperature is 36.7 C (98 F), blood pressure is 110/80 mm Hg, pulse is 85/min, and respirations are 18/min. On examination, he has periorbital edema and 1+ pretibial pitting. The remainder of the physical examination is normal. Laboratory findings are as follows:

Sodium	140 mEq/L
Potassium	3.7 mEq/L
Creatinine	0.8 mg/dL
Total protein	5.5 g/dL
Albumin	2.1 g/dL
Aspartate aminotransferase	17 U/L
Alanine aminotransferase	24 U/L
Total bilirubin	0.9 mg/dLM
Direct bilirubin	0.3 mg/dL

Urinalysis shows 4+ proteinuria and no red blood cells. What is the best next step in the management of this patient?

A. Echocardiography
B. Intravenous albumin
C. Liver ultrasound

Creatinine	0.8 mg/dL
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- A. Echocardiography
- B. Intravenous albumin
- C. Liver ultrasound
- D. Prednisone
- E. Renal biopsy
- F. Renal scintigraphy

Submit

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Urinalysis shows 4+ proteinuria and no red blood cells. What is the best next step in the management of this patient?

A. Echocardiography [0%]
B. Intravenous albumin [4%]
C. Liver ultrasound [1%]

Total protein	5.5 g/dL
Albumin	2.1 g/dL
Aspartate aminotransferase	17 U/L
Alanine aminotransferase	24 U/L
Total bilirubin	0.9 mg/dLM
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- A. Echocardiography [0%]
- B. Intravenous albumin [4%]
- C. Liver ultrasound [1%]
- ✔ D. Prednisone [74%]
- E. Renal biopsy [16%]
- F. Renal scintigraphy [3%]

Omitted
Correct answer
D

74%
Answered correctly

4 Seconds
Time Spent

08/22/2018
Last Updated

Explanation

Minimal change disease	
Epidemiology	• Most common cause of nephrotic syndrome in children • Median age 2–3; 85% of cases occur before 10 years of age
Pathogenesis	• T-cell mediated injury to podocytes causes increased molecular permeability to albumin • Majority of cases are idiopathic
Clinical features	• Edema • Fatigue • No hematuria
Diagnosis	• Proteinuria • Hypoalbuminemia • Renal biopsy without microscopic changes
Treatment	Corticosteroids

Minimal change disease (MCD) is the **most common cause** of nephrotic syndrome in children age <10 (accounting for approximately 80% cases). In general, affected children have significant **edema** and **proteinuria**. The edema may be periorbital in the morning but becomes more pronounced in the legs and genitals later in the day. Confirmation of nephrotic-range proteinuria is generally accomplished with a 24-hour urine collection, although a random level can be used in the appropriate clinical situation, such as in this case.

MCD is a highly steroid-sensitive condition, and the diagnosis of MCD is based on age, clinical presentation, and response to steroids. Empiric **steroid therapy** should be initiated upon suspicion of the diagnosis. Approximately 85% of children will respond to their first steroid course.

(Choice A) Echocardiography would be the test of choice to evaluate for the presence of heart failure, which can present with signs of volume overload and fatigue. However, this boy has no tachycardia, abnormal heart sounds (eg, gallop), or congestive hepatomegaly to suggest heart failure.

(Choice B) Hypoalbuminemia is common in children with nephrotic syndrome (as seen in this patient). However, the inherent problem is increased glomerular permeability to albumin; infusing additional albumin will not correct it and may contribute to volume overload instead.

(Choice C) Liver ultrasound would be warranted if hepatic failure was suspected to be the cause of this child's hypoalbuminemia. However, the normal liver function tests and bilirubin make hepatic dysfunction unlikely.

(Choice E) Renal biopsy is indicated in children age >10 with nephrotic syndrome, or in any child with nephritic syndrome or minimal change disease that is unresponsive to steroids. Biopsy is invasive and unlikely to change management when MCD is the most likely diagnosis.

(Choice F) Renal scintigraphy is used to evaluate renal function and is useful in the setting of kidney dysfunction. However, this patient has normal creatinine and urine output, suggesting no renal insufficiency. Scintigraphy would not be beneficial.

Educational objective:

(Choice A) Echocardiography would be the test of choice to evaluate for the presence of heart failure, which can present with signs of volume overload and fatigue. However, this boy has no tachycardia, abnormal heart sounds (eg, gallop), or congestive hepatomegaly to suggest heart failure.

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Educational objective:
Minimal change disease is the most common cause of nephrotic syndrome in young children. Renal biopsy is not required for initial diagnosis as the condition is highly responsive to steroids.

References

- Minimal change nephrotic syndrome in children: new aspects on pathogenesis and treatment
- The nephrotic syndrome

A 34-year-old sexually active female comes to your office because of urinary frequency and dysuria for two days. She has had two such episodes in the past, each treated with oral antibiotics. Physical examination reveals suprapubic tenderness and her urinalysis is positive for nitrite, leukocyte esterase, many WBC, and a moderate amount of bacteria. Which of the following is the most common reason for the higher incidence of urinary tract infections in females than in males?

- A. Closer proximity of the urethral meatus to the anus in females
- B. Frequent use of spermicide and diaphragms in females
- C. Shorter urethral length in females
- D. Higher post-void urine residual in females
- E. Hormonal fluctuation of females

Submit

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- A. Closer proximity of the urethral meatus to the anus in females [7%]
- B. Frequent use of spermicide and diaphragms in females [0%]
- ✔ C. Shorter urethral length in females [90%]
- D. Higher post-void urine residual in females [0%]
- E. Hormonal fluctuation of females [0%]

Omitted
Correct answer
C

90%
Answered correctly

2 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Urinary tract infections (UTI) are more commonly seen in females than males, and half of all adult women have a UTI at some time in their life. The high incidence of UTIs in women is primarily due to the shorter length of the female urethra. After the periurethral area becomes colonized by rectal flora, the bacteria ascend to the bladder to cause infection. This is facilitated in females by a short urethra. Predisposing factors for UTIs include alteration of the normal vaginal flora by recent antibiotic use, sexual intercourse,

Explanation

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Males, on the other hand, are less likely to develop a UTI mainly because they have a longer urethra than females. They also have a drier periurethral environment and antibacterial substances in prostatic fluid, which also help to fight off infection.

(Choice A) A shorter distance between the anus and urethra in women is associated with a higher incidence of recurrent UTIs within the female population, but is not the main reason for the difference in UTI frequency between men and women.

(Choice B) Spermicide and diaphragm use are risk factors for UTIs in females. A shorter urethral length in females, however, is responsible for their higher frequency of UTIs compared to males.

(Choice D) Increased post-void urine residual is not associated with a higher incidence of recurrent UTIs in females.

(Choice E) Female hormonal fluctuation is not related to an increased incidence of UTIs in females.

Educational objective:
The increased incidence of urinary tract infections in women is due to the shorter length of the female urethra compared to males. Other predisposing factors for UTIs include sexual intercourse, recent antibiotic use, the use of spermicidal contraceptives, and a close proximity of the urethra to the anus.

A 50-year-old man comes to the physician for a routine check up. He has no present complaints. He has diabetes mellitus, type 2, stable angina, and gout. He takes glyburide and atenolol. He smokes two packs a day and, occasionally, consumes alcohol. His father had an early myocardial infarction; his brother has diabetes mellitus. His blood pressure is 140/90 mm Hg and heart rate is 65/min. Physical examination shows no abnormalities. There is concern about end organ damage in this patient due to diabetes mellitus. Which of the following is the earliest renal abnormality to be present in this patient?

- A. Nodular sclerosis
- B. Glomerular basement membrane thickening
- C. Mesangial expansion
- D. Immune deposits
- E. Glomerular hyperfiltration

Submit

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- A. Nodular sclerosis [12%]
- B. Glomerular basement membrane thickening [22%]
- C. Mesangial expansion [10%]
- D. Immune deposits [1%]
- ✔ E. Glomerular hyperfiltration [53%]

Omitted
Correct answer
E

53%
Answered correctly

2 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

Glomerular hyperfiltration is believed to be the earliest renal abnormality present in patients with diabetes mellitus. It can be detected as early as several days after the diagnosis of diabetes was made. Moreover, glomerular hyperfiltration is the major pathophysiologic mechanism of glomerular injury in these patients. It creates intraglomerular hypertension leading to progressive glomerular damage

✔ E. Glomerular hyperfiltration [53%]

Omitted
Correct answer
E

53%
Answered correctly

2 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

Glomerular hyperfiltration is believed to be the earliest renal abnormality present in patients with diabetes mellitus. It can be detected as early as several days after the diagnosis of diabetes was made. Moreover, glomerular hyperfiltration is the major pathophysiologic mechanism of glomerular injury in these patients. It creates intraglomerular hypertension leading to progressive glomerular damage and renal function loss. You should remember that effectiveness of ACE inhibitors in diabetic nephropathy is related to their ability to *reduce intraglomerular hypertension* and, thereby, decrease glomerular damage.

(Choices A, B and C) Thickening of the glomerular basement membrane (GBM) is the first change that can be quantitated. This is followed by mesangial expansion. Nodular sclerosis is superimposed later and is specific for diabetic nephropathy.

(Choice D) Immune deposits are not characteristic for diabetic nephropathy.

Educational objective:
Glomerular hyperfiltration is the earliest renal abnormality seen in diabetic nephropathy. It is also the major pathophysiologic mechanism of glomerular injury in these patients. *Thickening of the glomerular basement* membrane is the first change that can be quantitated.

A 70-year-old man is brought to the hospital by his son because of worsening fatigue. His son states that his father does not like seeing doctors and has not seen a physician in the past 20 years. He has no medical problems. Physical examination of the prostate shows no abnormalities. Laboratory studies show:

Hb	10.5 g/dL
WBC	7,400/cmm
Platelets	160,000/cmm
Serum Na	135 mEq/L
Serum K	5.0 mEq/L
BUN	50 mg/dL
Serum creatinine	3.0 mg/dL

Ultrasonogram of the abdomen shows bilateral small kidneys and no evidence of hydronephrosis. Kidney biopsy shows intimal thickening and luminal narrowing of renal arterioles with evidence of sclerosis. Which of the following is the most likely cause of this patient's findings?

- A. Hypertension
- B. Diabetes mellitus
- C. Multiple myeloma
- D. Analgesic abuse

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- A. Hypertension [73%]
- B. Diabetes mellitus [18%]
- C. Multiple myeloma [3%]
- D. Analgesic abuse [3%]

Omitted
Correct answer
A

73%
Answered correctly

6 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

This patient most likely has benign nephrosclerosis secondary to hypertension. Hypertension is the second leading cause of end stage renal disease in the United States. It is inter-related with kidney disease; hypertension causes nephropathy, and vice versa. In particular, it is the renal vasculature that is exquisitely sensitive to damages incurred by systemic hypertension. Arteriosclerotic lesions of afferent and efferent renal arterioles and glomerular capillary tufts are the most common renal vascular lesions seen. As the hypertension progresses, there is a progressive decrease in renal blood flow and glomerular filtration rate. The sequence of kidney damage evolves from nephrosclerosis to glomerulosclerosis. Nephrosclerosis is characterized by hypertrophy and intimal medial fibrosis of renal arterioles; whereas, glomerulosclerosis is characterized by progressive loss of the glomerular capillary surface area with glomerular and peritubular fibrosis. Microscopic hematuria and proteinuria occur due to these glomerular lesions. The kidneys generally decrease in size.

(Choice B) Diabetic nephropathy is the leading cause of end stage renal disease in the United States. Increased extracellular matrix, basement membrane thickening, mesangial expansion, and fibrosis characterize diabetes mellitus nephropathy. The sequence of pathological changes in the kidneys of a patient with diabetes mellitus is as follows:

1. Within the first year of diabetes mellitus - Glomerular hyperperfusion and renal hypertrophy with increase in glomerular filtration rate.
2. First five years of diabetes mellitus - Glomerular basement membrane thickening, glomerular hypertrophy, and mesangial volume expansion with glomerular filtration rate returning to normal.
3. Within 5-10 years of diabetes mellitus - Microalbuminuria, which later progresses to overt nephropathy.

(Choice C) Some degree of renal insufficiency is seen in approximately 50% of patients with multiple myeloma along the course of

lesions of afferent and efferent renal arterioles and glomerular capillary tufts are the most common renal vascular lesions seen. As the hypertension progresses, there is a progressive decrease in renal blood flow and glomerular filtration rate. The sequence of kidney damage evolves from nephrosclerosis to glomerulosclerosis. Nephrosclerosis is characterized by hypertrophy and intimal medial fibrosis of renal arterioles; whereas, glomerulosclerosis is characterized by progressive loss of the glomerular capillary surface area with glomerular and peritubular fibrosis. Microscopic hematuria and proteinuria occur due to these glomerular lesions. The kidneys generally decrease in size.

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(Choice C) Some degree of renal insufficiency is seen in approximately 50% of patients with multiple myeloma along the course of their disease. This is due to obstruction of the distal and collecting tubules by large laminated casts consisting mainly of Bence-Jones proteins. Amyloid deposition and infiltration of the kidney by plasma cells are commonly seen.

(Choice D) Analgesic abuse nephropathy is primarily a tubulointerstitial disease characterized by focal glomerulosclerosis.

Educational Objective:
The most common renal vascular lesions seen in hypertension are arteriosclerotic lesions of afferent and efferent renal arterioles and glomerular capillary tufts. Diabetes mellitus nephropathy is characterized by increased extracellular matrix, basement membrane thickening, mesangial expansion, and fibrosis.

A 55-year-old man is evaluated for puffiness of the face and abdominal distension over the past 4 weeks. He has hypertension and type 2 diabetes mellitus. The patient does not use alcohol, tobacco, or illicit drugs. His temperature is 37.1 C (98.8 F), blood pressure is 133/75 mm Hg, pulse is 82/min, and respirations are 14/min. He also has periorbital edema, moderate ascites, and 1+ bilateral pitting pedal edema.

Serum chemistry	
Creatinine	2.2 mg/dL
Liver function studies	
Total protein	4.5 g/dL
Albumin	2.3 g/dL
Urinalysis	
Protein	+4
White blood cells	5-10/hpf
Red blood cells	0-1/hpf

Twenty-four-hour urinary protein excretion is 5 g/day. This patient is at increased risk for developing which of the following?

A. Accelerated atherosclerosis
B. Cobalamin deficiency anemia
C. Hypoparathyroidism

Protein	+4
White blood cells	5-10/hpf
Red blood cells	0-1/hpf

Twenty-four-hour urinary protein excretion is 5 g/day. This patient is at increased risk for developing which of the following?

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B. Cobalamin deficiency anemia
C. Hypoparathyroidism
D. Iron overload
E. Monoclonal gammopathy

Submit

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Red blood cells	0-1/hpf

Twenty-four-hour urinary protein excretion is 5 g/day. This patient is at increased risk for developing which of the following?

- A. Accelerated atherosclerosis [68%]
- B. Cobalamin deficiency anemia [6%]
- C. Hypoparathyroidism [8%]

Liver function studies

Total protein	4.5 g/dL
Albumin	2.3 g/dL

Urinalysis

Protein	+4
White blood cells	5-10/hpf
Red blood cells	0-1/hpf

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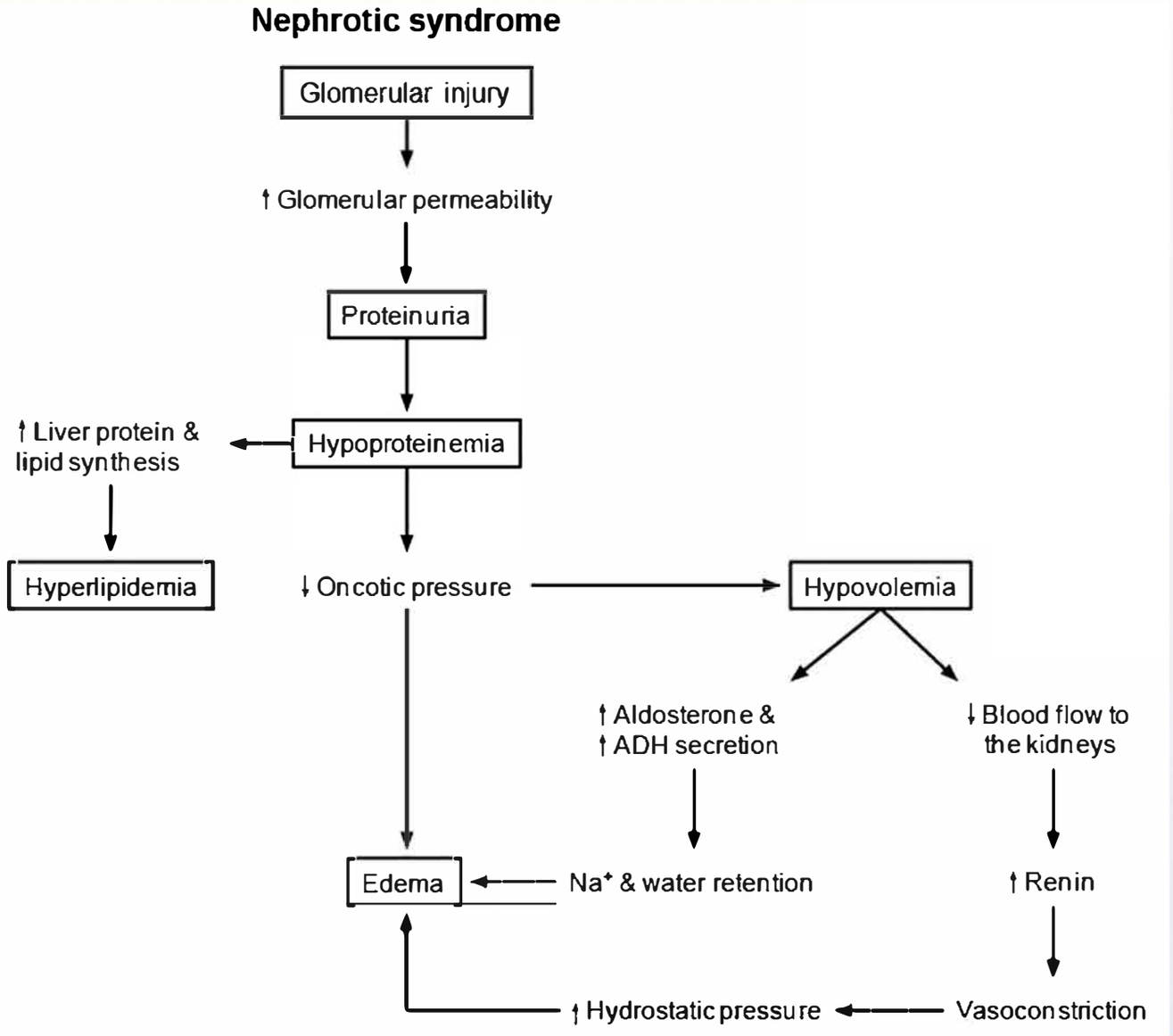
- A. Accelerated atherosclerosis [68%]
- B. Cobalamin deficiency anemia [6%]
- C. Hypoparathyroidism [8%]
- D. Iron overload [3%]
- E. Monoclonal gammopathy [13%]

Omitted
Correct answer
A

68%
Answered correctly

4 Seconds
Time Spent

10/28/2018
Last Updated


Nephrotic syndrome
Glomerular injury
↑ Glomerular permeability
Proteinuria
Hypoproteinemia
↑ Liver protein & lipid synthesis
Hyperlipidemia
↓ Oncotic pressure
Hypovolemia
↑ Aldosterone & ↑ ADH secretion
↓ Blood flow to the kidneys
Edema
Na+ & water retention
↑ Renin
↑ Hydrostatic pressure
Vasoconstriction

This patient with peripheral edema, hypoalbuminemia, and urinary protein excretion of >3.5 g/day meets all of the major criteria for **nephrotic syndrome**. Although some patients with nephrotic syndrome may develop acute renal failure, a normal creatinine level does not rule out the diagnosis. Low plasma oncotic pressure (due to hypoalbuminemia) increases hepatic lipoprotein synthesis. The result is an increase in serum cholesterol and triglyceride levels, which increases the risk for **atherosclerotic** disease, particularly in individuals with comorbid conditions for atherosclerosis (eg, hypertension, diabetes).

Patients with nephrotic syndrome are also hypercoagulable, possibly due to loss of antithrombin III in urine. This **hypercoagulability** tends to affect the venous more than the arterial system, particularly the renal veins. Atherosclerosis and hypercoagulability put affected patients at increased risk for stroke or myocardial infarction. Aggressive management with statins or other medications to lower cholesterol levels is indicated.

(Choices B and D) Significant cobalamin (vitamin B_{12}) deficiency occurs most commonly due to intestinal malabsorption (eg, pernicious anemia) or in those with dietary restrictions (eg, vegans). Anemia in nephrotic syndrome is more likely due to a combination of erythropoietin and iron deficiencies. Iron overload, as seen in hemochromatosis, is not generally associated with nephrotic syndrome.

(Choice C) Patients with nephrotic syndrome can lose vitamin D into the urine, leading to a compensatory increase in parathyroid hormone. Hypoparathyroidism is not typically seen with nephrotic syndrome.

(Choice E) Monoclonal gammopathy can lead to amyloidosis, which can be a cause of nephrotic syndrome. However, nephrotic syndrome itself does not lead to monoclonal gammopathy.

Educational objective:
Patients with nephrotic syndrome have increased risk for atherosclerosis (due to hyperlipidemia) and arteriovenous thrombosis (due to loss of antithrombin III). Atherosclerosis and hypercoagulability increase the risk for stroke and myocardial infarction.

A 66-year-old man comes to the clinic for a follow-up for hypertension. Two months ago, he had high blood pressure (165/95 mm Hg) during a routine office visit, and since then has had 2 follow-up visits, both documenting high blood pressure. The patient states that he currently has no symptoms and has never been diagnosed with hypertension, but he does have type 2 diabetes mellitus and hyperlipidemia. He underwent a stent placement for peripheral vascular disease 2 years ago. The patient is a former cigarette smoker with a 25-pack-year history. Currently, his blood pressure is 162/93 mm Hg, and his heart rate is 73/min and regular. His BMI is 31 kg/m^2, and physical examination is unremarkable. Serum creatinine is 0.8 mg/dL. CT angiography reveals 80% atherosclerotic narrowing of the right renal artery. In addition to antihyperlipidemic therapy, which of the following is the best next step in management of this patient?

- A. Doxazosin
- B. Lisinopril
- C. Loop diuretic
- D. Renal artery stenting
- E. Surgical revascularization

Submit

A 66-year-old man comes to the clinic for a follow-up for hypertension. Two months ago, he had high blood pressure (165/95 mm Hg) during a routine office visit, and since then has had 2 follow-up visits, both documenting high blood pressure. The patient states that he currently has no symptoms and has never been diagnosed with hypertension, but he does have type 2 diabetes mellitus and hyperlipidemia. He underwent a stent placement for peripheral vascular disease 2 years ago. The patient is a former cigarette smoker with a 25-pack-year history. Currently, his blood pressure is 162/93 mm Hg, and his heart rate is 73/min and regular. His BMI is 31 kg/m^2, and physical examination is unremarkable. Serum creatinine is 0.8 mg/dL. CT angiography reveals 80% atherosclerotic narrowing of the right renal artery. In addition to antihyperlipidemic therapy, which of the following is the best next step in management of this patient?

- A. Doxazosin [2%]
- ✔ B. Lisinopril [48%]
- C. Loop diuretic [2%]
- D. Renal artery stenting [41%]
- E. Surgical revascularization [4%]

Omitted
Correct answer
B

48%
Answered correctly

3 Seconds
Time Spent

10/26/2018
Last Updated

Explanation

Explanation

Renal artery stenosis (RAS) is a common finding in older patients, with a high prevalence in those with severe hypertension or peripheral arterial disease. Most patients with hypertension have essential hypertension. However, **renovascular hypertension** is the most common correctable cause of secondary hypertension and should be suspected in all patients with resistant hypertension and diffuse atherosclerosis.

Patients with RAS and renovascular hypertension should be managed with aggressive risk factor reduction (aspirin, optimal diabetes and hyperlipidemia control, smoking cessation) to prevent cardiovascular disease. Patients with hypertension should be managed initially with **angiotensin-converting enzyme inhibitors** (ACEIs) or **angiotensin II receptor blockers** (ARBs). Additional antihypertensive therapy should be instituted as needed for optimal blood pressure control.

RAS causes decreased renal blood flow (RBF) and activation of the renin-angiotensin system, resulting in hypertension. ACEI therapy reduces angiotensin II levels, dilating the glomerular efferent arterioles. With **unilateral** RAS, the stenotic kidney experiences reduced RBF and a resultant fall in glomerular filtration rate (GFR). However, the unaffected kidney compensates for this fall in GFR as it is no longer subject to angiotensin II-induced renal vasoconstriction. With bilateral RAS, the fall in GFR generally leads to a rise in serum creatinine (acceptable rise is <30%); in this setting, ACEIs are sometimes contraindicated but can still be used with close renal function monitoring due to their long-term nephroprotective effects.

(Choices A and C) Several antihypertensive agents are used in conjunction with ACEIs or ARBs for adequate blood pressure control. These include calcium channel blockers (eg, amlodipine, felodipine, diltiazem), thiazide diuretics (eg, chlorthalidone), beta blockers (eg, metoprolol, atenolol, labetalol), and mineralocorticoid receptor antagonists (eg, spironolactone). However, doxazosin or loop diuretics are not recommended as a first-line agent for hypertension.

(Choices D and E) Revascularization (surgical or percutaneous angioplasty with stenting) has not been proven superior to medical therapy for optimal blood pressure control or reduction of cardiovascular outcomes in patients with unilateral or bilateral RAS. It is reserved for selected patients who are intolerant or fail to achieve adequate blood pressure control with optimal medical therapy and

therapy reduces angiotensin II levels, dilating the glomerular efferent arterioles. With **unilateral** RAS, the stenotic kidney experiences reduced RBF and a resultant fall in glomerular filtration rate (GFR). However, the unaffected kidney compensates for this fall in GFR as it is no longer subject to angiotensin II-induced renal vasoconstriction. With bilateral RAS, the fall in GFR generally leads to a rise in serum creatinine (acceptable rise is <30%); in this setting, ACEIs are sometimes contraindicated but can still be used with close renal function monitoring due to their long-term nephroprotective effects.

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(Choices D and E) Revascularization (surgical or percutaneous angioplasty with stenting) has not been proven superior to medical therapy for optimal blood pressure control or reduction of cardiovascular outcomes in patients with unilateral or bilateral RAS. It is reserved for selected patients who are intolerant or fail to achieve adequate blood pressure control with optimal medical therapy and for those with recurrent flash pulmonary edema and/or refractory heart failure due to severe hypertension.

Educational objective:
Angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers are indicated for initial therapy in patients with hypertension and renal artery stenosis. Renal artery stenting or surgical revascularization is reserved for patients with resistant hypertension or recurrent flash pulmonary edema and/or refractory heart failure due to severe hypertension.

References

- Clinical practice. Renal-artery stenosis.
- Stenting and medical therapy for atherosclerotic renal-artery stenosis.
- Balloon angioplasty, with and without stenting, versus medical therapy for hypertensive patients with renal artery stenosis.

Explanation

Renal artery s
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Patients with R
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Exhibit Display

Clinical clues to renovascular disease	
HTN-related symptoms	• Resistant HTN (uncontrolled despite 3-drug regimen) • Malignant HTN (with end-organ damage) • Onset of severe HTN (>180/120 mm Hg) after age 55 • Severe HTN with diffuse atherosclerosis • Recurrent flash pulmonary edema with severe HTN
Supportive evidence	**Physical examination** • Asymmetric renal size (>1.5 cm) • Abdominal bruit **Laboratory results** • Unexplained rise in serum creatinine (>30%) after starting ACE inhibitors or ARBs **Imaging results** • Unexplained atrophic kidney

ARBs = angiotensin II receptor blockers; **HTN** = hypertension.

Add To Flash Card

A 55-year-old man comes to the office for follow-up for hypertension. He was diagnosed with hypertension 5 years ago and since then has been treated with benazepril and amlodipine. His blood pressure has been consistently high over the last 6 months despite dietary modifications to decrease salt intake. Medical history is significant for hypercholesterolemia. The patient does not smoke or consume alcohol. Blood pressure is 152/94 mm Hg and pulse is 80/min. BMI is 30 kg/m^2 with increased waist-to-hip ratio. Adding chlorthalidone to his current therapy is considered for improving control of hypertension. Which of the following metabolic effects may be expected as a result of this therapy?

- A. Decreased LDL cholesterol
- B. Decreased plasma triglycerides
- C. Hyperglycemia
- D. Hyperkalemia
- E. Hypermagnesemia
- F. Hypocalcemia

Submit

A 55-year-old man comes to the office for follow-up for hypertension. He was diagnosed with hypertension 5 years ago and since then has been treated with benazepril and amlodipine. His blood pressure has been consistently high over the last 6 months despite dietary modifications to decrease salt intake. Medical history is significant for hypercholesterolemia. The patient does not smoke or consume alcohol. Blood pressure is 152/94 mm Hg and pulse is 80/min. BMI is 30 kg/m² with increased waist-to-hip ratio. Adding chlorthalidone to his current therapy is considered for improving control of hypertension. Which of the following metabolic effects may be expected as a result of this therapy?

- A. Decreased LDL cholesterol [3%]
- B. Decreased plasma triglycerides [4%]
- ✔ C. Hyperglycemia [53%]
- D. Hyperkalemia [17%]
- E. Hypermagnesemia [8%]
- F. Hypocalcemia [13%]

Omitted
Correct answer
C

53%
Answered correctly

2 Seconds
Time Spent

11/17/2018
Last Updated

Explanation

Thiazide diuretics (eg, chlorthalidone, hydrochlorothiazide) impair both insulin release from the pancreas and glucose utilization in

Explanation

Thiazide diuretics (eg, chlorthalidone, hydrochlorothiazide) impair both insulin release from the pancreas and glucose utilization in peripheral tissues. Thiazide-induced **glucose intolerance** is seen more commonly in patients with diabetes mellitus and metabolic syndrome (hypertension, dyslipidemia, abdominal obesity).

The metabolic adverse effects of thiazide diuretics are dose-dependent and occur more frequently with chlorthalidone than with other thiazides. However, chlorthalidone remains the preferred agent in its class based on the results of the ALLHAT trial. In this trial, chlorthalidone was associated with an overall decrease in cardiovascular mortality comparable to that seen with novel antihypertensive agents (eg, ACE inhibitors, dihydropyridine calcium channel blockers).

(Choices A and B) Thiazide diuretics can also unfavorably affect lipid metabolism by increasing LDL cholesterol and plasma triglyceride levels.

(Choices D, E, and F) Electrolyte abnormalities that can be induced by thiazide diuretics include hyponatremia, hypokalemia, hypomagnesemia, and hypercalcemia. Thiazide diuretics also reduce renal uric acid excretion, and the resulting hyperuricemia predisposes to gout.

Educational objective:
Unfavorable metabolic side effects of thiazide diuretics include hyperglycemia, increased LDL cholesterol and plasma triglycerides, and hyperuricemia. Electrolyte abnormalities that can be induced by thiazide diuretics include hyponatremia, hypokalemia, hypomagnesemia, and hypercalcemia.

References

- Metabolic issues in the Antihypertensive and Lipid-Lowering Heart Attack Trial Study

A 57-year-old homeless man with known HIV infection comes to the emergency department complaining of shortness of breath and a nonproductive cough over the past week. He has a history of type 2 diabetes mellitus with poor follow-up and medication compliance. His last CD4 count was 120 cells/μL 3 months ago. His temperature is 38.9° C (102° F), pulse is 110/min, respirations are 24/min, and blood pressure is 120/80 mm Hg. Oxygen saturation is 86% on room air. Lung examination is notable for diffuse rales bilaterally. The abdomen is soft and nontender.

Laboratory results are as follows:

Creatinine	1.2 mg/dL
Potassium	4.2 mEq/L

Chest x-ray shows bilateral patchy opacities in the lower lungs. The patient is treated with azithromycin, ceftriaxone, trimethoprim-sulfamethoxazole, prednisone, and albuterol. He is also given insulin before meals for his diabetes mellitus. On the third day of hospitalization, his serum potassium is 5.9 mEq/L and his creatinine is 1.5 mg/dL. Which of the following medications is most likely responsible for these laboratory changes?

- A. Albuterol
- B. Azithromycin
- C. Ceftriaxone
- D. Insulin
- E. Prednisone
- F. Trimethoprim

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- A. Albuterol [13%]
- B. Azithromycin [13%]
- C. Ceftriaxone [7%]
- D. Insulin [6%]
- E. Prednisone [19%]
- ✔ F. Trimethoprim [40%]

Explanation

Medications that can cause hyperkalemia	
Medication	**Mechanism**
Nonselective beta-adrenergic blockers	Interferes with beta-2-mediated intracellular potassium uptake
ACE inhibitor, ARB, K^+ sparing diuretics	Inhibition of aldosterone or the ENaC channel
Digitalis	Inhibition of the Na-K-ATPase pump
Cydosporine	Blocks aldosterone activity
Heparin	Blocks aldosterone production
NSAIDs	Decreases renal perfusion resulting in decreased K^+ delivery to the collecting ducts
Succinylcholine	Causes extracellular leakage of potassium through acetylcholine receptors

Trimethoprim (TMP) and sulfamethoxazole (SMX) work sequentially to block the bacterial synthesis of tetrahydrofolic acid. TMP-SMX is commonly used for treating uncomplicated urinary tract infections (Gram-negative rods), *Pneumocystis jirovecii* pneumonia, community-acquired MRSA skin infections, and nocardiosis.

Trimethoprim can cause hyperkalemia by blocking the epithelial sodium channel in the collecting tubule, similar to the action of the potassium-sparing diuretic amiloride. This occurs more commonly in HIV-infected patients who are treated with high doses of trimethoprim, but even normal doses can produce a modest elevation in the plasma potassium concentration. Thus, patients treated with high-dose trimethoprim require serial monitoring of potassium to avoid serious complications. Trimethoprim also competitively inhibits renal tubular creatinine secretion and may cause an artificial increase in serum creatinine; however, glomerular filtration rate is unchanged.

(Choices A and D) Albuterol (a selective beta-2 agonist) and insulin lower serum potassium temporarily by driving potassium intracellularly. They are used to treat hyperkalemia.

(Choices B and C) Macrolide antibiotics (eg, azithromycin) can cause prolonged QT and cholestasis. Ceftriaxone is also associated with cholestasis and is contraindicated in neonates at high risk of developing cholestasis. Neither drug affects potassium levels.

(Choice E) Prednisone has some mineralocorticoid activity, but it would cause potassium loss, not retention.

Educational objective:
Trimethoprim can cause hyperkalemia due to blockade of the epithelial sodium channel in the collecting tubule. Trimethoprim also competitively inhibits renal tubular creatinine secretion and may cause an artificial increase in serum creatinine without affecting the glomerular filtration rate.

References

- Trimethoprim-sulfamethoxazole: hyperkalemia is an important complication regardless of dose.

unchanged from prior tracings. Laboratory studies are normal except for serum creatinine of 2.0 mg/dL. The patient is given oxygen and furosemide and is started on a nitroprusside drip. After 36 hours, his blood pressure is 140/80 mm Hg and he is no longer dyspneic; however, he is confused and disoriented and has diffuse hyperreflexia. There are no other focal neurologic findings. Repeat laboratory results are as follows:

Sodium	140 mEq/L
Potassium	4.4 mEq/L
Chloride	100 mEq/L
Bicarbonate	14 mEq/L
Creatinine	2.5 mg/dL

Which of the following is the most likely cause of this patient's symptoms?

A. Alcohol withdrawal
B. Cyanide toxicity
C. Ischemic brain injury
D. Lacunar infarcts
E. Uremic encephalopathy

Submit

A 72-year-old man comes for evaluation of shortness of breath, headache, and fatigue. He has a history of hypertension, lives alone, and is not compliant with his medications. The patient drinks 1 or 2 glasses of wine daily but does not smoke. Blood pressure is 184/128 mm Hg, pulse is 96/min, and oxygen saturation is 90% by pulse oximetry on room air. He has early papilledema on funduscopic examination. Pulmonary examination shows bibasilar coarse crackles. Cardiac examination shows a grade 2/6 systolic ejection murmur along the left sternal border. There is trace pedal edema. Chest x-ray reveals mild pulmonary edema. An ECG is unchanged from prior tracings. Laboratory studies are normal except for serum creatinine of 2.0 mg/dL. The patient is given oxygen and furosemide and is started on a nitroprusside drip. After 36 hours, his blood pressure is 140/80 mm Hg and he is no longer dyspneic; however, he is confused and disoriented and has diffuse hyperreflexia. There are no other focal neurologic findings. Repeat laboratory results are as follows:

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Which of the following is the most likely cause of this patient's symptoms?

- A. Alcohol withdrawal [5%]
- ✔ B. Cyanide toxicity [52%]
- C. Ischemic brain injury [10%]
- D. Lacunar infarcts [4%]

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- A. Alcohol withdrawal [5%]
- ✔ B. Cyanide toxicity [52%]
- C. Ischemic brain injury [10%]
- D. Lacunar infarcts [4%]
- E. Uremic encephalopathy [27%]

Omitted
Correct answer
B

52%
Answered correctly

8 Seconds
Time Spent

07/25/2018
Last Updated

Explanation

Manifestations of cyanide accumulation & toxicity

- Skin: **Flushing** (cherry-red color), cyanosis (occurs later)
- Central nervous system: Headache, **altered mental status**, seizures, coma
- Cardiovascular: Arrhythmias
- Respiratory: Tachypnea followed by respiratory depression, pulmonary edema
- Gastrointestinal: Abdominal pain, nausea, vomiting
- Renal: **Metabolic acidosis** (from lactic acidosis), renal failure

This patient's presentation suggests **cyanide toxicity**. Common etiologies include combustion of carbon- and nitrogen-containing compounds (eg, wool, silk), industrial exposure (eg, metal extraction in mining), and medications (eg, **sodium nitroprusside**). Cyanide binds to cytochrome oxidase and inhibits mitochondrial oxidative phosphorylation. Cells then shift to anaerobic metabolism with decreased ATP production and eventual lactic acidosis. Patients can develop symptoms affecting the neurologic (eg, headache, confusion, hyperreflexia due to thiocyanate accumulation), cardiovascular (eg, arrhythmias), respiratory (eg, tachypnea followed by respiratory depression), and gastrointestinal (eg, vomiting) systems, and skin (eg, flushing).

Sodium nitroprusside is a potent arterial and venous vasodilator often used for hypertensive emergencies. The drug contains 5 cyanide groups and undergoes rapid conversion to cyanide and eventually thiocyanate, which is eliminated by the kidneys. Prolonged infusion (>24 hours) at high rates (5-10 µg/kg/min) can lead to cyanide toxicity, especially in patients with chronic kidney disease. As

respiratory depression), and gastrointestinal (eg, vomiting) systems, and skin (eg, flushing).

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(Choice A) Alcohol withdrawal can cause agitation, tachycardia, hypertension, and mental status changes. However, withdrawal is more common in patients with heavy alcohol use who stop suddenly. This patient's moderate alcohol use and absence of tachycardia make this less likely.

(Choice C) Cerebral vessels may not auto-regulate in chronic hypertension. Significant decreases in blood pressure with failed autoregulation may significantly lower cerebral blood flow and cause ischemia. However, this patient's symptoms after sodium nitroprusside use are more concerning for cyanide toxicity.

(Choice D) Lacunar infarcts are small strokes from occluded penetrating arteries, usually located in deep brain structures. Lacunar infarcts usually present with sensory or motor deficits, impaired cognition, or dysarthria. This patient's absence of focal neurological findings makes this less likely.

(Choice E) Uremic encephalopathy can present with altered mentation and occasional myoclonus and usually occurs in patients with advanced renal failure. This patient's mild creatinine rise from 2.0 mg/dL to 2.5 mg/dL is unlikely to cause uremic encephalopathy.

Educational objective:
Prolonged infusion of sodium nitroprusside at high rates can lead to cyanide toxicity, especially in patients with chronic kidney disease. Typical findings include headache, confusion, arrhythmias, flushing, and respiratory depression.

References

A 34-year-old man comes to the emergency department with severe abdominal pain. He describes the pain as "sharp" and "unbearable." It is located in the lower left abdomen and radiates to the groin. He has vomited twice since it began. The patient has no burning with urination. He is afebrile. He has difficulty lying still on the examination table due to the discomfort. Mucous membranes appear slightly dry. Lungs are clear to auscultation. Heart sounds are normal and there are no murmurs or gallops. The abdomen is soft and nontender to palpation. Inspection and palpation show normal genitalia. Extremities have no cyanosis, clubbing, or edema. Which of the following is the most appropriate test to diagnose this patient's condition?

- A. Abdominal and pelvic x-ray
- B. Abdominal ultrasonogram
- C. Amylase and lipase
- D. Colonoscopy
- E. CT of the abdomen and pelvis with contrast
- F. Exploratory laparotomy
- G. Intravenous pyelography
- H. Radioisotope (HIDA) scanning
- I. Urinalysis and urine culture

Submit

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- A. Abdominal and pelvic x-ray [15%]
- ✔ B. Abdominal ultrasonogram [35%]
- C. Amylase and lipase [0%]
- D. Colonoscopy [0%]
- E. CT of the abdomen and pelvis with contrast [36%]
- F. Exploratory laparotomy [0%]
- G. Intravenous pyelography [4%]
- H. Radioisotope (HIDA) scanning [0%]
- I. Urinalysis and urine culture [6%]

Omitted
Correct answer
B

35%
Answered correctly

7 Seconds
Time Spent

08/15/2018
Last Updated

Explanation

This patient, with severe left lower abdominal pain radiating to the groin, vomiting, and unremarkable findings on abdominal examination, has typical features of obstructive ureterolithiasis. Acute obstruction of the ureter at the ureterovesical junction produces severe flank pain that radiates to the perineum, penis, scrotum, or inner thigh. Patients with ureteral stones are often writhing in pain and unable to sit still in the examination room. Obstruction of hollow viscera often produces severe pain that is colicky in nature and poorly localized. Other causes of visceral pain include obstruction of the small intestine, colon, common bile duct, gall bladder, or urinary bladder, each of which produces a **characteristic pattern of referred pain**.

The preferred modalities for diagnosing a ureteral **stone** are **ultrasonography** or **noncontrast spiral CT** scan of the abdomen and pelvis. Ultrasonography provides good imaging of the kidney and the proximal ureter and has lower radiation exposure. It is preferred in patients with a low likelihood of alternate diagnosis and in pregnant patients. However, it can also miss small stones, and its quality is dependent on the technician's expertise.

(Choice A) Abdominal and pelvic x-ray (kidney-ureter-bladder study) will identify radiopaque, calcium-containing stones. Other types of urinary stones, accounting for at least 15% of cases, are not visible on plain x-ray. This method can also miss small stones at the ureterovesical junction. X-ray gives no information about obstruction of the ureter or kidney. However, it may be useful in serial follow-up of stones in the kidney or renal pelvis.

(Choice C) Amylase and lipase help in the diagnosis of acute pancreatitis. In acute pancreatitis, pain typically radiates to the back (upper lumbar region).

(Choice D) Colonoscopy is usually not performed in patients with acute abdominal pain due to the risk of perforation. If colonic obstruction is suspected and there is no evidence of perforation, a contrast enema study (barium or water-soluble contrast) can be diagnostic.

(Choice E) Contrast is not required to visualize ureteral stones. A noncontrast CT of the abdomen and pelvis can be obtained

obstruction is suspected and there is no evidence of perforation, a contrast enema study (barium or water-soluble contrast) can be diagnostic.

(Choice E) Contrast is not required to visualize ureteral stones. A noncontrast CT of the abdomen and pelvis can be obtained quickly, is very sensitive and specific, can visualize small or non-calcium stones, and may identify other potential causes of abdominal pain.

(Choice F) Exploratory laparotomy is indicated when there is evidence of peritonitis, as from a perforated viscus (free air under the diaphragm), ruptured aortic aneurysm, or abdominal trauma. Individuals with peritonitis typically lie still and avoid any movement that irritates the inflamed peritoneum. There are no physical signs of peritonitis, such as guarding, rigidity, or rebound tenderness, in this patient.

(Choice G) An intravenous pyelogram (IVP) uses IV contrast and plain x-ray to visualize the urinary system. IVP was previously the test of choice for diagnosing urinary stones. However, due to the risk of contrast administration (eg, allergy, acute kidney injury), noncontrast CT is now preferred.

(Choice H) Radioisotope (HIDA) scan is indicated in patients with acute cholecystitis when ultrasonography cannot definitively demonstrate obstruction at the neck of the gall bladder. In acute cholecystitis, pain is located predominantly in the upper abdomen and radiates to the tip of the right scapula or right shoulder.

(Choice I) Urinalysis in patients with urinary stones will show microscopic or gross hematuria in over 90% of cases. However, the presence of hematuria is nonspecific. It is important to exclude coexistent urinary tract infection in patients with urinary calculi as this would significantly change management. This patient has no history of fever or dysuria to suggest an infection.

Educational objective:
Ureteral calculi may cause flank or abdominal pain radiating to the perineum, often with nausea and vomiting. Ultrasonography or a noncontrast spiral CT scan of the abdomen and pelvis are the imaging modalities of choice to confirm the diagnosis. Ultrasonography is preferred in pregnant patients to reduce radiation exposure.

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(Choice E) C
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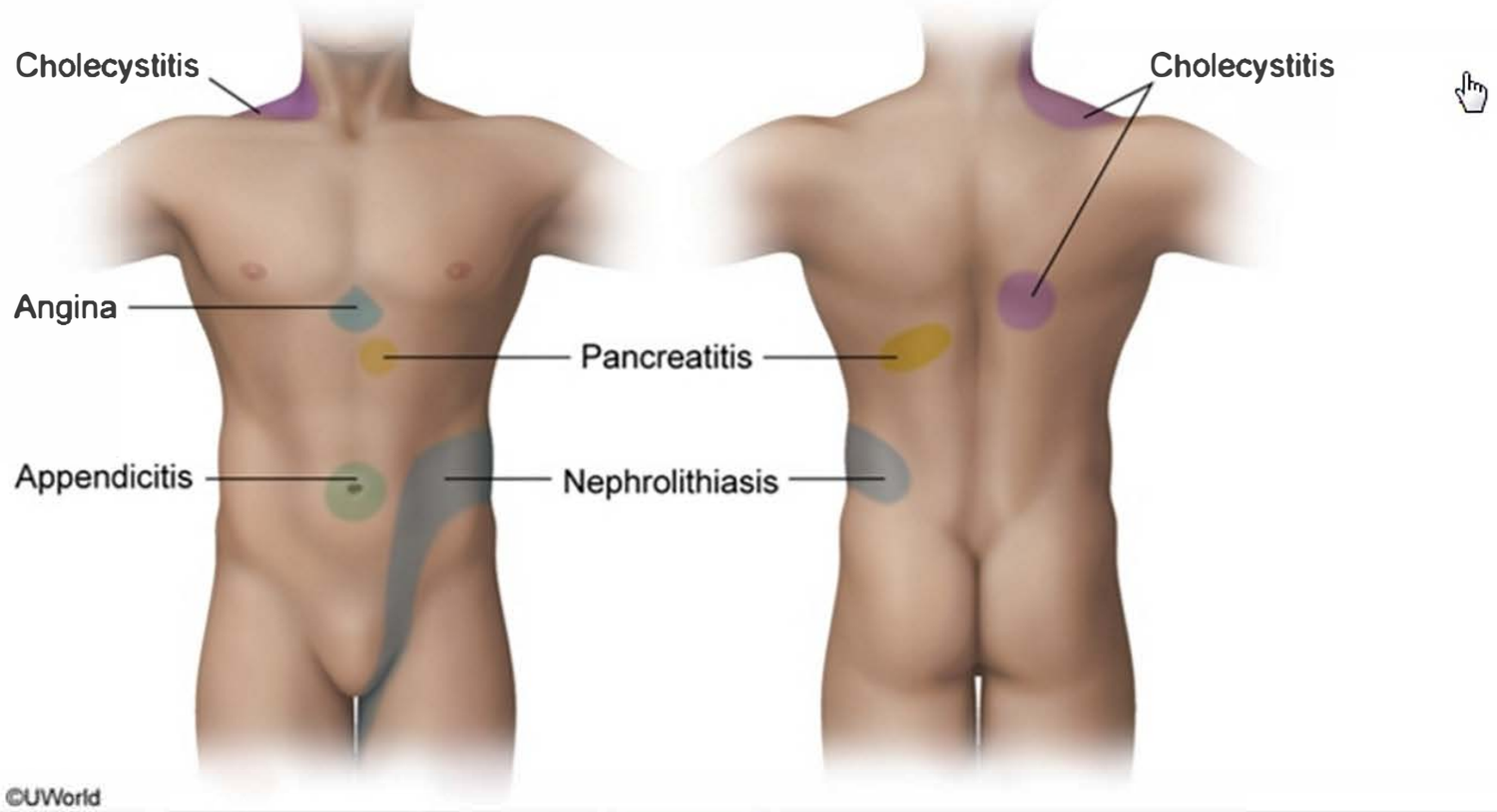
diaphragm), ruptured aortic aneurysm, or abdominal trauma. Individuals with peritonitis typically lie still and avoid any movement that

Exhibit Display

Areas of referred pain to the abdomen



Zoom In | Zoom Out | Reset | Add To Flash Card

A 44-year-old woman, gravida 5 para 5, comes to the office due to increasingly frequent loss of urine over the last year. Throughout the day and once or twice a night, the patient feels a strong urge to void and occasionally leaks urine on the way to the restroom. These symptoms persist despite 3 months of pelvic floor exercises, which she performs regularly. The patient has difficulty concentrating at work due to these symptoms, despite trying to hold her urine for longer periods. She underwent a midurethral sling procedure 3 years ago for leakage of urine when laughing, coughing, and running. The patient has well-controlled asthma and uses an inhaled beta-2 agonist infrequently. She was diagnosed with depression last year but does not take any medications for it. Physical examination shows a slight bulge at the anterior vaginal wall. Urinalysis is normal. Postvoid residual volume is 50 mL. Which of the following is the most appropriate therapy for this patient's urinary symptoms?

- A. Intermittent urethral catheterization
- B. Oral bethanechol therapy
- C. Oral duloxetine therapy
- D. Oral oxybutynin therapy
- E. Vaginal estrogen therapy
- F. Vaginal pessary placement

Submit

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- A. Intermittent urethral catheterization [2%]
- B. Oral bethanechol therapy [9%]
- C. Oral duloxetine therapy [2%]
- ✔ D. Oral oxybutynin therapy [53%]
- E. Vaginal estrogen therapy [1%]
- F. Vaginal pessary placement [30%]

Omitted
Correct answer
D

53%
Answered correctly

2 Seconds
Time Spent

12/14/2018
Last Updated

Explanation

Explanation

Urinary incontinence		
Type	**Symptoms**	**Treatment**
Stress	Leakage with Valsalva (coughing, sneezing, laughing)	• Lifestyle modifications • Pelvic floor exercises • Pessary • Pelvic floor surgery
Urgency	Sudden, overwhelming, or frequent need to void	• Lifestyle modifications • Bladder training • Antimuscarinic drugs
Mixed	Features of stress & urgency incontinence	• Variable treatment depending on predominant symptoms
Overflow	Constant involuntary dribbling & incomplete emptying	• Identify & correct underlying cause • Cholinergic agonists • Intermittent self-catheterization

This patient has **urge incontinence**, the result of **detrusor overactivity**. Risk factors include age >40, female gender, and pelvic surgery, all of which are present in this patient. Symptoms occur throughout the day and night and are not related to events that increase intra-abdominal pressure. The evaluation of incontinence must include urinalysis to rule out a urinary tract infection. Measurement of a postvoid residual (PVR) may be considered to rule out overflow incontinence.

Initial treatment for urinary incontinence (stress, urgency, mixed) includes **bladder training** and pelvic floor muscle **(Kegel)** exercises. For urge incontinence, bladder training involves resisting the urge to void for progressively longer periods to increase bladder

	incomplete emptying	• Intermittent self-catheterization

This patient has **urge incontinence**, the result of **detrusor overactivity**. Risk factors include age >40, female gender, and pelvic surgery, all of which are present in this patient. Symptoms occur throughout the day and night and are not related to events that increase intra-abdominal pressure. The evaluation of incontinence must include urinalysis to rule out a urinary tract infection. Measurement of a postvoid residual (PVR) may be considered to rule out overflow incontinence.

Initial treatment for urinary incontinence (stress, urgency, mixed) includes **bladder training** and pelvic floor muscle **(Kegel)** exercises. For urge incontinence, bladder training involves resisting the urge to void for progressively longer periods to increase bladder capacity. Reducing consumption of alcoholic, caffeinated, and carbonated beverages is also recommended.

Nonresponders require pharmacological therapy. Voiding is mediated through the parasympathetic system, and **antimuscarinic** drugs (eg, oxybutynin) increase bladder capacity and decrease detrusor contractions by reducing acetylcholine activity. Adverse antimuscarinic effects can include dry mouth, constipation, and blurry vision. Patients on antimuscarinics should be monitored for urinary retention.

(Choices A and B) Treatment of urinary retention due to neurogenic bladder consists of bethanechol (cholinergic agonist) and/or intermittent urethral catheterization. This patient's PVR is normal (≤150 mL in women, ≤50 mL in men) and she does not have urinary retention.

(Choice C) Duloxetine (serotonin/norepinephrine reuptake inhibitor) may be considered for treatment of stress incontinence in patients with unipolar depression. However, it is not effective for urge incontinence.

(Choice E) Vaginal estrogen is used for peri- or postmenopausal women with vaginal atrophy accompanied by stress or urge incontinence. Estrogen increases urethral resistance, raises the sensory threshold of bladder fullness, and promotes relaxation of the detrusor muscle. This patient has not reported atrophy or menopausal symptoms.

(Choice F) A vaginal pessary is used for symptomatic pelvic organ prolapse and stress incontinence when surgical correction (eg, midurethral sling surgery) fails or is contraindicated. A pessary can reduce urine loss in patients with stress incontinence by

urinary retention.

(Choices A and B) Treatment of urinary retention due to neurogenic bladder consists of bethanechol (cholinergic agonist) and/or intermittent urethral catheterization. This patient's PVR is normal (≤150 mL in women, ≤50 mL in men) and she does not have urinary retention.

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(Choice F) A vaginal pessary is used for symptomatic pelvic organ prolapse and stress incontinence when surgical correction (eg, midurethral sling surgery) fails or is contraindicated. A pessary can reduce urine loss in patients with stress incontinence by increasing bladder outlet resistance. Although the patient has anterior vaginal wall prolapse and previously had stress incontinence, the current urinary symptoms are consistent with urge incontinence and would be exacerbated by a pessary.

Educational objective:
First-line treatments for urgency incontinence are bladder training and pelvic floor muscle exercises. Nonresponders can use an antimuscarinic agent to decrease detrusor activity.

References

- Pharmacotherapy of overactive bladder in adults: a review of efficacy, tolerability, and quality of life.
- ACOG practice bulletin no. 155: urinary incontinence in women.

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 72-year-old man is hospitalized for a right femoral fracture following a motor vehicle collision. Six days after undergoing surgical repair, he is found to be lethargic. The patient has hypertension and osteoarthritis. His medications include hydrochlorothiazide, lisinopril, and naproxen, which were continued in the hospital. On examination, the patient is drowsy but awakens briefly when addressed by name. Temperature is 36.7 C (98 F), blood pressure is 144/76 mm Hg, pulse is 88/min, and respirations are 16/min. Pulse oximetry is 95% on room air. Multiple contusions are present in the anterior abdominal wall and thighs. Physical examination findings are shown in the video clip. Laboratory results are as follows:

Hemoglobin	8.4 g/dL
Blood urea nitrogen	78 mg/dL
Albumin	3.8 g/dL
Total bilirubin	0.4 mg/dL
Aspartate aminotransferase	112 U/L
Alanine aminotransferase	42 U/L
Creatine kinase, serum	32,000 U/L

Which of the following is the most appropriate next step in management of this patient?

Play Media

Blood urea nitrogen	78 mg/dL
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Total bilirubin	0.4 mg/dL
Aspartate aminotransferase	112 U/L
Alanine aminotransferase	42 U/L
Creatine kinase, serum	32,000 U/L

Which of the following is the most appropriate next step in management of this patient?

Play Media

- A. Blood transfusion
- B. Hemodialysis
- C. Intravenous lorazepam
- D. Intravenous morphine
- E. Lactulose
- F. Stress test

Submit

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Play Media

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Aspartate aminotransferase	112 U/L
Alanine aminotransferase	42 U/L
Creatine kinase, serum	32,000 U/L

Which of the following is the most appropriate next step in management of this patient?

Play Media

- A. Blood transfusion [1%]
- ✔ B. Hemodialysis [45%]
- C. Intravenous lorazepam [6%]
- D. Intravenous morphine [0%]
- E. Lactulose [46%]
- F. Stress test [0%]

Omitted
Correct answer
B

45%
Answered correctly

7 Seconds
Time Spent

10/26/2018
Last Updated

Indications for urgent dialysis (AEIOU)	
Acidosis	• Metabolic acidosis ◦ pH <7.1 refractory to medical therapy

Electrolyte abnormalities	• Symptomatic hyperkalemia
	  ◦ ECG changes or ventricular arrhythmias
	• Severe hyperkalemia
	  ◦ Potassium >6.5 mEq/L refractory to medical therapy

Ingestion	• Toxic alcohols (methanol, ethylene glycol) • Salicylate • Lithium • Sodium valproate, carbamazepine
Overload	• Volume overload refractory to diuretics

Uremia	• Symptomatic:
	  ◦ Encephalopathy
	  ◦ Pericarditis
	  ◦ Bleeding

The video clip demonstrates **asterixis**, a bilateral, nonrhythmic, alternate flexion and extension movement at the wrist (flapping) that occurs when the wrist is extended with the arms outstretched. Although classically associated with hepatic encephalopathy (HE), asterixis can also be seen with **uremic encephalopathy** (due to acute or chronic renal failure) and CO_2 retention. It likely develops due to interruption of the neural pathways that sustain muscle contraction.

This patient has numerous factors that together can cause a severe degree of acute **kidney injury**. These include rhabdomyolysis

- Bleeding

The video clip demonstrates **asterixis**, a bilateral, nonrhythmic, alternate flexion and extension movement at the wrist (flapping) that occurs when the wrist is extended with the arms outstretched. Although classically associated with hepatic encephalopathy (HE), asterixis can also be seen with **uremic encephalopathy** (due to acute or chronic renal failure) and CO_2 retention. It likely develops due to interruption of the neural pathways that sustain muscle contraction.

This patient has numerous factors that together can cause a severe degree of acute **kidney injury**. These include rhabdomyolysis from trauma (elevated creatine kinase), use of nonsteroidal anti-inflammatory drugs (eg, naproxen) and diuretics (eg, hydrochlorothiazide), loss of glomerular autoregulation with lisinopril, and anemia. Reduced renal clearance of uremic toxins leads to **high levels of blood urea nitrogen** (BUN) and symptoms of lethargy and somnolence. The diagnosis of uremia is based on **clinical symptoms/signs** and not on an absolute BUN level. Symptoms typically appear at a BUN level of >100 mg/dL but can develop at lower levels. Uremic encephalopathy is an indication for **urgent hemodialysis**.

(Choice A) This patient has anemia but does not need a **blood transfusion** because his hemoglobin is >8 g/dL, he is asymptomatic, and he does not have ongoing bleeding.

(Choice C) Intravenous lorazepam can be used to treat an ongoing seizure.

(Choice D) Intravenous morphine is an opioid analgesic that may worsen this patient's drowsiness and lethargy. It may occasionally cause seizures or myoclonus.

(Choice E) Lactulose is used for the management of HE in decompensated cirrhosis. Although asterixis may be seen in HE, this patient has normal bilirubin and albumin levels, suggesting normal liver synthetic function. His mildly increased aspartate aminotransferase level is likely from skeletal muscle injury rather than liver injury.

(Choice F) This patient's high creatine kinase level is due to rhabdomyolysis associated with the motor vehicle collision and is not typically seen in acute coronary syndrome.

Educational objective:

high levels of blood urea nitrogen (BUN) and symptoms of lethargy and somnolence. The diagnosis of uremia is based on **clinical symptoms/signs** and not on an absolute BUN level. Symptoms typically appear at a BUN level of >100 mg/dL but can develop at lower levels. Uremic encephalopathy is an indication for **urgent hemodialysis**.

(Choice A) This patient has anemia but does not need a **blood transfusion** because his hemoglobin is >8 g/dL, he is asymptomatic, and he does not have ongoing bleeding.

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(Choice F) This patient's high creatine kinase level is due to rhabdomyolysis associated with the motor vehicle collision and is not typically seen in acute coronary syndrome.

Educational objective:
Asterixis is a flapping movement of the hands that occurs when the wrist is extended with the arms outstretched. Common causes include hepatic encephalopathy, uremic encephalopathy, and hypercapnia. Treating the underlying cause will improve neurological status and resolve asterixis.

References

- Asterixis.

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References

- Asterixis.

Exhibit Display

Red blood cell transfusion thresholds	
Hemoglobin (g/dL)	**Recommendation**
<7	• Generally indicated
7-8	• Cardiac surgery • Oncology patients in treatment • Heart failure
8-10	• Symptomatic anemia • Ongoing bleeding • Acute coronary syndrome • Noncardiac surgery
>10	• Not generally indicated

Zoom In | Zoom Out | Reset | Add To Flash Card

A 17-year-old white female comes to the office for the evaluation of fatigue which has been present for the past 4 months. Her past medical history is insignificant. She denies the use of any drugs. Her pulse is 74/min, blood pressure is 110/70 mm Hg, and temperature is 36.7° C (98.0° F). Physical examination shows scars on the dorsum of her hands and dental erosions. Laboratory studies show:

Plasma sodium	139 mEq/L
Serum potassium	2.3 mEq/L
Bicarbonate	40 mEq/L

Urine chloride concentration is 15 mEq/L (Normal = 80-250 mEq/L). Based on these findings, what is the most likely diagnosis?

- A. Chronic diarrhea
- B. Diuretic abuse
- C. Surreptitious vomiting
- D. Primary hyperaldosteronism
- E. Bartter's syndrome

Submit

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Urine chloride concentration is 15 mEq/L (Normal = 80-250 mEq/L). Based on these findings, what is the most likely diagnosis?

- A. Chronic diarrhea [1%]
- B. Diuretic abuse [3%]
- ✔ C. Surreptitious vomiting [92%]
- D. Primary hyperaldosteronism [1%]
- E. Bartter's syndrome [1%]

Omitted
Correct answer
C

92%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Suspect the following conditions whenever a patient presents with hypokalemia, alkalosis and normotension:

1. Surreptitious vomiting
2. Diuretic abuse
3. Bartter syndrome
4. Gitelman's syndrome

Physical findings that are characteristic of surreptitious vomiting are scars/calluses on the dorsum of the hands, and dental erosions. The dorsal scars result from repeated chemical/mechanical injury as the patient uses his/her hands to induce vomiting. Dental erosions result due to increased exposure to gastric acid. Surreptitious vomiting may also result in hypovolemia and hypochloremia, which in turn lead to a low urine chloride concentration.

(Choice B and E) Patients with diuretic abuse and Bartter/Gitelman's syndrome may also present with hypokalemia, alkalosis and normotension, but their urine chloride concentrations are high. Patients with diuretic abuse may give a history of diuretic abuse and have a positive urine assay for diuretics.

(Choice A) Patients with chronic diarrhea have metabolic acidosis and hypokalemia. Metabolic acidosis occurs due to the loss of bicarbonate in the stool.

(Choice D) Patients with primary hyperaldosteronism have hypokalemia, metabolic alkalosis and hypertension.

Educational Objective:
Always suspect surreptitious vomiting as a cause of hypokalemic alkalosis in a normotensive patient, and be able to distinguish it from other entities (e.g., diuretic abuse, Bartter's syndrome) using the urine chloride concentration.

A 40-year-old woman is brought to the physician by her husband due to the recent onset of lethargy and confusion. She has been complaining of intense thirst, craving for ice water, and increased urination for the past few days. She also gets up frequently at night to urinate. The patient's medications include lithium and olanzapine for bipolar disorder and lansoprazole for acid reflux disease. Her temperature is 37.2 C (99 F), blood pressure is 83/59 mm Hg, pulse is 122/min, and respirations are 15/min. Physical examination shows a disoriented woman with dry skin and mucous membranes. Laboratory results are as follows:

Sodium	156 mEq/L
Chloride	110 mEq/L
Potassium	4.1 mEq/L
Bicarbonate	24 mEq/L
Blood glucose	180 mg/dL
Blood urea nitrogen	27 mg/dL
Creatinine	1.6 mg/dL
Serum osmolality	328 mOsm/kg
Urine osmolality	180 mOsm/kg

Serum lithium level is pending. Which of the following is the most appropriate next step in management of this patient?

A. Hemodialysis for lithium removal
B. Intravenous 0.45% saline
C. Intravenous 0.9% saline

Bicarbonate	24 mEq/L
Blood glucose	180 mg/dL
Blood urea nitrogen	27 mg/dL
Creatinine	1.6 mg/dL
Serum osmolality	328 mOsm/kg
Urine osmolality	180 mOsm/kg

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A. Hemodialysis for lithium removal
B. Intravenous 0.45% saline
C. Intravenous 0.9% saline
D. Intravenous 5% dextrose
E. Intravenous regular insulin
F. Oral free water
G. Water deprivation test

Submit

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Serum lithium level is pending. Which of the following is the most appropriate next step in management of this patient?

A. Hemodialysis for lithium removal [7%]
B. Intravenous 0.45% saline [13%]
✔ C. Intravenous 0.9% saline [58%]

Blood glucose	180 mg/dL
Blood urea nitrogen	27 mg/dL
Creatinine	1.6 mg/dL
Serum osmolality	328 mOsm/kg
Urine osmolality	180 mOsm/kg

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- B. Intravenous 0.45% saline [13%]
- ✔ C. Intravenous 0.9% saline [58%]
- D. Intravenous 5% dextrose [3%]
- E. Intravenous regular insulin [0%]
- F. Oral free water [3%]
- G. Water deprivation test [12%]

Omitted
Correct answer
C

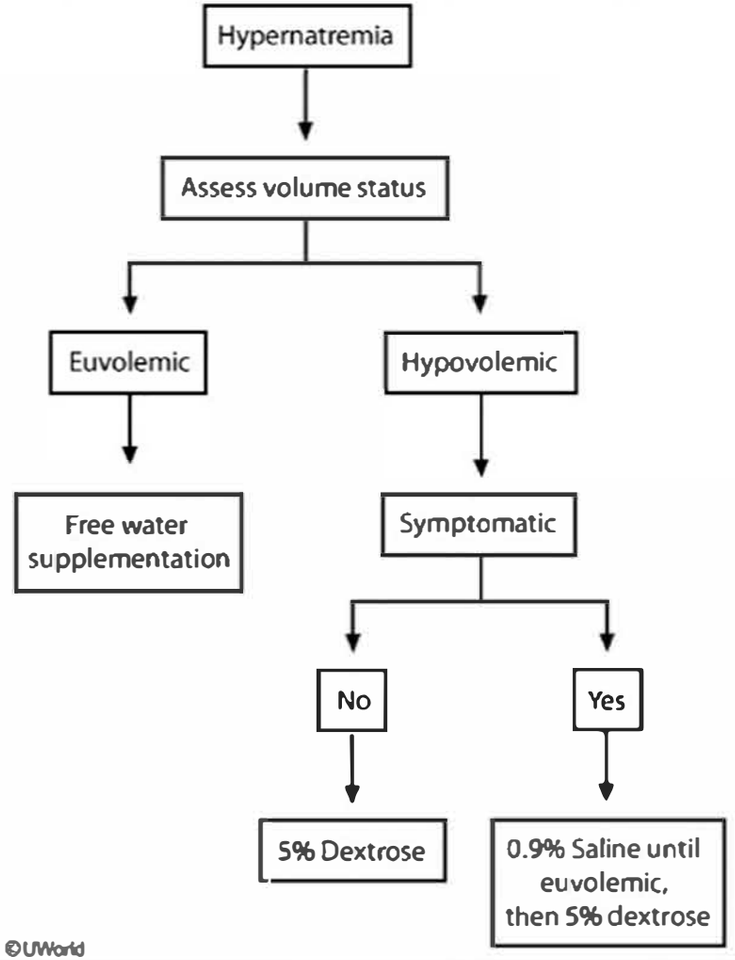
58%
Answered correctly

4 Seconds
Time Spent

11/26/2018
Last Updated

Hypernatremia

Exhibit Display



Zoom In | Zoom Out | Reset | Add To Flash Card

This patient's clinical presentation (hypotension, tachycardia, poor skin turgor) is most consistent with hypovolemic hypernatremia. The first step is to restore volume with isotonic fluids (0.9% saline). Isotonic fluid is not usually used in hypernatremia, but it is recommended in patients with marked volume depletion and hemodynamic instability. Once the patient is euvolemic, the fluid can be switched to a hypotonic fluid (5% dextrose preferred over 0.45% saline) for free water supplementation **(Choices B and D)**. The serum sodium should be corrected by 0.5 mEq/dL/hr without exceeding 12 mEq/dL/24 hr. Cerebral edema can occur if the sodium is corrected too quickly.

This patient's high serum and low urine osmolality indicate an inability to concentrate the urine due to inadequate anti-diuretic hormone (ADH) response. This is most likely due to lithium-induced **nephrogenic diabetes insipidus**. Lithium induces ADH resistance by impairing water reabsorption in the collecting duct. Patients typically develop acute-onset nocturia, polyuria and polydipsia. If water intake is inadequate, significant hypernatremia and central nervous system symptoms can develop. Discontinuing lithium is recommended, with salt restriction and selected diuretics (eg, amiloride) as an alternative for patients who cannot stop lithium.

(Choice A) Hemodialysis is usually indicated for serum lithium level >4 mEq/L or lithium level >2.5 mEq/L **plus** signs of significant lithium toxicity (eg, seizures, depressed mental status) or inability to excrete lithium (eg, renal disease, decompensated heart failure). Hemodialysis may be necessary in this patient once the lithium level is known and hypovolemia has been corrected.

(Choice E) Intravenous insulin is indicated in diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar coma (HHS). Both conditions can lead to an osmotic diuresis with some patients developing hypernatremia, but HHS and DKA are associated with severe hyperglycemia (often >800). Mild hyperglycemia, as in this patient, does not require intravenous insulin.

(Choice F) Hypernatremia typically indicates free water loss (dehydration), but this patient also has isotonic fluid loss (volume depletion) which must be corrected first. Due to her confusion, intravenous fluids are more effective than oral free water for treating the dehydration and hypernatremia.

(Choice G) A **water deprivation test** can differentiate between central (decreased ADH from pituitary) and nephrogenic (normal ADH with renal ADH resistance) diabetes insipidus. It can also definitively exclude primary polydipsia. However, this test would not

lithium.

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(Choice G) A **water deprivation test** can differentiate between central (decreased ADH from pituitary) and nephrogenic (normal ADH with renal ADH resistance) diabetes insipidus. It can also definitively exclude primary polydipsia. However, this test would not typically be done until the patient is euvolemic.

Educational objective:
Intravenous normal (0.9%) saline is preferred for treating hypovolemic hypernatremia. The fluid can be switched to a hypotonic fluid (5% dextrose in water preferred over 0.45% saline) for free water supplementation once the patient is euvolemic.

References

- A clinical approach to the treatment of chronic hypernatremia.
- Lithium-induced nephrogenic diabetes insipidus: new clinical and experimental findings.

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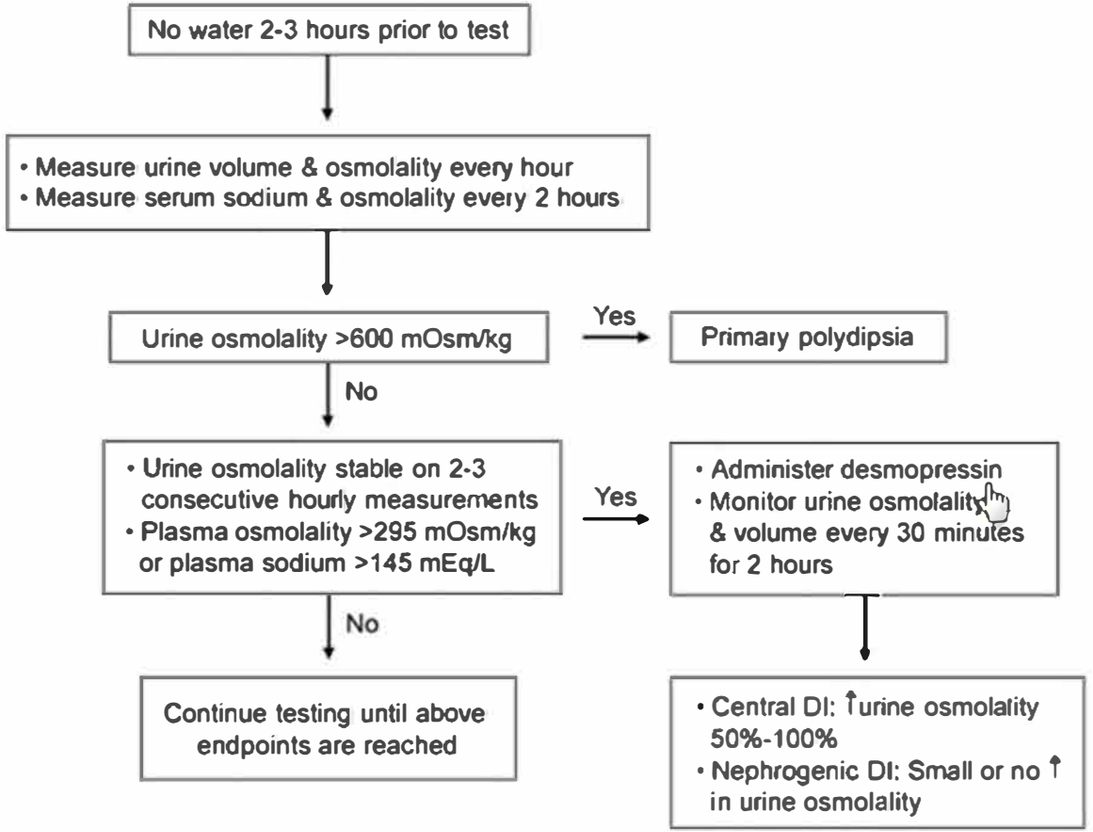
- A clinical a
- Lithium-inc

Exhibit Display

Water deprivation test



Zoom In | Zoom Out | Reset | Add To Flash Card

A 60-year-old woman is brought to the hospital due to lethargy and confusion that have progressed over the last 2 days. This morning she was hard to arouse. The patient's husband says that she has lost 4.5 kg (10 lb) over the preceding 2-month period and has had a dry cough. She has no other medical problems and takes no medication. The patient has a 40-pack-year smoking history. She does not drink alcohol. Temperature is 37 C (98.6 F), blood pressure is 110/70 mm Hg, pulse is 75/min, and respirations are 16/min. She is not orthostatic. The patient is markedly confused. Her mucous membranes are moist. There is no jugular venous distension. Breath and heart sounds are normal. Neurologic examination shows no focal abnormalities. There is no ankle edema or ascites. Laboratory results are as follows:

Sodium	117 mEq/L
Potassium	3.9 mEq/L
Bicarbonate	22 mEq/L
Blood glucose	100 mg/dL
Blood urea nitrogen	10 mg/dL

Serum osmolality is 250 mOsm/kg H_2O, and urine osmolality is 500 mOsm/kg H_2O. Chest x-ray reveals a mass in the right hilar region. CT scan of the head is unremarkable. Which of the following is the best next step in management of this patient?

A. Hypertonic saline
B. Intravenous 0.45% solution
C. Intravenous desmopressin
D. Intravenous hydrocortisone
E. Mannitol therapy

Sodium	117 mEq/L
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C. Intravenous desmopressin
D. Intravenous hydrocortisone
E. Mannitol therapy
F. Normal saline
G. Sodium bicarbonate therapy

Submit

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- A. Hypertonic saline [46%]
- B. Intravenous 0.45% solution [3%]
- C. Intravenous desmopressin [11%]
- D. Intravenous hydrocortisone [0%]
- E. Mannitol therapy [5%]

Potassium	3.9 mEq/L
Bicarbonate	22 mEq/L
Blood glucose	100 mg/dL
Blood urea nitrogen	10 mg/dL

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- B. Intravenous 0.45% solution [3%]
- C. Intravenous desmopressin [11%]
- D. Intravenous hydrocortisone [0%]
- E. Mannitol therapy [5%]
- F. Normal saline [30%]
- G. Sodium bicarbonate therapy [1%]

Omitted
Correct answer
A

46%
Answered correctly

4 Seconds
Time Spent

10/22/2018
Last Updated

Explanation

Syndrome of inappropriate antidiuretic hormone	
Etiologies	• CNS disturbance (eg, stroke, hemorrhage, trauma) • Medications (eg, carbamazepine, SSRIs, NSAIDs) • Lung disease (eg, pneumonia) • Ectopic ADH secretion (eg, small cell lung cancer) • Pain &/or nausea
Clinical features	• Mild/moderate hyponatremia - nausea, forgetfulness • Severe hyponatremia - seizures, coma • Euvolemia (eg, moist mucous membranes, no edema, no JVD)
Laboratory findings	• Hyponatremia • Serum osmolality <275 mOsm/kg H_2O (hypotonic) • Urine osmolality >100 mOsm/kg H_2O • Urine sodium >40 mEq/L
Management	• Fluid restriction ± salt tablets • Hypertonic (3%) saline for severe hyponatremia

ADH = antidiuretic hormone; **CNS** = central nervous system; **JVD** = jugular venous distension; **NSAIDs** = nonsteroidal anti-inflammatory drugs; **SSRIs** = selective serotonin reuptake inhibitors.

This patient's chest x-ray findings of a hilar mass in the setting of weight loss, cough, and a significant smoking history are highly suspicious for **small cell lung cancer**, which is a common cause of the **syndrome of inappropriate antidiuretic hormone**

This patient's chest x-ray findings of a hilar mass in the setting of weight loss, cough, and a significant smoking history are highly suspicious for **small cell lung cancer**, which is a common cause of the **syndrome of inappropriate antidiuretic hormone secretion (SIADH)**. The characteristic findings of SIADH are **hyponatremia**, **serum osmolality <275 mOsm/kg H_2O**, and **urine osmolality >100 mOsm/kg H_2O** (inappropriately elevated) in a **euvolemic** patient.

The symptoms of SIADH depend on the degree of hyponatremia and the rate at which it occurred. Rapid decreases (over <48 hours) in serum sodium concentration lead to profound fluid shifts and severe symptoms. Patients with serum sodium 130-135 mEq/L are usually asymptomatic; those with chronic hyponatremia with serum sodium 120-130 mEq/L may be asymptomatic or display **mild symptoms** (lethargy, forgetfulness). These patients usually respond to fluid restriction (<800 mL/day), sometimes with the addition of salt tablets.

Patients with serum sodium <120 mEq/L may have **severe symptoms** (eg, profound confusion, seizures, coma), which signal an increased risk of **cerebral edema** and brainstem herniation. These patients require an urgent increase in their serum sodium concentration that is best accomplished with administration of **hypertonic (3%) saline**. Extreme care must be taken with the rate of correction (≤8 mEq/L over the first 24 hours) as rapid correction can lead to **osmotic demyelination syndrome**, a devastating and potentially fatal complication (eg, dysarthria, quadriparesis, coma).

(Choices B and F) Effective management of SIADH requires the infused fluid to have a higher electrolyte concentration than that of urine (500 mOsm/kg H_2O in this patient). Normal saline (0.9%) and half normal saline (0.45%) have electrolyte concentrations of approximately 300 and 150 mOsm/kg H_2O, respectively. Intravenous infusion of either of these fluids would cause a net increase in total body free water and worsen the hyponatremia.

(Choice C) Desmopressin is an antidiuretic hormone analog used to treat diabetes insipidus. It would worsen hyponatremia in a patient with SIADH.

(Choice D) Intravenous hydrocortisone is the appropriate treatment for hyponatremia due to adrenal insufficiency, which would present with hypovolemia (eg, dry mucous membranes), hypotension, and possibly orthostasis.

A 17-year-old African American boy comes to the physician after an episode of gross hematuria that resolved spontaneously. He has no other complaints. He has no other medical conditions and takes no medications. The boy smokes occasionally but does not use illicit drugs or alcohol. His temperature is 36.7 C (98 F), blood pressure is 120/70 mm Hg, pulse is 70/min, and respirations are 14/min. Physical examination shows no abnormalities. Laboratory studies show a creatinine level of 0.9 mg/dL. Dipstick urinalysis results in the office are as follows:

Urinalysis	
Specific gravity	1.010
pH	6.2
Protein	None
Blood	Moderate
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Negative
Nitrite	Negative
Bacteria	None
Red blood cells	Many/hpf
Crystals	None

Urine microscopic examination shows numerous intact red blood cells with no other abnormal findings. Which of the following is the

Blood	Moderate
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Negative
Nitrite	Negative
Bacteria	None
Red blood cells	Many/hpf
Crystals	None

Urine microscopic examination shows numerous intact red blood cells with no other abnormal findings. Which of the following is the most likely cause of this patient's hematuria?

- A. Acute cystitis
- B. Acute glomerulonephritis
- C. Acute interstitial nephritis
- D. Acute tubular necrosis
- E. Renal papillary necrosis

Submit

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Bacteria	None
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Crystals	None

Urine microscopic examination shows numerous intact red blood cells with no other abnormal findings. Which of the following is the most likely cause of this patient's hematuria?

- A. Acute cystitis [19%]
- B. Acute glomerulonephritis [19%]
- C. Acute interstitial nephritis [6%]
- D. Acute tubular necrosis [6%]
- ✔ E. Renal papillary necrosis [47%]

Omitted
Correct answer
E

47%
Answered correctly

13 Seconds
Time Spent

11/25/2018
Last Updated

Explanation

Explanation

Sickle cell trait	
Clinical features	• Usually asymptomatic • No change in overall life expectancy
Laboratory findings	• Normal hemoglobin, reticulocyte count, RBC indices & morphology • Hemoglobin electrophoresis: Hb A > Hb S
Complications	• Hematuria/papillary necrosis, hyposthenuria • Splenic infarction (especially at higher altitudes), venous thromboembolism, priapism • Exertional rhabdomyolysis

Hb A = hemoglobin A; **Hb S** = hemoglobin S; **RBC** = red blood cell.

Gross hematuria can be from bladder (eg, cystitis, cancer), renal (eg, glomerulonephritis), ureteral (eg, nephrolithiasis), or prostate (eg, benign prostatic hyperplasia) pathology. This patient's gross hematuria and urinalysis with normal-appearing red blood cells (RBCs) suggest an extra-glomerular etiology. Sickle cell trait is a benign condition associated with hemoglobin AS that is more common in Africa, the Middle East, and Mediterranean countries, and in African American and Hispanic individuals. Patients with sickle cell trait have no clinical symptoms of sickle cell disease and no change in overall life expectancy. The diagnosis is confirmed by hemoglobin electrophoresis showing hemoglobin AS. However, patients have normal hemoglobin concentration, reticulocyte count, and RBC indices and morphology.

Renal complications of **sickle cell trait** include hematuria, renal medullary carcinoma, and urinary tract infections. Painless hematuria is likely due to renal papillary ischemia or necrosis. The relatively low local partial pressure of oxygen in the vasa rectae predisposes the affected red blood cells to sickling. **Papillary necrosis** can occur with massive **hematuria**, but the episodes are usually mild and

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(Choice A) Acute cystitis usually presents with dysuria and increased urinary frequency. Urinalysis usually shows many white blood cells and bacteria with positive nitrite and leukocyte esterase. However, this patient has no urinary symptoms or urinalysis showing bacteria, leukocyte esterase, or nitrite.

(Choice B) Acute glomerulonephritis typically presents with hematuria and evidence of glomerular injury (eg, hypertension, proteinuria, dysmorphic RBCs, RBC casts). This patient's urinalysis without dysmorphic RBCs or proteinuria makes this less likely.

(Choice C) Acute interstitial nephritis is usually associated with history of medication use, rash, eosinophilia, and increased serum creatinine. Urinalysis usually shows abundant white blood cells, many of which are eosinophils. However, acute interstitial nephritis does not typically cause gross hematuria.

(Choice D) Acute tubular necrosis is suspected in patients with acute kidney injury due to ischemic or nephrotoxic insult. Serum creatinine is elevated. Urinalysis usually shows granular casts, hematuria, and renal tubular epithelial cells. However, acute tubular necrosis typically does not cause the isolated hematuria seen in this patient.

Educational objective:
Sickle cell trait is a benign condition with hemoglobin AS that is more common in Africa, the Middle East, and Mediterranean countries, and in African American and Hispanic individuals. Patients with sickle cell trait have no specific clinical symptoms of sickle cell disease and no change in overall life expectancy. Renal complications include painless hematuria, urinary tract infections, and renal medullary cancer.

A 58-year-old man hospitalized for confusion is evaluated for elevated serum creatinine. He has a history of cirrhosis due to chronic hepatitis C infection, and evaluation at the time of admission revealed hepatic encephalopathy and spontaneous bacterial peritonitis. Since admission, the patient's mental status has gradually improved with lactulose and cefotaxime therapy, but his renal functioning has worsened. At home, he takes furosemide, spironolactone, and nadolol, all of which were stopped at the time of admission. Temperature is 37 C (98.6 F), blood pressure is 98/62 mm Hg, and pulse is 102/min. Physical examination shows jaundice, spider angiomas, ascites, and leg edema. He has no skin rash. Serum creatinine and urine output since admission are shown below.

	Admission	Day 1	Day 2	Day 3	Day 4
Serum creatinine (mg/dL)	1.1	1.6	2.4	3.1	3.9
Urine output (mL/24 hr)	-	2100	1400	800	500

Urinalysis reveals 1-2 erythrocytes/hpf with no significant casts or protein. Renal ultrasound shows normal-size kidneys with no hydronephrosis. There has been no improvement in renal function with intravenous saline and albumin infusion. Which of the following is the most likely mechanism of this patient's renal dysfunction?

A. Drug-mediated renal tubular injury
B. Glomerular immune complex deposition
C. Intravascular volume depletion
D. Renal interstitial inflammation
E. Splanchnic arterial dilation

Submit

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- A. Drug-mediated renal tubular injury [15%]
- B. Glomerular immune complex deposition [7%]
- C. Intravascular volume depletion [32%]
- D. Renal interstitial inflammation [11%]
- ✔ E. Splanchnic arterial dilation [32%]

Omitted
Correct answer
E

32%
Answered correctly

5 Seconds
Time Spent

12/13/2018
Last Updated

Explanation

Hepatorenal syndrome	
Risk factors	• Advanced cirrhosis with portal hypertension & edema
Precipitating factors	• Reduced renal perfusion • GI bleed, vomiting, sepsis, excessive diuretic use, SBP • Reduced glomerular pressure & GFR ◦ NSAID use (constricts afferent arterioles)
Diagnosis	• Renal hypoperfusion • FeNa <1% (or urine Na <10 mEq/L) • Absence of tubular injury • No RBC, protein, or granular casts in urine • No improvement in renal function with fluids
Treatment	• Address precipitating factors (eg, hypovolemia, anemia, infection) • Splanchnic vasoconstrictors (midodrine, octreotide, norepinephrine) • Liver transplantation

FeNa = fractional excretion of sodium; **GFR** = glomerular filtration rate; **GI** = gastrointestinal; **RBC** = red blood cells; **SBP** = spontaneous bacterial peritonitis.

Hepatorenal syndrome (HRS) is an important complication of end-stage liver disease. It is characterized by a significant decrease in glomerular filtration in the absence of another clear cause of renal dysfunction, minimal hematuria (<50 erythrocytes/hpf), and lack of improvement with volume resuscitation.

HRS occurs as **cirrhosis** progresses and patients develop **splanchnic arterial dilation** and an overall decrease in vascular resistance. This, in turn, activates the renin-angiotensin system and results in local **renal vasoconstriction** with **decreased** perfusion and **glomerular filtration**. The most common inciting factors of HRS include spontaneous bacterial peritonitis and gastrointestinal bleeding.

Definitive therapy involves return of liver function through either hepatic recovery (eg, abstinence from alcohol) or transplantation. For patients who are unlikely to have a quick recovery and are not candidates for liver transplantation, other temporizing options to increase renal perfusion include splanchnic vasoconstrictors (eg, midodrine, octreotide, norepinephrine) and albumin. Dialysis is of limited benefit but may be attempted to help bridge to liver transplantation.

(Choice A) Acute tubular necrosis is one of the main differential diagnoses of HRS, and patients with cirrhosis are especially susceptible. Aminoglycosides, iodinated contrast dye, and hypotension are the most common causes; however, urinalysis shows muddy brown, granular casts.

(Choice B) Patients with hepatitis C are at risk for a number of immune-mediated complications, including membranoproliferative glomerulonephritis, which commonly occurs in association with mixed cryoglobulinemia syndrome (an immune complex deposition disorder). Urinalysis usually reveals dysmorphic red cells, red cell casts, and protein.

(Choice C) Patients with intravascular volume depletion may demonstrate acute kidney injury, but in the absence of another insult such as acute tubular necrosis, renal function should improve with volume resuscitation and discontinuation of diuretics.

(Choice D) Acute interstitial nephritis (AIN) is frequently caused by antibiotics (such as those administered for spontaneous bacterial peritonitis) or infection (although AIN is not typically associated with hepatitis C infection). However, urinalysis demonstrates an active sediment with white cells and white cell casts.

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Educational objective:
Hepatorenal syndrome is characterized by a decrease in glomerular filtration in the absence of other causes of renal dysfunction, minimal hematuria, and lack of improvement with volume resuscitation. It occurs due to splanchnic arterial dilation, decreased vascular resistance, and local renal vasoconstriction with decreased perfusion.

References

- Hepatorenal syndrome: aetiology, diagnosis, and treatment.
- Hepatorenal syndrome: update on diagnosis and treatment.

An 82-year-old man is found to be agitated and confused on day 2 following surgery for a right hip fracture. The surgery was uncomplicated and was not accompanied by hemodynamic instability. The patient has a history of mild cognitive impairment and lives at home with his daughter. He also has hypertension, gout, and benign prostatic hyperplasia. Temperature is 37.2 C (99 F), blood pressure is 134/90 mm Hg, and pulse is 100/min. The patient has 2 peripheral intravenous lines that appear clean. He does not have a Foley catheter. Lungs are clear on auscultation. Heart rhythm is regular with a faint mid-systolic murmur heard over the left sternal border. The lower abdomen is tender and palpation exacerbates the agitation. The surgical site appears clean with minimal discharge. Laboratory results are as follows:

Complete blood count	
Hemoglobin	10.5 g/dL
Leukocytes	10,000/mm^3
Platelets	155,000/mm^3
Serum chemistry	
Sodium	132 mEq/L
Potassium	3.6 mEq/L
Creatinine	1.2 mg/dL

Which of the following is the best next step in management of this patient?

A. Abdominal CT scan without contrast [15%]

B. Abdominal x-rays [8%]

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Which of the following is the best next step in management of this patient?

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B. Abdominal x-rays
C. Bladder ultrasound
D. Blood cultures followed by intravenous vancomycin
E. Quiet environment and intramuscular haloperidol

Submit

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Which of the following is the best next step in management of this patient?

- A. Abdominal CT scan without contrast [15%]
- B. Abdominal x-rays [8%]
- ✔ C. Bladder ultrasound [59%]
- D. Blood cultures followed by intravenous vancomycin [6%]
- E. Quiet environment and intramuscular haloperidol [10%]

Omitted
Correct answer
C

59%
Answered correctly

3 Seconds
Time Spent

08/24/2018
Last Updated

Explanation

This elderly male patient with **agitation**, tachycardia, and **lower abdominal (suprapubic) tenderness** 2 days following surgical repair of a hip fracture likely has **acute urinary retention (AUR)**. The major risk factors for development of AUR include:

- **Male** sex (AUR rarely occurs in women)
- Advanced **age** (~33% of men age >80 will develop AUR)
- History of **benign prostatic hyperplasia**
- **History of neurologic disease** (eg, mild cognitive impairment)
- **Surgery** (especially abdominal surgery, pelvic surgery, and joint arthroplasty)

It is likely that this patient has also recently taken medications (eg, anesthetics, **opioids**, **anticholinergics**) that are common precipitants of AUR.

In a patient with suspected AUR who is unable to void, the diagnosis is confirmed by **bladder ultrasound** demonstrating **≥300 mL of urine**. Treatment is with insertion of a **Foley catheter**, and urinalysis should be collected to rule out urinary tract infection (a potential cause of AUR). Obesity, abdominal ascites, or tissue edema may render bladder ultrasound inaccurate; in such cases, Foley catheter insertion can be both diagnostic and therapeutic.

(Choices A and B) CT scan and x-rays of the abdomen are often helpful in diagnosing causes of acute abdominal pain (eg, colitis, nephrolithiasis, appendicitis). However, AUR should be suspected first in this patient with numerous risk factors.

(Choice D) Blood cultures followed by intravenous vancomycin would be appropriate for treatment of suspected gram-positive bacteremia, which may present with confusion and tachycardia. This patient's clean surgical site, lower abdominal tenderness, and absence of fever and leukocytosis make AUR more likely.

(Choice E) A quiet environment may help improve delirium, which is a common cause of agitation and confusion in hospitalized

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(Choice D) Blood cultures followed by intravenous vancomycin would be appropriate for treatment of suspected gram-positive bacteremia, which may present with confusion and tachycardia. This patient's clean surgical site, lower abdominal tenderness, and absence of fever and leukocytosis make AUR more likely.

(Choice E) A quiet environment may help improve delirium, which is a common cause of agitation and confusion in hospitalized elderly patients. However, AUR commonly causes confusion and agitation and should be suspected first in this patient with numerous risk factors. If possible, the use of haloperidol and other typical antipsychotics should be avoided in elderly patients.

Educational objective:
Acute urinary retention (AUR) is common in elderly men, especially in the setting of underlying benign prostatic hyperplasia. The risk of AUR is further increased during the postoperative period. Diagnosis is made using bladder ultrasound.

References

- Risk factors for postoperative urinary retention in men undergoing total hip arthroplasty.

A 56-year-old diabetic male is brought to the emergency department due to nausea and vomiting. While you are examining the patient, a nurse asks if determination of the patient's acid-base status will help ascertain the etiology and subsequent management of the patient's primary problem. Which of the following pairs of laboratory values will help get the best picture of the patient's acid-base status?

- A. pH and PaCO2
- B. pH and PaO2
- C. PaO2 and PaCO2
- D. Urinary pH and PaCO2
- E. Urinary pH and HCO3-

Submit

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- ✔ A. pH and PaCO2 [88%]
- B. pH and PaO2 [1%]
- C. PaO2 and PaCO2 [0%]
- D. Urinary pH and PaCO2 [1%]
- E. Urinary pH and HCO3- [7%]

Omitted
Correct answer
A

88%
Answered correctly

3 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

The major extracellular buffer in human blood is the carbon dioxide - bicarbonate buffer pair, which has a pK of 6.1. Acid-base disturbances in patients can be diagnosed by assessing the pH and the changes in either the serum bicarbonate concentration or the serum partial pressure of carbon dioxide. Classically, the acid-base status of a buffer can be determined using the Henderson-Hasselbalch equation with its three variables, pH, [acid] and [conjugate base], as follows:

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$$pH = pK + \log([\text{conjugate base}] / [\text{acid}])$$

In the case of the carbon dioxide - bicarbonate buffer pair, where CO_2 is the acid and HCO_3^- is the conjugate base, the equation is as follows:

$$pH = 6.1 + \log([HCO_3^-]/(0.03 \times PaCO_2))$$

Using the Henderson-Hasselbalch equation, any of the three variables can be calculated if the other two are given. Thus, with respect to the above question, if the pH and $PaCO_2$ are given **(Choice A)**, the HCO_3^- can be calculated and with these three data points the acid-base status of the patient can be determined.

(Choices B, C, D and E) The PO_2 gives information about a patient's oxygenation status. The urinary pH, which can vary widely between 4.5 and 8, can give information about renal handling of acids and bases, but it can not be used to determine a patient's serum acid-base status with the same clarity that pH, HCO_3^- and $PaCO_2$ can.

Educational objective:
In any patient, the pH and $PaCO_2$ are the two lab values that provide the best picture of acid-base status; the HCO_3^- can be calculated from these values using the Henderson-Hasselbalch equation.

A 45-year-old woman is brought to the physician after she was found to have nausea, fever, and upper abdominal discomfort. Her medical problems include severe depression, migraine, and rheumatoid arthritis. The patient describes severe tinnitus and vertigo. She admits that she overdosed on one of her medications several hours ago. Her temperature is 38.1 C (100.5 F), blood pressure is 120/76 mm Hg, pulse is 115/min, and respirations are 26/min. Physical examination is otherwise unremarkable. Which of the following acid-base findings is most likely to be present on arterial blood gas analysis?

	pH	$PaCO_2$ (mm Hg)	HCO_3^- (mEq/L)
A.	7.30	27	13
B.	7.32	50	25
C.	7.39	20	12
D.	7.40	40	24
E.	7.42	54	32

Submit

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	pH	$PaCO_2$ (mm Hg)	HCO_3^- (mEq/L)
A. [42%]	7.30	27	13
B. [7%]	7.32	50	25
✔ C. [41%]	7.39	20	12
D. [4%]	7.40	40	24
E. [4%]	7.42	54	32

Omitted
Correct answer
C

41%
Answered correctly

3 Seconds
Time Spent

09/23/2018
Last Updated

Explanation

This patient has **tinnitus, fever, tachypnea**, nausea, and gastrointestinal irritation after an overdose of one of her medications. This scenario is highly suggestive of **salicylate intoxication** (eg, due to **aspirin** taken for rheumatoid arthritis or migraines). Acute salicylate toxicity leads to **respiratory alkalosis** by stimulating the respiratory center in the medulla and causing tachypnea (with resultant low $PaCO_2$ as the CO_2 is blown off). It then causes an **anion gap metabolic acidosis** by uncoupling of oxidative phosphorylation in the mitochondria leading to anaerobic metabolism (with resultant low HCO_3^- from acid buildup).

Therefore, the arterial blood gas (ABG) in salicylate toxicity is most likely to show:

- Low $PaCO_2$: due to primary respiratory alkalosis (and respiratory compensation for metabolic acidosis)
- Low HCO_3^-: due to primary metabolic acidosis (and metabolic compensation for respiratory alkalosis)
- Near-normal arterial pH: as the 2 primary acid-base disturbances shift the pH in opposite directions

This patient's ABG is most likely to show a **near-normal pH** (eg, pH = 7.39) with **mixed** respiratory alkalosis and metabolic acidosis. The low HCO_3^- in the corresponding answer choice (HCO_3^- = 12 mEq/L) suggests a metabolic acidosis. Based on the corresponding formula for respiratory compensation (**Winter's formula**), the expected $PaCO_2 = [1.5 * HCO_3^-] + 8 \pm 2 = [1.5 * 12] + 8 \pm 2 = 26 \pm 2$ mm Hg. Because the observed $PaCO_2$ (20 mm Hg) is lower than the expected value (26 ± 2 mm Hg), there is a coexisting primary respiratory alkalosis.

A common mistake in management of salicylate toxicity is to find a pH that is close to 7.4 and not recognize a mixed-acid base disorder. As a result, treatment (alkalinization or dialysis) is delayed as it may seem that the patient is compensating adequately for the acidosis. Over time, however, as this metabolic acidosis worsens, the patient will reach a point at which he or she will be unable to ventilate quickly enough to compensate.

(Choice A) Acidemia (pH 7.30) with a low HCO_3^- (13 mEq/L) suggests primary metabolic acidosis, accompanied by a compensatory decrease in $PaCO_2$ (27 mm Hg) that is adequate in this case ($PaCO_2 = [1.5 * HCO_3^-] + 8 \pm 2 = [1.5 * 13] + 8 \pm 2 = 27.5 \pm 2$ mm Hg).

(Choice A) Acidemia (pH 7.30) with a low HCO_3^- (13 mEq/L) suggests primary metabolic acidosis, accompanied by a compensatory decrease in $PaCO_2$ (27 mm Hg) that is adequate in this case ($PaCO_2 = [1.5 * HCO_3^-] + 8 \pm 2 = [1.5 * 13] + 8 \pm 2 = 27.5 \pm 2$ mm Hg). Primary metabolic acidosis alone is rarely seen in adults with aspirin toxicity, and would be unlikely in this patient several hours after ingestion.

(Choice B) Acidemia (pH 7.32) with a high $PaCO_2$ (50 mm Hg) suggests respiratory acidosis. Metabolic compensation (HCO_3^- 25 mEq/L) is adequate ($\Delta HCO_3^- = 0.1 * \Delta PaCO_2 = 0.1 * 10 = 1$ mEq/L, assuming normal $PaCO_2$ of 40 mm Hg).

(Choice D) This ABG reflects normal acid-base status.

(Choice E) A normal pH in an acid-base disturbance (ie, abnormal $PaCO_2$ and HCO_3^-) typically signifies a **mixed** respiratory and metabolic disorder. In this case, the high HCO_3^- (32 mEq/L) suggests a metabolic alkalosis; the $PaCO_2$ (54 mm Hg) is higher than that expected with respiratory compensation so there is a concomitant respiratory acidosis. The computation for the expected $PaCO_2$ is as follows: $\Delta PaCO_2 = 0.7 * \Delta HCO_3^- = 0.7 * 8 = 5.6$ mm Hg (assuming normal HCO_3^- of 24 mEq/L). As the normal $PaCO_2$ range is given as 33-45 mm Hg, expected $PaCO_2$ is 39-51 mm Hg. This is mixed metabolic alkalosis and respiratory acidosis. Starting with respiratory acidosis and computing the expected compensation would lead to the same conclusion.

Educational objective:
Aspirin intoxication should be suspected in a patient with the triad of fever, tinnitus, and tachypnea. Adults with aspirin toxicity develop a mixed respiratory alkalosis and anion gap metabolic acidosis. A normal pH in an acid-base disturbance typically signifies a mixed respiratory and metabolic acid-base disorder.

References

- Emergency department management of the salicylate-poisoned patient.

Therefore, the arterial blood gas (ABG) in salicylate toxicity is most likely to show:

- Low PaC
- Low HC
- Near-nor

This patient's A
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(Choice A) Ac
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(Choice B) Ac
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(Choice D) Th

(Choice E) A
metabolic disorder. In this case, the high HCO$_3^-$ (22 mEq/L) suggests a metabolic alkalosis; the PaCO$_2$ (54 mm Hg) is higher than that

Exhibit Display

Acid-base disorders	
Primary disorder	**Appropriate compensation**
Metabolic acidosis	$PaCO_2 = 1.5$ (serum HCO_3) + 8 ± 2
Metabolic alkalosis	↑ $PaCO_2$ by 0.7 mm Hg for every 1 mEq/L rise in serum HCO_3
Acute respiratory acidosis	↑ Serum HCO_3 by 1 mEq/L for every 10 mm Hg rise in $PaCO_2$
Acute respiratory alkalosis	↓ Serum HCO_3 by 2 mEq/L for every 10 mm Hg decrease in $PaCO_2$

Add To Flash Card

A 35-year-old African American man comes to the physician complaining of 2 weeks of abdominal distension. He has been in a drug rehabilitation program for 2 years due to prior heroin abuse. Recent viral hepatitis and HIV profiles were negative. His temperature is 37.1 C (98.7 F), blood pressure is 145/82 mm Hg, pulse is 80/min, and respirations are 14/min. The patient is obese with a body mass index of 40 kg/m^2. Examination shows periorbital edema, ascites, and 2+ pitting edema in both legs up to the knees. Breath sounds are decreased at the right lung base. Twenty-four-hour urine shows protein excretion of 7.5 g. Which of the following is most likely to be present on kidney biopsy?

- A. Focal segmental glomerulosclerosis
- B. IgA nephropathy
- C. Membranous nephropathy
- D. Minimal change disease
- E. Systemic amyloidosis

Submit

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- ✔ A. Focal segmental glomerulosclerosis [70%]
- B. IgA nephropathy [1%]
- C. Membranous nephropathy [22%]
- D. Minimal change disease [1%]
- E. Systemic amyloidosis [3%]

Omitted
Correct answer
A

70%
Answered correctly

3 Seconds
Time Spent

12/01/2018
Last Updated

Explanation

Primary renal causes of nephrotic syndrome

Primary renal causes of nephrotic syndrome	
Etiology	**Clinical associations**
Focal segmental glomerulosclerosis	African American & Hispanic ethnicity; obesity; HIV & heroin use
Membranous nephropathy	Adenocarcinoma (eg, breast, lung); nonsteroidal antiinflammatory drugs (NSAIDs); hepatitis B; systemic lupus erythematosus
Membranoproliferative glomerulonephritis	Hepatitis B & C; lipodystrophy
Minimal change disease	NSAIDs; lymphoma
IgA nephropathy	Upper respiratory tract infection

This patient's presentation is consistent with nephrotic syndrome, defined as heavy proteinuria (>3.5 g/24 hr) with hypoalbuminemia and edema. Other findings include hyperlipidemia and increased lipids in the urine. Causes of nephrotic syndrome in adults include primary glomerular (eg, membranous nephropathy, focal segmental glomerulosclerosis [FSGS], and minimal change disease) and secondary glomerular (eg, diabetes mellitus, amyloidosis) diseases.

This patient has volume overload (periorbital edema, peripheral edema, and ascites) likely due to hypoalbuminemia. His pulmonary examination suggests a likely right pleural effusion. Because he does not have known diabetes or amyloidosis, he most likely has a

secondary glomerular (eg, diabetes mellitus, amyloidosis) diseases.

This patient has volume overload (periorbital edema, peripheral edema, and ascites) likely due to hypoalbuminemia. His pulmonary examination suggests a likely right pleural effusion. Because he does not have known diabetes or amyloidosis, he most likely has a primary renal etiology. Recent United States registry data indicate that FSGS is the most common cause of nephrotic syndrome in adults, especially in African Americans. **FSGS** can also be associated with **HIV**, **heroin** use, and morbid **obesity**.

(Choice B) IgA nephropathy can present with nephrotic syndrome (<10%), but more commonly presents with hematuria following an upper respiratory infection (about 40%-50% of patients).

(Choice C) Membranous nephropathy is the second leading cause of nephrotic syndrome in the overall population. FSGS is statistically more likely in this patient given that he is African American with obesity and heroin use.

(Choice D) Minimal change disease is more common among children. In adults, it is associated with use of nonsteroidal anti-inflammatory drugs and lymphoma (usually Hodgkin).

(Choice E) Amyloidosis is usually associated with multiple myeloma or a chronic inflammatory disease (eg, rheumatoid arthritis, bronchiectasis).

Educational objective:
Nephrotic syndrome is defined as heavy proteinuria (>3.5 g/24 hr) with hypoalbuminemia and edema. Focal segmental glomerulosclerosis (FSGS) and membranous nephropathy are the most common causes of nephrotic syndrome in adults in the absence of a systemic disease. FSGS is more common in African American patients and in those with obesity, heroin use, and HIV.

References

- Changing incidence of glomerular disease in Olmsted County, Minnesota: a 30-year renal biopsy study.
- Focal segmental glomerulosclerosis in African Americans.

A 4-year-old boy is brought to the physician for painful urination and fever. For the past 2 days, the child has had pain with urination and has urinated more frequently than usual. The patient's parents say that he awoke twice overnight to urinate and woke up this morning with a fever. He has a history of a urinary tract infection at age 23 months, and a renal ultrasound at that time was normal. Temperature is 38.9 C (102 F), blood pressure is 102/48 mm Hg, and pulse is 104/min. The patient's abdominal examination shows mild suprapubic tenderness with no rebound or guarding.

Urinalysis results are as follows:

Specific gravity	1.016
pH	6.9
Protein	None
Blood	Trace
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Positive
Nitrites	Positive
Bacteria	Few
White blood cells	20-30/hpf

A voiding cystourethrogram demonstrates moderate (Grade III) vesicoureteral reflux. His symptoms improve with antibiotic therapy. What is the most likely long-term complication of this patient's vesicoureteral reflux if left untreated?

Protein	None
Blood	Trace
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Positive
Nitrites	Positive
Bacteria	Few
White blood cells	20-30/hpf

A voiding cystourethrogram demonstrates moderate (Grade III) vesicoureteral reflux. His symptoms improve with antibiotic therapy. What is the most likely long-term complication of this patient's vesicoureteral reflux if left untreated?

- A. Bladder cancer
- B. Renal abscess
- C. Renal calculi
- D. Renal cell carcinoma
- E. Renal scarring

Submit

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White blood cells	20-30/hpf

A voiding cystourethrogram demonstrates moderate (Grade III) vesicoureteral reflux. His symptoms improve with antibiotic therapy. What is the most likely long-term complication of this patient's vesicoureteral reflux if left untreated?

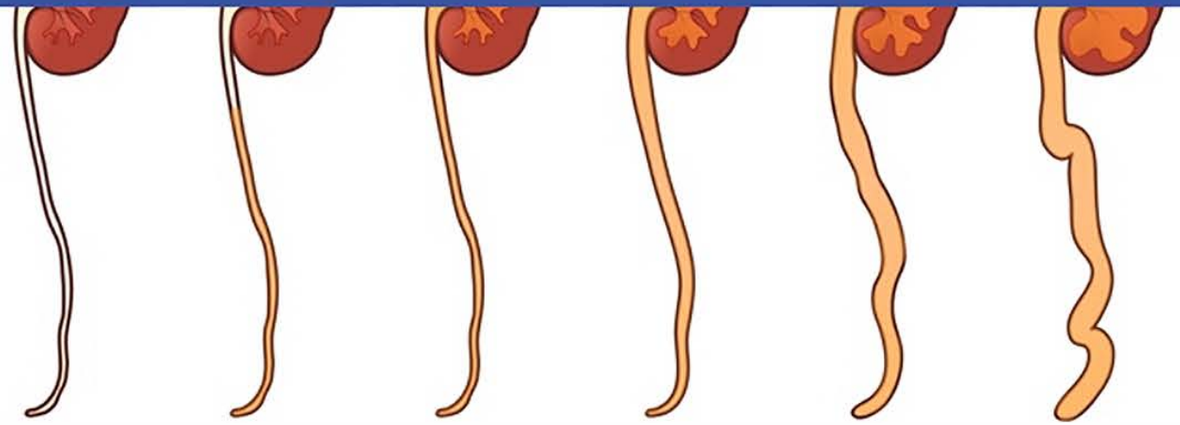
A. Bladder cancer [1%]
B. Renal abscess [7%]
C. Renal calculi [4%]
D. Renal cell carcinoma [1%]
✔ E. Renal scarring [85%]

Omitted
Correct answer
E

85%
Answered correctly

3 Seconds
Time Spent

12/10/2018
Last Updated



Normal Grade I Grade II Grade III Grade IV Grade V

Grade	Description
I	Into a nondilated ureter
II	Into the pelvis & calyces without dilation
III	Mild to moderate dilation of the ureter, renal pelvis & calyces, with minimal blunting of the fornices
IV	Moderate ureteral tortuosity & dilation of the pelvis & calyces
V	Gross dilation of the ureter, pelvis & calyces; loss of papillary impressions; ureteral tortuosity

Vesicoureteral reflux (VUR) is defined as the **retrograde flow of urine** from the bladder up into the ureter and renal pelvis. VUR is the most common pediatric urologic problem and is present in ~30%-45% of children with urinary tract infections (UTI). It is a risk

Vesicoureteral reflux (VUR) is defined as the **retrograde flow of urine** from the bladder up into the ureter and renal pelvis. VUR is the most common pediatric urologic problem and is present in ~30%-45% of children with urinary tract infections (UTI). It is a risk factor for **recurrent UTIs**, which in turn place a patient at significant risk for **renal scarring**. The growing kidney is particularly prone to scarring, renal insufficiency, and end-stage renal disease.

The gold standard modality for diagnosing VUR is a **voiding cystourethrogram** (VCUG). The American Academy of Pediatrics recommends that children with a first UTI at age 2-24 months undergo **renal and bladder ultrasound** to evaluate for any anatomic abnormalities that might predispose to VUR. Routine VCUGs after a first UTI are not usually recommended, as a normal renal ultrasound is generally reassuring. However, VCUGs are recommended in patients with recurrent UTIs, such as this patient, as a renal ultrasound is less sensitive in detecting VUR.

(Choice A) VUR is not associated with bladder cancer.

(Choice B) Renal abscesses are a short-term complication of acute pyelonephritis, which also increase the risk of permanent renal scarring.

(Choice C) The development of renal calculi is not associated with VUR. Rather, risk factors for renal calculi include a diet that is high in animal protein and sodium and low in calcium and fluid.

(Choice D) Risk factors for the development of renal cell carcinoma include cigarette smoking, hypertension, and diabetes mellitus. VUR does not increase the risk for renal cell carcinoma.

Educational objective:
Vesicoureteral reflux (VUR) is a risk factor for recurrent urinary tract infections (UTIs), which can lead to progressive renal scarring. As such, all children with a first febrile UTI at age 2-24 months should undergo a renal ultrasound to evaluate for anatomic abnormalities. Those with recurrent UTIs should also undergo a voiding cystourethrogram to evaluate for VUR.

References

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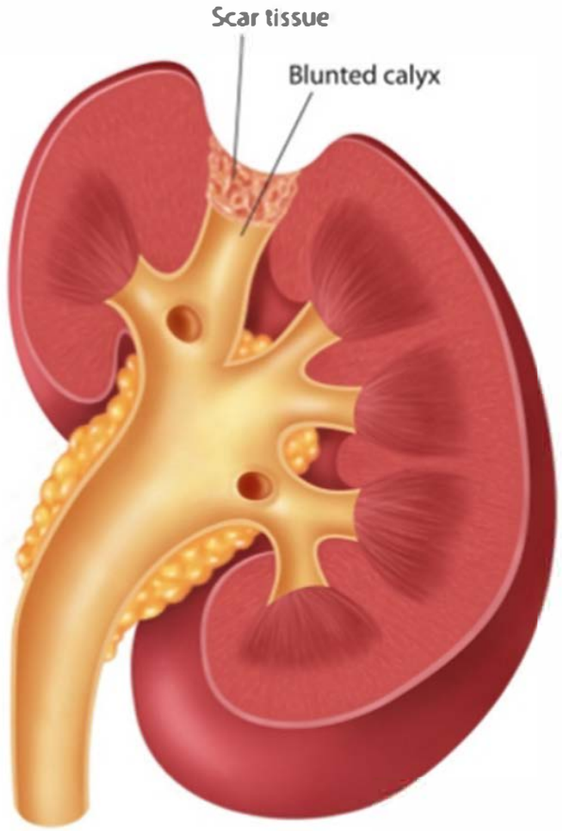
References

Exhibit Display

Chronic pyelonephritis of the kidney



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A 73-year-old man comes to the office due to 3 months of progressive urinary urgency, hesitancy, nocturia, and weak urinary stream. He has no fever, abdominal pain, hematuria, malaise, or weight loss. His only medication is lisinopril for essential hypertension. The patient has no history of diabetes mellitus or ischemic heart disease. He does not use tobacco, alcohol, or illicit drugs. Digital rectal examination reveals a smooth, firm, and enlarged prostate without induration or asymmetry. Neurological examination is normal. Urinalysis shows no proteinuria or hematuria. The patient's serum creatinine is 2.1 mg/dL, which is higher than his baseline creatinine of 1.2 mg/dL 4 months ago. Prostate-specific antigen is normal. Which of the following is the most appropriate next step in evaluation of this patient's acute kidney injury?

- A. Cystoscopy
- B. Kidney biopsy
- C. Prostate biopsy
- D. Renal ultrasound
- E. Urine cytology

Submit

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- A. Cystoscopy [10%]
- B. Kidney biopsy [1%]
- C. Prostate biopsy [5%]
- ✔ D. Renal ultrasound [66%]
- E. Urine cytology [16%]

Omitted
Correct answer
D

66%
Answered correctly

3 Seconds
Time Spent

11/02/2018
Last Updated

Explanation

This patient has **lower urinary tract symptoms** (LUTS) (eg, urinary urgency, hesitancy, nocturia, weak urinary stream) with a smooth,

Explanation

This patient has **lower urinary tract symptoms** (LUTS) (eg, urinary urgency, hesitancy, nocturia, weak urinary stream) with a smooth, enlarged prostate on examination consistent with **benign prostatic hyperplasia** (BPH). Initial evaluation of patients with LUTS should include a urinalysis (to exclude infection and hematuria) and serum prostate-specific antigen (PSA) to assess risk for prostate cancer. A serum creatinine test is not required in the routine evaluation of uncomplicated BPH. However, it is recommended by some expert panels, especially for patients with more significant symptoms or additional risk factors (eg, hypertension, diabetes) for chronic kidney disease.

This patient has evidence of **acute kidney injury**, as indicated by an interval rise in serum creatinine. Creatinine is generally not elevated in unilateral urinary obstruction (eg, ureteral calculus) but can be elevated in bilateral obstruction, such as in patients with severe bladder outlet obstruction due to BPH. Patients with acutely elevated creatinine require imaging (preferably **renal ultrasound**) to assess for hydronephrosis and exclude other causes of obstruction. Placement of a **urinary catheter** in patients with hydronephrosis can provide quick relief of the obstruction.

(Choices A and E) Cystoscopy in patients with BPH can reveal signs of chronic bladder obstruction, but it is nonspecific and usually reserved for those who have failed initial management. It is also used to visualize the lower urinary tract in patients with hematuria, usually in combination with imaging of the upper tract (eg, CT scan of the kidneys and ureters). Urine cytology is sometimes performed in place of cystoscopy for low-risk patients with hematuria. Cytology can also be done for patients with LUTS and additional risk factors for bladder cancer (eg, smoking).

(Choice B) Kidney biopsy is used to diagnose intrinsic renal causes of acute kidney injury. This patient's presentation is more consistent with obstructive (post-renal) acute kidney injury.

(Choice C) Prostate biopsy is indicated for patients with signs of prostate cancer, such as grossly asymmetric enlargement of the prostate, palpable nodules, or persistently elevated PSA levels >4 ng/dL.

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(Choice C) Prostate biopsy is indicated for patients with signs of prostate cancer, such as grossly asymmetric enlargement of the prostate, palpable nodules, or persistently elevated PSA levels >4 ng/dL.

Educational objective:
Patients with severe bladder outlet obstruction due to benign prostatic hyperplasia can develop acute kidney injury. A renal ultrasound is advised for assessment of hydronephrosis in those with worsening kidney function.

References

- Clinical utility of gray scale renal ultrasound in acute kidney injury.

A 59-year-old woman is hospitalized due to lower gastrointestinal bleeding. Her past medical history includes hypertension, chronic obstructive pulmonary disease with cor pulmonale, and diverticulosis. She takes albuterol, aspirin, furosemide, and lisinopril. On admission, her blood pressure was 80/50 mm Hg and hemoglobin was 6.5 g/dL. Her condition stabilized with fluid resuscitation, blood transfusions, and by withholding her home medications. The patient's urine output has been 300-400 mL per day for the past 3 days. On the fourth day of hospitalization, she is found lethargic and difficult to arouse. Her blood pressure is 110/70 mm Hg and pulse oximetry is 93% on 6 L per minute oxygen. Laboratory testing reveals:

Hemoglobin	10.2 g/dL
Leukocytes	14,300/mm^3
Blood glucose	93 mg/dL
Blood urea nitrogen	62 mg/dL
Creatinine	2.7 mg/dL
Sodium	132 mEq/L
Potassium	5.0 mEq/L
Chloride	102 mEq/L
Bicarbonate	18 mEq/L
Arterial blood gases	
pH	7.15
PaO_2	80 mm Hg

Potassium	5.0 mEq/L
Chloride	102 mEq/L
Bicarbonate	18 mEq/L

Arterial blood gases

pH	7.15
PaO_2	80 mm Hg
$PaCO_2$	60 mm Hg

Which of the following is most likely contributing to this patient's lethargy?

A. Alveolar diffusion block
B. Decreased anion gap
C. Hypoventilation
D. Impaired tubular bicarbonate reabsorption
E. Low tubular ammonia production
F. Renal tubular chloride loss

Submit

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Which of the following is most likely contributing to this patient's lethargy?

- A. Alveolar diffusion block [7%]
- B. Decreased anion gap [1%]
- ✔ C. Hypoventilation [53%]
- D. Impaired tubular bicarbonate reabsorption [8%]
- E. Low tubular ammonia production [27%]
- F. Renal tubular chloride loss [1%]

Omitted
Correct answer
C

53%
Answered correctly

4 Seconds
Time Spent

09/05/2018
Last Updated

Explanation

Explanation

This patient has acidemia (pH <7.35) in the setting of a mixed **acid-base disorder**. Indeed, she has elevated $PaCO_2$ (>40 mm Hg) indicating **respiratory acidosis**; she also has low serum bicarbonate (<24 mEq/L) consistent with **metabolic acidosis**. Acidemia by itself does not lead to central nervous system depression, but often, the underlying cause of acidemia results in lethargy and altered mental status.

In this case, the patient's respiratory acidosis with acute-on-chronic CO_2 retention (hypercarbia) is likely due to hypoventilation in the setting of her underlying chronic obstructive pulmonary disease (COPD), leading to **CO_2 narcosis** (usually seen at $PaCO_2$ >60 mm Hg). In patients with COPD, acute-on-chronic hypercarbia can be differentiated from chronic CO_2 retention by the associated acidosis and low bicarbonate level (chronic CO_2 retainers have normal pH and high serum bicarbonate).

This patient also has **acute kidney injury (AKI)** most likely due to hypotension during her gastrointestinal (GI) bleed. AKI can cause a non-anion gap metabolic acidosis from impaired H^+ excretion, ammonia generation, or bicarbonate reabsorption. Her anion gap (serum $[Na^+] - [Cl^- + HCO_3^-]$) is 12 mEq/L, indicating a **non-anion gap metabolic acidosis** (normal 6-12 mEq/L, varies by laboratory) **(Choice B)**. AKI can also cause an anion gap acidosis due to retention of unmeasured uremic toxins, which can also cause encephalopathy; however, in this patient, the normal anion gap suggests that the elevated blood urea nitrogen is due to her GI bleed (ie, metabolism of blood proteins to urea) and there is not likely to be an excessive concentration of other unmeasured uremic toxins. Her mental status changes are therefore less likely to be due directly to her AKI.

(Choice A) A decrease in alveolar diffusing capacity is characterized by hypoxia and tachypnea. Tachypnea would cause lowering of the pCO_2 because CO_2 is more readily diffusible than O_2.

(Choice D) Defective tubular bicarbonate reabsorption in the proximal tubules is the defect in type 2 renal tubular acidosis (RTA) also known as proximal RTA. Type 2 RTA is often times inherited and may be a component of Fanconi syndrome. Type 2 RTA can also occur with use of carbonic anhydrase inhibitors (acetazolamide).

(Choice E) Hepatic encephalopathy is associated with elevated levels of ammonia in the blood. Although impaired generation of

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(Choice D) Defective tubular bicarbonate reabsorption in the proximal tubules is the defect in type 2 renal tubular acidosis (RTA) also known as proximal RTA. Type 2 RTA is often times inherited and may be a component of Fanconi syndrome. Type 2 RTA can also occur with use of carbonic anhydrase inhibitors (acetazolamide).

(Choice E) Hepatic encephalopathy is associated with elevated levels of ammonia in the blood. Although impaired generation of ammonia in AKI decreases renal acid excretion, it does not raise blood ammonia levels or cause changes in mental status.

(Choice F) Renal tubular chloride loss as seen in aggressive loop or thiazide diuretic use causes metabolic alkalosis, not acidosis. This is due to loss of extracellular volume that accompanies chloride loss, thus stimulating renin-angiotensin-aldosterone activity and increasing urinary excretion of H^+.

Educational objective:
Alveolar hypoventilation with CO_2 retention can lead to respiratory acidosis along with CO_2 narcosis. Acute kidney injury can cause a

Explanation

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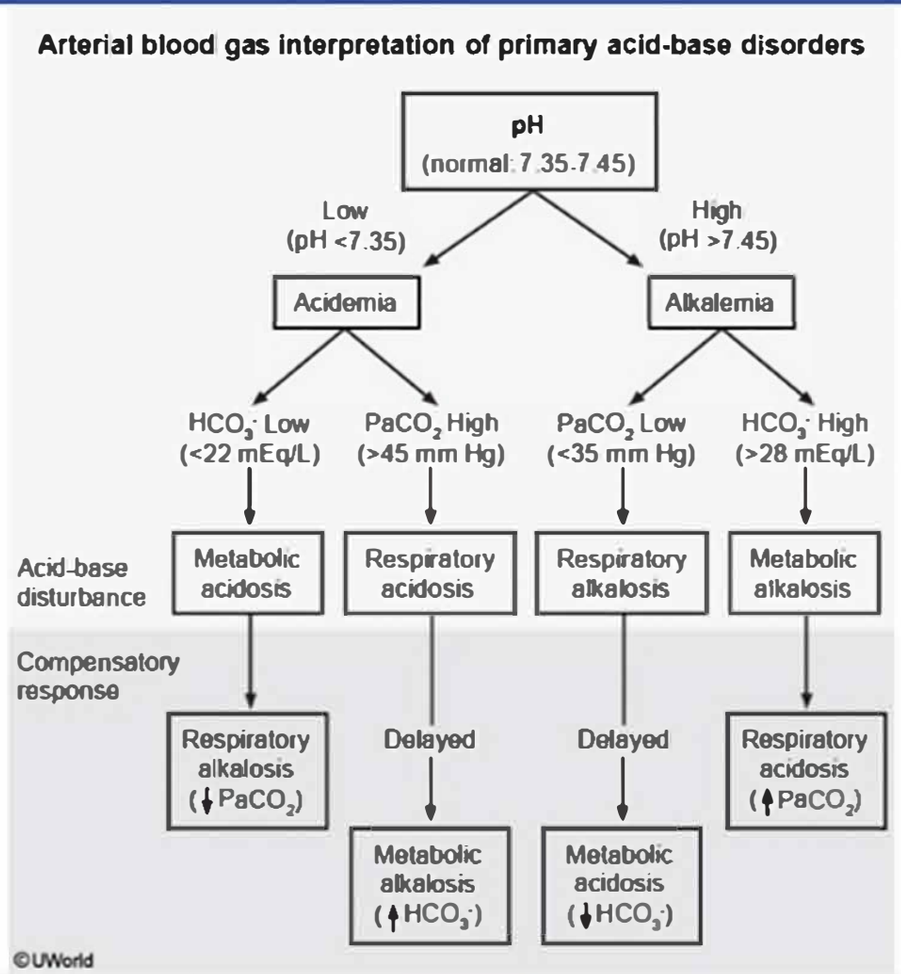

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A 50-year-old man comes to the physician because of a skin rash, joint pains, malaise and fatigue. He has a history of intravenous drug abuse. His temperature is 37.1° C (98.9° F), blood pressure is 140/90 mm Hg, pulse is 80/min, and respirations are 14/min. Examination shows palpable purpura and hepatosplenomegaly. Urinalysis shows hematuria, red blood cell casts and proteinuria. The results of the laboratory studies are as follows:

BUN	30 mg/dL
Creatinine	2.0 mg/dL
Serum complement	Low
Anti-HCV	Positive

Which of the following is the most likely diagnosis?

A. Alport's syndrome
B. Acute interstitial nephritis
C. Acute post infectious glomerulonephritis
D. Anti-glomerular basement membrane disease
E. Benign recurrent hematuria
F. Goodpasture's syndrome
G. Henoch-Schonlein purpura
H. Idiopathic anti-GBM antibody mediated glomerulonephritis

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- H. Idiopathic anti-GBM antibody mediated glomerulonephritis
- I. IgA nephropathy
- J. Mixed essential cryoglobulinemia
- K. Microscopic polyangiitis
- L. Systemic lupus erythematosus
- M. Thin basement membrane disease
- N. Wegener's granulomatosis

Submit

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BUN	30 mg/dL
Creatinine	2.0 mg/dL
Serum complement	Low
Anti-HCV	Positive

Which of the following is the most likely diagnosis?

A. Alport's syndrome [1%]
B. Acute interstitial nephritis [3%]
C. Acute post infectious glomerulonephritis [7%]
D. Anti-glomerular basement membrane disease [4%]
E. Benign recurrent hematuria [0%]
F. Goodpasture's syndrome [1%]
G. Henoch-Schonlein purpura [20%]
H. Idiopathic anti-GBM antibody mediated glomerulonephritis [2%]

A. Alport's syndrome [1%]
B. Acute interstitial nephritis [3%]
C. Acute post infectious glomerulonephritis [7%]
D. Anti-glomerular basement membrane disease [4%]
E. Benign recurrent hematuria [0%]
F. Goodpasture's syndrome [1%]
G. Henoch-Schonlein purpura [20%]
H. Idiopathic anti-GBM antibody mediated glomerulonephritis [2%]
I. IgA nephropathy [4%]
✔ J. Mixed essential cryoglobulinemia [39%]
K. Microscopic polyangiitis [6%]
L. Systemic lupus erythematosus [3%]
M. Thin basement membrane disease [1%]
N. Wegener's granulomatosis [1%]

Omitted
Correct answer
J

39%
Answered correctly

6 Seconds
Time Spent

08/09/2018
Last Updated

Omitted
Correct answer
J

39%
Answered correctly

6 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Suspect mixed cryoglobulinemia in a patient who presents with palpable purpura, proteinuria and hematuria. Other suggestive clinical manifestations include nonspecific systemic symptoms, arthralgias, hepatosplenomegaly and hypocomplementemia. The demonstration of circulating cryoglobulins is confirmatory. Majority of patients have an underlying HCV infection. For this reason, all such patients should be tested for HCV antibodies.

(Choice G) Henoch-Schonlein purpura usually presents in childhood as palpable purpura on the buttocks, abdominal pain, arthralgias, proteinuria and hematuria with RBC casts on urinalysis. Serum complement levels are normal. HCV infection is not associated with this disease.

(Choice L) SLE usually occurs in young adult females. Skin manifestations include malar or discoid rash. Serology is positive for anti-nuclear antibodies; anti-DNA and Anti-Sm antibodies are very specific for SLE. Renal involvement is quite common.

(Choice K) Microscopic polyangiitis usually presents with constitutional symptoms of fever and malaise. Other features may include abdominal pain and hematuria with active urinary sediment and purpura. Serology is usually negative, except for ANCAs; serum complement levels are normal.

Educational Objective:
The common presentation of cryoglobulinemia includes palpable purpura, glomerulonephritis, non-specific systemic symptoms, arthralgias, hepatosplenomegaly, peripheral neuropathy, and hypocomplementemia. Most patients also have Hepatitis C.

A 66-year-old man comes to the physician for regular follow-up. He experiences mild fatigue but otherwise has no complaints. The patient has a history of hypertension, type 2 diabetes mellitus, hyperlipidemia, chronic kidney disease, and ischemic cardiomyopathy. His temperature is 36.7 C (98 F), blood pressure is 122/70 mm Hg, and pulse is 82/min. Physical examination shows moist mucous membranes. There is a 2/6 holosystolic murmur at the apex that is unchanged from a previous finding. He has trace bilateral lower-extremity edema. Laboratory results are as follows:

Sodium	135 mEq/L
Potassium	6.0 mEq/L
Bicarbonate	24 mEq/L
Blood urea nitrogen	38 mg/dL
Creatinine	2.4 mg/dL
Calcium	8.2 mg/dL
Glucose	196 mg/dL

Two months ago, his creatinine was 2.3 mg/dL. Electrocardiogram shows normal sinus rhythm without other abnormalities. Which of the following is the best next step in management of this patient?

A. Administer calcium gluconate
B. Measure serum renin and aldosterone levels
C. Obtain serum ketone levels
D. Obtain urinalysis and urine electrolytes

Sodium	135 mEq/L
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E. Review current medications

Submit

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A. Administer calcium gluconate [19%]
B. Measure serum renin and aldosterone levels [3%]
C. Obtain serum ketone levels [1%]
D. Obtain urinalysis and urine electrolytes [6%]

Potassium	6.0 mEq/L
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- B. Measure serum renin and aldosterone levels [3%]
- C. Obtain serum ketone levels [1%]
- D. Obtain urinalysis and urine electrolytes [6%]
- ✔ E. Review current medications [69%]

Omitted
Correct answer
E

69%
Answered correctly

4 Seconds
Time Spent

07/22/2018
Last Updated

Explanation

Medications that can cause hyperkalemia	
Medication	**Mechanism**
Nonselective beta-adrenergic blockers	Inhibit beta-2–mediated intracellular potassium uptake
ACE inhibitors	Inhibit angiotensin II formation, leading to decreased aldosterone secretion
ARBs	Inhibit AT_1 receptor, leading to decreased aldosterone secretion
K^+-sparing diuretics	Inhibit ENaC or aldosterone receptor
Cardiac glycosides (eg, digoxin)	Inhibit the Na^+/K^+-ATPase pump
NSAIDs	Inhibit local prostaglandin synthesis, leading to decreased renin & aldosterone secretion

ARBs = angiotensin II receptor blockers; **AT_1** = angiotensin II type 1; **ENaC** = epithelial sodium channel; **Na^+/K^+-ATPase** = sodium/potassium adenosine triphosphatase; **NSAIDs** = nonsteroidal anti-inflammatory drugs.

This patient has hyperkalemia (K^+ >5.0 mEq/L), which is most often due to decreased urinary potassium excretion. The most common etiologies of hyperkalemia are acute or chronic kidney disease and medications or disorders that impair the renin-angiotensin axis. Other etiologies include increased potassium movement out of cells (eg, uncontrolled hyperglycemia, metabolic acidosis) or increased tissue catabolism (eg, trauma, tumor lysis syndrome). Patients with chronic hyperkalemia may be asymptomatic until the potassium gradually rises ≥7.0 mEq/L. However, acute hyperkalemia can cause symptoms at lower levels. Patients may develop ascending

sodium/potassium adenosine triphosphatase; **NSAIDs** = nonsteroidal anti-inflammatory drugs.

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Initial evaluation of hyperkalemia includes ECG to evaluate for conduction abnormalities. Acute therapy (eg, calcium gluconate, insulin with glucose) is typically reserved for patients with ECG changes, potassium ≥7.0 mEq/L without characteristic ECG changes, or rapidly rising potassium due to tissue breakdown. However, this patient has a normal ECG and does not require calcium gluconate **(Choice A)**. The next step is to exclude acute treatable secondary causes (eg, uncontrolled hyperglycemia, tumor lysis syndrome). Patients should then have a review of recent/current medications as they frequently can cause hyperkalemia. Common offending medications include nonselective beta-adrenergic blockers, potassium-sparing diuretics (eg, triamterene), angiotensin-converting-enzyme inhibitors, angiotensin II receptor blockers, and nonsteroidal anti-inflammatory drugs.

(Choice B) Patients with persistent, unexplained hyperkalemia should be evaluated further for hypoaldosteronism with serum renin and aldosterone levels. However, this patient should first have a review of his medications.

(Choice C) Uncontrolled hyperglycemia due to insulin deficiency can shift potassium out of cells to cause hyperkalemia. However, these patients tend to have significant hyperglycemia (>300 mg/dL) and anion gap metabolic acidosis due to ketoacidosis. This patient's absence of a low bicarbonate or significant hyperglycemia makes this less likely.

(Choice D) Urinalysis has limited value for evaluating hyperkalemia. However, urine electrolytes can help differentiate renal from extra-renal causes of hyperkalemia and would be indicated if no offending medications are found.

Educational objective:

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Educational objective:
The most common causes of hyperkalemia include acute or chronic kidney disease, medications, or disorders impairing the renin-angiotensin axis. Common offending medications include nonselective beta-adrenergic blockers, potassium-sparing diuretics (eg, triamterene), angiotensin-converting-enzyme inhibitors, angiotensin II receptor blockers, and nonsteroidal anti-inflammatory drugs.

References

- Management of hyperkalemia.
- Hyperkalemia.

A 55-year-old man is being evaluated for fatigue. He has a history of hypertension and chronic low back pain due to a traumatic injury 20 years ago. His medications include lisinopril and over-the-counter pain medications. His father has diabetes and his mother has hypertension. He does not use tobacco, alcohol, or illicit drugs. His blood pressure is 130/80 mm Hg and pulse is 80/min. Physical examination is unremarkable. Laboratory studies are as follows:

Urinalysis	
Specific gravity	1.013
pH	6.2
Protein	Trace
Blood	Moderate
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Negative
Nitrites	Negative
Bacteria	None
White blood cells	20-30/hpf
Red blood cells	2-3/hpf
Casts	White blood cells

Casts	White blood cells
Crystals	None

Laboratory studies show serum creatinine level of 2.2 mg/dL and blood urea nitrogen of 34 mg/dL. Which of the following is the most likely cause of this patient's urinary findings?

- A. Glomerulonephritis
- B. Nephrolithiasis
- C. Tubulointerstitial nephritis
- D. Urinary tract infection
- E. Urothelial malignancy

Submit

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Laboratory studies show serum creatinine level of 2.2 mg/dL and blood urea nitrogen of 34 mg/dL. Which of the following is the most likely cause of this patient's urinary findings?

- A. Glomerulonephritis [11%]
- B. Nephrolithiasis [2%]
- ✔ C. Tubulointerstitial nephritis [76%]
- D. Urinary tract infection [6%]
- E. Urothelial malignancy [3%]

Omitted
Correct answer
C

76%
Answered correctly

11 Seconds
Time Spent

07/24/2018
Last Updated

Explanation

Clinical features of analgesic nephropathy	
Clinical presentation	• Associated with long-term use of 1 or multiple analgesics (eg, aspirin, ibuprofen) for chronic headaches or other somatic complaints • Usually asymptomatic but can have chronic tubulointerstitial nephritis or hematuria due to papillary necrosis
Diagnosis	• Elevated creatinine with urinalysis showing hematuria or sterile pyuria • Can have mild proteinuria (<1.5 g/day) • CT can show small kidneys with bilateral renal papillary calcifications

This patient's abnormal urinalysis (painless hematuria, sterile pyuria, white blood cell casts, trace proteinuria) suggests a non-glomerular disorder affecting the tubulointerstitium or lining of the urinary tract. Given his chronic low back pain treated with over-the-counter analgesics, he likely has analgesic nephropathy.

Chronic analgesic use with 1 or more analgesics (eg, nonsteroidal anti-inflammatory drug such as aspirin) can cause chronic kidney disease due to chronic tubulointerstitial nephritis. Patients are typically asymptomatic with an elevated creatinine found incidentally. Patients can also develop painless and prominent hematuria due to papillary ischemia from analgesic-induced vasoconstriction of medullary blood vessels (vasa recta). Significant papillary necrosis and sloughing may cause renal colic.

counter analgesics, he likely has analgesic nephropathy.

Chronic analgesic use with 1 or more analgesics (eg, nonsteroidal anti-inflammatory drug such as aspirin) can cause chronic kidney disease due to chronic tubulointerstitial nephritis. Patients are typically asymptomatic with an elevated creatinine found incidentally. Patients can also develop painless and prominent hematuria due to papillary ischemia from analgesic-induced vasoconstriction of medullary blood vessels (vasa recta). Significant papillary necrosis and sloughing may cause renal colic.

(Choice A) Glomerulonephritis is typically accompanied by more characteristic abnormalities seen on urine studies (dysmorphic red blood cells, subnephrotic/nephrotic range proteinuria, and red blood cell casts). However, this patient's predominant presence of urine white blood cells suggests an interstitial nephritis.

(Choice B) Nephrolithiasis usually presents with renal colic accompanied by hematuria. However, nephrolithiasis is typically not associated with trace proteinuria, pyuria, or acute kidney injury unless there is infection or bilateral obstruction.

(Choice D) Urinary tract infection presents with dysuria, fever/chills, abdominal pain, and pyuria (with positive leukocyte esterase or nitrite).

(Choice E) Malignancy typically presents with painless hematuria but is less likely in this patient without tobacco use or family history of cancer. Urothelial malignancy rarely causes trace proteinuria, pyuria, or chronic kidney disease.

Educational objective:
Long-term analgesic use with 1 or more analgesics (eg, nonsteroidal anti-inflammatory drug such as aspirin) can cause chronic kidney disease due to tubulointerstitial nephritis and hematuria due to papillary necrosis.

References

- Analgesic nephropathy

A 7-year-old boy is brought to the emergency department due to tea-colored urine this morning. The patient has had no fever, dysuria, urinary frequency, or urgency. He has had a runny nose and periorbital swelling for 2 days that his parents treated with antihistamines. The rhinorrhea has improved, but the facial swelling has worsened. Three weeks ago, the patient had a facial skin infection that resolved with a topical antibiotic cream. He is otherwise healthy and up to date with vaccinations. The patient's mother has systemic lupus erythematosus. Temperature is 37.4 C (99.4 F) and blood pressure is elevated. Physical examination shows periorbital swelling and mild pedal edema. The remainder of the examination is unremarkable. Laboratory results are as follows:

Serum chemistry		
Blood urea nitrogen	10 mg/dL	
Creatinine	1.4 mg/dL	
Urinalysis		
Specific gravity	1.013	
Protein	+1	
Red blood cells	>50/hpf	
Casts	RBC casts	
Immunologic and rheumatologic studies		
C3	33 mg/dL	(normal: 55-120)
Anti--double-stranded DNA (dsDNA) antibody	negative	

Which of the following is the most likely diagnosis in this patient?

Urinalysis		
Specific gravity	1.013	
Protein	+1	
Red blood cells	>50/hpf	
Casts	RBC casts	
Immunologic and rheumatologic studies		
C3	33 mg/dL	(normal: 55-120)
Anti-double-stranded DNA (dsDNA) antibody	negative	

Which of the following is the most likely diagnosis in this patient?

A. Drug-induced acute interstitial nephritis
B. Granulomatosis with polyangiitis
C. IgA nephropathy
D. Lupus nephritis
E. Poststreptococcal glomerulonephritis

Submit

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Urinalysis

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Red blood cells	>50/hpf	
Casts	RBC casts	

Immunologic and rheumatologic studies

C3	33 mg/dL	(normal: 55-120)
Anti–double-stranded DNA (dsDNA) antibody	negative	

Which of the following is the most likely diagnosis in this patient?

- A. Drug-induced acute interstitial nephritis [1%]
- B. Granulomatosis with polyangiitis [1%]
- C. IgA nephropathy [13%]
- D. Lupus nephritis [1%]
- ✔ E. Poststreptococcal glomerulonephritis [82%]

Explanation

Acute poststreptococcal glomerulonephritis	

Clinical features	• Can be asymptomatic
	• If symptomatic:
	  ◦ Gross hematuria (tea- or cola-colored urine)
	  ◦ Edema (periorbital, generalized)
	  ◦ Hypertension

Laboratory findings	• Urinalysis: + protein, + blood, ± red blood cell casts
	• Serum:
	  ◦ ↓ C3 & possible ↓ C4
	  ◦ ↑ Serum creatinine
	  ◦ ↑ Anti-DNase B & ↑ AHase
	  ◦ ↑ ASO & ↑ anti-NAD (from preceding pharyngitis)

AHase = antihyaluronidase; **anti-DNase B** = antideoxyribonuclease-B; **ASO** = antistreptolysin O.

Acute poststreptococcal glomerulonephritis (APSGN) is an immune complex–mediated disease that presents 1–4 weeks after **group A streptococcal** (GAS) **impetigo** (eg, facial skin infection) or **pharyngitis**. The antigens on specific nephritogenic strains of GAS form immune complexes that are then deposited within the glomerular basement membrane. Following immune complex deposition, the complement system is activated, leading to accumulation of C3 in the glomerular deposits and **low complement component C3** serum levels. Treatment of initial infection does not appear to prevent immune complex deposition and, in turn, APSGN.

component C3 serum levels. Treatment of initial infection does not appear to prevent immune complex deposition and, in turn, APSGN.

Patients may be asymptomatic, with microscopic hematuria, or have gross **hematuria**, **edema** (eg, periorbital, generalized), and **hypertension**, as in this case. Serum creatinine may be elevated, and urinalysis shows red blood cells with or without red blood cell casts and proteinuria. Treatment is supportive (eg, blood pressure control). Symptoms typically resolve within weeks whereas laboratory abnormalities may take months to normalize.

(Choice A) Drug-induced interstitial nephritis most commonly occurs after administration of nonsteroidal anti-inflammatory drugs or antibiotics (eg, penicillins, cephalosporins). Manifestations include fever, rash, and arthralgia. Laboratory evaluation reveals elevated serum creatinine in addition to white blood cells (specifically eosinophils) and white blood cell casts in the urine. Antihistamines, which were used to treat this patient's likely allergic rhinitis, are not typically associated with drug-induced interstitial nephritis.

(Choice B) Renal manifestations of granulomatosis with polyangiitis include elevated serum creatinine and hematuria. However, patients classically have nonspecific, systemic symptoms including fever, fatigue, and weight loss, none of which are seen in this case. In addition, ear, nose, and throat (eg, sinusitis, otitis media) and pulmonary (eg, dyspnea) manifestations are common, and this disease is extremely rare in children.

(Choice C) IgA nephropathy typically presents with hematuria 1–2 days after the onset of an upper respiratory tract infection, and serum complement levels are normal. This patient had 3 weeks between a skin infection and the development of hematuria, and decreased complement component C3, findings suggestive of APSGN.

(Choice D) Lupus nephritis may be asymptomatic or present with a wide range of findings, including hematuria, proteinuria, hypertension, and elevated serum creatinine. The absence of a positive anti–double-stranded DNA antibody and other findings consistent with systemic lupus erythematosus (eg, arthritis, rash) make this diagnosis unlikely.

Educational objective:
Acute poststreptococcal glomerulonephritis occurs 1–4 weeks after group A streptococcal pharyngitis or impetigo. Patients may be asymptomatic or have hematuria, hypertension, and edema. Complement component C3 is low.

A 62-year-old woman presents to your office complaining of urinary frequency and burning during urination. She denies fever, chills, nausea, back pain or abdominal pain. Her past medical history is significant for a long history of diabetes mellitus and hypertension. She does not use tobacco or consume alcohol. Her blood pressure is 160/100 mmHg and her heart rate is 70/min. Her hematocrit is 43% and her WBC count is 8,500/mm^3. Urinalysis reveals the following:

Glucose	negative
Ketones	negative
Nitrites	positive
Protein	2+
WBC	20-25/hpf
RBC	3-5/hpf

She is given a three day course of levofloxacin. Urinalysis two weeks later reveals 2+ protein but no nitrites, WBCs, or RBCs. Which of the following is most likely responsible for her persistent urinalysis abnormality?

A. Atherosclerotic narrowing of the renal arteries
B. Glomerular basement membrane changes
C. Cystic transformation of the renal parenchyma
D. Parenchymal atrophy due to calyceal dilation
E. Insoluble crystal precipitation in the tubular lumen

45% and her WBC count is 8,500/mm³. Urinalysis reveals the following:

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A. Atherosclerotic narrowing of the renal arteries [9%]
✔ B. Glomerular basement membrane changes [81%]
C. Cystic transformation of the renal parenchyma [2%]
D. Parenchymal atrophy due to calyceal dilation [2%]
E. Insoluble crystal precipitation in the tubular lumen [3%]

Omitted
Correct answer
B

81%
Answered correctly

5 Seconds
Time Spent

12/16/2018
Last Updated

Explanation

This patient's findings on urinalysis are a reflection of two disease processes. Initially she presents with a urinary tract infection characterized by urinary frequency, dysuria, pyuria, and positive nitrites. After the UTI is treated, however, the proteinuria persists. This is most likely a reflection of diabetic nephropathy secondary to her longstanding diabetes mellitus. Diabetes mellitus is the leading cause of end stage renal disease in the United States, occurring in 30-40% and 20% of patients with type I and type II diabetes mellitus after 20 years, respectively. The most common histologic lesion in diabetic nephropathy is diffuse glomerulosclerosis. Nodular glomerulosclerosis (with Kimmelstiel-Wilson nodules) is pathognomonic. Disease progression can be slowed with strict glycemic control, treatment of hypertension, and angiotensin axis blockade.

(Choice A) Renal artery stenosis is typically due to atherosclerotic vascular disease. It presents as difficult-to-control hypertension with or without renal dysfunction. Renal artery stenosis is the most common cause of secondary hypertension in adults. It does not typically cause proteinuria.

(Choice C) Autosomal dominant polycystic kidney disease is the leading cause of inherited renal disease in adults. It typically presents as abdominal or flank pain with microscopic or gross hematuria. There is a positive family history in 75% of cases and over half of patients have hypertension at presentation. Proteinuria is not a classic finding.

(Choice D) Parenchymal atrophy due to calyceal dilation occurs in obstructive nephropathy. This patient does not have any symptoms of urinary obstruction. Urinary obstruction is much more common in men than in women.

(Choice E) Insoluble crystal deposition resulting in acute renal failure is seen with hyperuricemia (tumor lysis syndrome), indinavir, acyclovir, and sulfonamide therapy.

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(Choice A) Renal artery stenosis is typically due to atherosclerotic vascular disease. It presents as difficult-to-control hypertension with or without renal dysfunction. Renal artery stenosis is the most common cause of secondary hypertension in adults. It does not typically cause proteinuria.

(Choice C) Autosomal dominant polycystic kidney disease is the leading cause of inherited renal disease in adults. It typically presents as abdominal or flank pain with microscopic or gross hematuria. There is a positive family history in 75% of cases and over half of patients have hypertension at presentation. Proteinuria is not a classic finding.

(Choice D) Parenchymal atrophy due to calyceal dilation occurs in obstructive nephropathy. This patient does not have any symptoms of urinary obstruction. Urinary obstruction is much more common in men than in women.

(Choice E) Insoluble crystal deposition resulting in acute renal failure is seen with hyperuricemia (tumor lysis syndrome), indinavir, acyclovir, and sulfonamide therapy.

Educational objective:
Diabetic nephropathy is characterized by proteinuria and a progressive decline in GFR. The pathologic hallmark is nodular glomerulosclerosis but diffuse glomerulosclerosis is more common.

A 14-year-old girl is brought to the physician for evaluation of facial puffiness, fatigue, and decreased appetite for the past few days. The patient recently immigrated from China to the United States. Temperature is 36.7 C (98 F), blood pressure is 110/70 mm Hg, pulse is 80/min, and respirations are 18/min. Physical examination shows periorbital and pretibial edema. Serum laboratory results are as follows:

Creatinine	0.9 mg/dL
Albumin	2.2 mg/dL
Total bilirubin	0.5 mg/dL
Aspartate aminotransferase	56 U/L
Alanine aminotransferase	64 U/L
Alkaline phosphatase	97 U/L
HBsAg	Positive
HBeAg	Positive
Anti-HBsAg antibodies	Negative
Anti-HCV antibodies	Negative
Anti-HIV antibodies	Negative

Aspartate aminotransferase	56 U/L
Alanine aminotransferase	64 U/L
Alkaline phosphatase	97 U/L
HBsAg	Positive
HBeAg	Positive
Anti-HBsAg antibodies	Negative
Anti-HCV antibodies	Negative
Anti-HIV antibodies	Negative

Urinalysis shows 4+ proteinuria, no red blood cells, and no casts. Which of the following is the most likely diagnosis in this patient?

A. Focal segmental glomerulosclerosis
B. Membranoproliferative glomerulonephritis
C. Membranous nephropathy
D. Minimal change disease
E. Poststreptococcal glomerulonephritis

Submit

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Anti-HCV antibodies	Negative
Anti-HIV antibodies	Negative

Urinalysis shows 4+ proteinuria, no red blood cells, and no casts. Which of the following is the most likely diagnosis in this patient?

- A. Focal segmental glomerulosclerosis [13%]
- B. Membranoproliferative glomerulonephritis [18%]
- ✔ C. Membranous nephropathy [44%]
- D. Minimal change disease [22%]
- E. Poststreptococcal glomerulonephritis [1%]

Omitted
Correct answer
C

44%
Answered correctly

4 Seconds
Time Spent

08/22/2018
Last Updated

Explanation

Nephrotic versus nephritic syndrome		
	Nephrotic syndrome	**Nephritic syndrome**
Clinical features	• Edema • Fatigue • Proteinuria • Absence of hematuria • Hypoalbuminemia	• Hypertension • Oliguria • Hematuria • Proteinuria • Casts
Pediatric etiologies	• Minimal change disease	• Poststreptococcal glomerulonephritis • Hemolytic uremic syndrome
Adult etiologies	• FSGS • Membranous nephropathy • Membranoproliferative glomerulonephritis	• IgA nephropathy • Membranoproliferative glomerulonephritis • Crescentic glomerulonephritis

This patient's **edema**, **hypoalbuminemia**, and **markedly elevated urine protein** are consistent with **nephrotic syndrome (NS)**.

Common causes of NS include minimal change disease in young children and focal segmental glomerulosclerosis (FSGS) and

This patient's **edema**, **hypoalbuminemia**, and **markedly elevated urine protein** are consistent with **nephrotic syndrome (NS)**. Common causes of NS include minimal change disease in young children and focal segmental glomerulosclerosis (FSGS) and membranous nephropathy in adolescents and adults.

Although **membranous nephropathy** is less common in children, **hepatitis B infection** is a significant risk factor. Universal **vaccination** has dramatically reduced rates of hepatitis B virus-associated membranous nephropathy (HBVMN), and unvaccinated children who have immigrated from endemic areas should be screened for hepatitis B. This patient is positive for HBsAg and HBeAg and negative for anti-HBsAg antibody, findings consistent with active hepatitis B infection. The pathogenesis of HBVMN may involve deposition of HBeAg or its corresponding antibody in the glomeruli.

Additional workup for this patient should include a 24-hour urine sample (protein excretion **>3 g/day** is consistent with NS), serum C3 (typically low with HBVMN), antinuclear antibody (elevated in lupus), and renal biopsy.

(Choice A) FSGS is a common cause of NS in adults and adolescents. HIV is the most commonly associated infection.

(Choice B) Membranoproliferative glomerulonephritis can cause NS or nephritic syndrome, most commonly in adults, and has been associated with hepatitis B infection. However, it is significantly less common than membranous nephropathy.

(Choice D) Minimal change disease is the most common cause of NS in preadolescent children. This patient is a teenager and has active hepatitis B infection, making membranous nephropathy more likely.

(Choice E) Poststreptococcal glomerulonephritis is the most common cause of nephritic syndrome in children. The absence of hypertension, oliguria, hematuria, and casts makes nephritic syndrome unlikely.

Educational objective:
Membranous nephropathy is a common cause of nephrotic syndrome (edema, proteinuria, and hypoalbuminemia) in adolescents and adults. Active hepatitis B infection is an important risk factor, and vaccination reduces this risk.

A 55-year-old woman comes to the physician for an annual physical examination. She has no new complaints, except fatigue. She has an 8-year history of chronic low back pain; severe degenerative joint disease has been documented on MRI. She had an anterior wall myocardial infarction four years ago. Her current medications include naproxen, acetaminophen, oxycodone, aspirin, atenolol, and simvastatin. Her blood pressure is 130/80 mm Hg and pulse is 72/min. Laboratory studies show:

Hb	10 g/dL
WBC	6,000/cmm
Blood sugar	82 mg/dL
BUN	36 mg/dL
Serum creatinine	2.0 mg/dL

Urinalysis:

Protein	2+
Glucose	Absent
RBC	Absent
WBC	10-15/HPF
Nitrite	Negative
Esterase	Negative
Sediment	WBC casts

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Protein	2+
Glucose	Absent
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Esterase	Negative
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Serum protein electrophoresis is negative for monoclonal gammopathy. Two years ago, her BUN level was 22 mg/dL, and creatinine level was 1.6 mg/dL. Which of the following is the most likely pathology involved in this patient's renal failure?

A. Acute tubular necrosis
B. Chronic glomerulonephritis
C. Tubulointerstitial nephritis
D. Recurrent pyelonephritis

Submit

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Serum protein electrophoresis is negative for monoclonal gammopathy. Two years ago, her BUN level was 22 mg/dL, and creatinine level was 1.6 mg/dL. Which of the following is the most likely pathology involved in this patient's renal failure?

A. Acute tubular necrosis [10%]
B. Chronic glomerulonephritis [16%]
✔ C. Tubulointerstitial nephritis [61%]
D. Recurrent pyelonephritis [11%]

Omitted
Correct answer
C

61%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

Analgesic nephropathy is the most common form of drug-induced chronic renal failure. It accounts for 3-5% of end stage renal disease in the USA, and is most commonly seen in females (peak at age 50-55 years) who habitually use combined analgesics (e.g., aspirin and naproxen). It is generally seen after cumulative ingestion of 2-3 kg (4.4-6.6 lbs) of the index drug. Papillary necrosis and chronic tubulointerstitial nephritis are the most common pathologies seen. Polyuria and sterile pyuria (WBC casts may also be seen) are early manifestations. Microscopic hematuria and renal colic may occur following sloughing of renal papilla. Hypertension, mild proteinuria, and impaired urinary concentration commonly occur as the disease advances. In severe cases, nephrotic range proteinuria can be seen. Patients with chronic analgesic abuse are also more likely to develop premature aging, atherosclerotic vascular disease, and urinary tract cancer.

(Choice A) Acute tubular necrosis causes acute renal failure, rather than insidious progression of renal dysfunction. It is most commonly seen in ischemic or nephrotoxic acute renal failure. Muddy brown granular casts are characteristic.

(Choice B) Glomerulonephritis manifests as nephritic syndrome. Hematuria (RBC casts), edema, hypertension, and proteinuria are characteristic.

(Choice D) Chronic pyelonephritis can cause chronic tubulointerstitial nephritis; however, it is associated with a history of recurrent urinary tract infections and symptoms such as fever, back pain, and dysuria.

Educational Objective:
Analgesic nephropathy is the most common form of drug-induced chronic renal failure. *Papillary necrosis* and *chronic tubulointerstitial nephritis* are the most common pathologies seen. Patients with chronic analgesic abuse are also more likely to develop premature aging, atherosclerotic vascular disease, and urinary tract cancer.

A 52-year-old man with alcoholism comes to the emergency department due to generalized weakness, anxiety, and tremors. His last drink was 2 days ago. He has no significant medical history and no medical follow-up. The patient smokes cigarettes but does not use illicit drugs. He takes no medications. On examination, he appears disheveled and malnourished. His initial electrolyte panel results are as follows:

Sodium	132 mEq/L
Potassium	2.9 mEq/L
Chloride	100 mEq/L
Bicarbonate	25 mEq/L

He is treated for alcohol withdrawal and given aggressive intravenous potassium as well as oral potassium supplementation. Three days later, his electrolyte panel results are as follows:

Sodium	135 mEq/L
Potassium	3.1 mEq/L
Chloride	102 mEq/L
Bicarbonate	28 mEq/L

Which of the following best explains why this patient's potassium level is very difficult to correct?

A. Alcohol withdrawal
B. Hypoalbuminemia
C. Hypomagnesemia

Bicarbonate 25 mEq/L

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- A. Alcohol withdrawal
- B. Hypoalbuminemia
- C. Hypomagnesemia
- D. Hypophosphatemia
- E. Poor oral absorption
- F. Renal tubular acidosis
- G. Thiamine deficiency

Submit

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Which of the following best explains why this patient's potassium level is very difficult to correct?

A. Alcohol withdrawal [2%]
B. Hypoalbuminemia [6%]
C. Hypomagnesemia [63%]

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Potassium	3.1 mEq/L
Chloride	102 mEq/L
Bicarbonate	28 mEq/L

Which of the following best explains why this patient's potassium level is very difficult to correct?

- A. Alcohol withdrawal [2%]
- B. Hypoalbuminemia [6%]
- ✔ C. Hypomagnesemia [63%]
- D. Hypophosphatemia [4%]
- E. Poor oral absorption [1%]
- F. Renal tubular acidosis [12%]
- G. Thiamine deficiency [9%]

Omitted
Correct answer
C

63%
Answered correctly

5 Seconds
Time Spent

11/06/2018
Last Updated

Explanation

Chronic alcoholism is associated with a high incidence of several electrolyte abnormalities, of which **hypomagnesemia** is the most common (likely due to **poor nutritional intake**, alcohol-induced renal losses, and diarrhea). Hypomagnesemia commonly occurs together with hypokalemia and is a well-known cause of **refractory hypokalemia** (hypokalemia that cannot be corrected with potassium replacement).

Intracellular magnesium is thought to **inhibit potassium secretion** by **renal outer medullary potassium (ROMK) channels** in the collecting tubules of the kidney. Therefore, low intracellular magnesium concentrations result in excessive renal potassium loss and refractory hypokalemia. Normalization of magnesium levels restores ROMK channel potassium transport regulation, decreases renal potassium losses, and allows for successful correction of hypokalemia with oral (preferred) or intravenous potassium replacement.

(Choice A) Acute alcohol withdrawal may initially contribute to hypokalemia through an increase in sympathetic nervous system activity, which shifts potassium into cells. However, it is unlikely to play a significant role in this patient's hypokalemia, which has persisted throughout 3 days of treatment.

(Choice B) Hypoalbuminemia is commonly seen in patients with alcoholism due to poor nutrition or hepatic synthetic dysfunction. It is a common cause of hypocalcemia (total but not ionized); however, it is not a significant cause of hypokalemia.

(Choice D) Hypophosphatemia is common in patients with alcoholism and, when severe, can result in weakness, rhabdomyolysis, paresthesias, and respiratory failure. It does not directly contribute to hypokalemia.

(Choice E) Poor oral absorption may result in hypokalemia refractory to oral replacement, but it is unlikely in this patient with hypokalemia that has also been refractory to aggressive intravenous replacement.

(Choice F) Type I or II renal tubular acidosis can cause hypokalemia but is usually associated with metabolic acidosis. This patient has a relatively normal serum bicarbonate level, and his history of alcoholism makes hypomagnesemia a more likely cause of his refractory hypokalemia.

(Choice B) Hypoalbuminemia is commonly seen in patients with alcoholism due to poor nutrition or hepatic synthetic dysfunction. It is a common cause of hypocalcemia (total but not ionized); however, it is not a significant cause of hypokalemia.

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(Choice G) Thiamine deficiency is common in patients with alcoholism due to poor nutrition, but it does not cause refractory hypokalemia.

Educational objective:
Patients with chronic alcoholism often present with multiple electrolyte abnormalities (eg, hypokalemia, hypomagnesemia, hypophosphatemia). Hypomagnesemia can lead to refractory hypokalemia due to removal of inhibition of renal potassium excretion and should be suspected in patients with hypokalemia that is difficult to correct with potassium replacement.

References

- Pathogenetic mechanisms of hypomagnesemia in alcoholic patients.
- Mechanism of hypokalemia in magnesium deficiency.

A 26-year-old man comes to the emergency department because of a sudden onset of severe, colicky, left-sided flank pain that radiates to the scrotum. He also has nausea, vomiting and dark-colored urine. He has never had these symptoms before. Examination shows no abnormalities. Non-contrast helical CT shows a 5 mm radiopaque stone in the left upper ureter. His laboratory studies are as follows:

Serum calcium	9.8 mg/dL
Serum creatinine	0.9 mg/dL
BUN	15 mg/dL

Urinalysis shows hematuria but no casts. Which of the following is the most likely cause of this patient's symptoms?

- A. Calcium oxalate stones
- B. Calcium phosphate stones
- C. Uric acid stones
- D. Cysteine stones
- E. Struvite stones

Submit

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Serum calcium	9.8 mg/dL
Serum creatinine	0.9 mg/dL
BUN	15 mg/dL

Urinalysis shows hematuria but no casts. Which of the following is the most likely cause of this patient's symptoms?

- A. Calcium oxalate stones [73%]
- B. Calcium phosphate stones [13%]
- C. Uric acid stones [5%]
- D. Cysteine stones [4%]
- E. Struvite stones [4%]

Omitted
Correct answer
A

73%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Correct answer
A

73%
Answered correctly

3 Seconds
Time Spent

08/09/2018
Last Updated

Explanation

75% to 90% of kidney stones are composed of calcium oxalate. Calcium oxalate crystals are envelope- shaped, and can be seen on microscopic examination of urine. Small bowel disease, surgical resection or chronic diarrhea can lead to malabsorption of fatty acids and bile salts; this predisposes to the formation of calcium oxalate stones. (Fat malabsorption leads to the increased absorption of oxalic acid because the unabsorbed fatty acids chelate calcium, making oxalic acid free for absorption.)

(Choice B) Calcium phosphate stones are common in primary hyperparathyroidism and renal tubular acidosis.

(Choice C) Uric acid stones are formed when the urine is acidic or when there is increased cell turnover, thereby resulting in hyperuricemia and hyperuricosuria. Dehydration is another important risk factor.

(Choice D) Cysteine stones are formed when there is increased excretion of cysteine, which is an inborn error of metabolism. A positive family history may be found in such cases.

(Choice E) Struvite stones are formed when urine is alkaline because of infection with urease producing bacteria (e.g., *Proteus*). In such cases, a history of recurrent UTI may be present.

Educational Objective:
Colicky flank pain with radiation to the groin indicates renal colic. 75% to 90% of the kidney stones are composed of calcium oxalate. Small bowel disease, surgical resection or chronic diarrhea can lead to malabsorption of fatty acids and bile salts; this in turn predisposes to the formation calcium oxalate stones.

A 47-year-old woman, gravida 4, para 4, comes to the physician with lower abdominal pain that is relieved with urination. The pain has been present for over 2 months. The patient has also been voiding more frequently than usual. She is occasionally sexually active with her husband, but intercourse is painful. The woman has a history of 4 uncomplicated vaginal deliveries during her 30s. She has no fever or chills. On examination, the patient has mild lower abdominal pain with no rebound or guarding. Her external genitalia appear normal. On bimanual examination, palpation of the anterior vaginal wall elicits severe pain. No cervical motion tenderness is present. The rest of the examination is normal. Urinalysis results are as follows:

Specific gravity	1.013
Protein	None
Blood	Negative
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Negative
Nitrites	Negative
Bacteria	None
White blood cells	1-2/hpf
Red blood cells	0/hpf

Which of the following is the most likely diagnosis?

A. Cystocele

White blood cells	1-2/hpf
Red blood cells	0/hpf

Which of the following is the most likely diagnosis?

- A. Cystocele
- B. Interstitial cystitis
- C. Pelvic inflammatory disease
- D. Stress incontinence
- E. Urinary tract infection

Submit

Blood	Negative
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Negative
Nitrites	Negative
Bacteria	None
White blood cells	1-2/hpf
Red blood cells	0/hpf

Which of the following is the most likely diagnosis?

- A. Cystocele [45%]
- ✔ B. Interstitial cystitis [44%]
- C. Pelvic inflammatory disease [5%]
- D. Stress incontinence [2%]
- E. Urinary tract infection [1%]

Omitted
Correct answer
B

44%
Answered correctly

4 Seconds
Time Spent

12/31/2018
Last Updated

Explanation

Interstitial cystitis (bladder pain syndrome)	
Epidemiology	• More common in women • Associated with psychiatric & pain disorders (eg, fibromyalgia)
Clinical presentation	• Bladder pain with filling, relief with voiding • ↑ Urinary frequency, urgency • Dyspareunia
Diagnosis	• Bladder pain with no other cause for ≥6 weeks • Normal urinalysis
Treatment	• Not curative, focus is on improving quality of life • Behavioral modification, avoidance of triggers, physical therapy • Amitriptyline, pentosan polysulfate sodium • Analgesics for acute exacerbations

Interstitial cystitis (also known as painful bladder syndrome) is a chronic, painful bladder condition of uncertain etiology. The pelvic pain in interstitial cystitis is classically **exacerbated by bladder filling** and **relieved by voiding**. The onset of the symptoms is typically gradual, and the symptoms worsen over a period of months. Other characteristic symptoms include urinary **urgency** and **frequency**, and **chronic pelvic pain**. The pain can be exacerbated by exercise, sexual intercourse (dyspareunia), and alcohol consumption.

The diagnosis of interstitial cystitis is primarily clinical. Urinalysis is obtained to exclude other causes of bladder pain (eg, urinary tract

consumption.

The diagnosis of interstitial cystitis is primarily clinical. Urinalysis is obtained to exclude other causes of bladder pain (eg, urinary tract infection, sexually transmitted disease, cancer); results are normal in interstitial cystitis. Treatment is palliative and includes trigger avoidance, amitriptyline, and analgesics for pain flares.

(Choice A) A cystocele refers to bladder prolapse into the anterior vaginal wall, which can cause vaginal pressure, dyspareunia, urinary frequency and urgency, and incontinence. This patient has no evidence of a prolapse on examination.

(Choice C) Pelvic inflammatory disease is characterized by pelvic pain, cervical motion tenderness, and fever. Urinary symptoms are usually not present.

(Choice D) Stress incontinence refers to involuntary leakage of urine with exertion, sneezing, or coughing. Pain is typically not a symptom of stress incontinence.

(Choice E) Urinalysis (and urine culture, if urinalysis is abnormal) is performed to rule out urinary tract infection. The normal urinalysis in this patient excludes a urinary tract infection.

Educational objective:
Interstitial cystitis (painful bladder syndrome) is an idiopathic, chronic condition characterized by bladder pain that is worsened by filling and relieved by voiding. Dyspareunia, urinary frequency and urgency can also be present.

References

- Advances in diagnosis and treatment of interstitial cystitis/painful bladder syndrome.
- Treating interstitial cystitis/bladder pain syndrome as a chronic disease.

An 18-year-old girl comes to the emergency department with a new-onset skin rash and malaise. She is concerned as her aunt has lupus treated with corticosteroids. Her medical history is unremarkable except for dysuria and increased urinary frequency a week ago, which was effectively treated with sulfamethoxazole-trimethoprim. The patient takes no medications and uses no illicit drugs. She has had the same sexual partner for the past 4 months. Her temperature is 38.0 C (100.4 F), pulse is 86/min, and respirations are 16/min. Physical examination reveals a disseminated maculopapular rash. There is no costovertebral tenderness. The patient has no joint swelling or effusion. Her serum creatinine is 2.0 mg/dL. Urinalysis reveals 2-5 red blood cells/hpf, numerous white blood cell casts, and mild proteinuria. Which of the following is the most likely diagnosis?

A. Disseminated gonococcemia
B. Interstitial nephritis
C. Lupus nephritis
D. Postinfectious acute glomerulonephritis
E. Pyelonephritis

Submit

An 18-year-old girl comes to the emergency department with a new-onset skin rash and malaise. She is concerned as her aunt has lupus treated with corticosteroids. Her medical history is unremarkable except for dysuria and increased urinary frequency a week ago, which was effectively treated with sulfamethoxazole-trimethoprim. The patient takes no medications and uses no illicit drugs. She has had the same sexual partner for the past 4 months. Her temperature is 38.0 C (100.4 F), pulse is 86/min, and respirations are 16/min. Physical examination reveals a disseminated maculopapular rash. There is no costovertebral tenderness. The patient has no joint swelling or effusion. Her serum creatinine is 2.0 mg/dL. Urinalysis reveals 2-5 red blood cells/hpf, numerous white blood cell casts, and mild proteinuria. Which of the following is the most likely diagnosis?

- A. Disseminated gonococcemia [11%]
- ✔ B. Interstitial nephritis [65%]
- C. Lupus nephritis [7%]
- D. Postinfectious acute glomerulonephritis [10%]
- E. Pyelonephritis [5%]

Omitted
Correct answer
B

65%
Answered correctly

4 Seconds
Time Spent

09/16/2018
Last Updated

Explanation

Acute interstitial nephritis

Explanation

Acute interstitial nephritis	
Causes	• **Drugs (penicillins, TMP-SMX, cephalosporins**, NSAIDS)
Clinical features	• Maculopapular rash • Fever • New drug exposure • +/- Arthralgias
Laboratory findings	• Acute kidney injury • Pyuria, hematuria, WBC casts • Eosinophilia, **urinary eosinophils** • Renal biopsy: Inflammatory infiltrate, edema
Management	• Discontinue offending drug • +/- Systemic glucocorticoids

NSAIDS = nonsteroidal anti-inflammatory drugs;
TMP-SMX = trimethoprim-sulfamethoxazole; **WBC** = white blood cell.

The clinical presentation is typical of **allergic interstitial nephritis** (AIN): **acute renal failure**, **fever**, **rash**, a recent history of para-aminobenzoic acid analogue antibiotic (sulfonamide) ingestion, and **white blood cell (WBC) casts** on urinalysis. Patients may also have arthralgias and eosinophiluria. Symptoms usually appear 5 days to several weeks after use of an offending agent. Drugs cause

NSAIDS = nonsteroidal anti-inflammatory drugs;
TMP-SMX = trimethoprim-sulfamethoxazole; **WBC** = white blood cell.

The clinical presentation is typical of **allergic interstitial nephritis** (AIN): **acute renal failure**, **fever**, **rash**, a recent history of para-aminobenzoic acid analogue antibiotic (sulfonamide) ingestion, and **white blood cell (WBC) casts** on urinalysis. Patients may also have arthralgias and eosinophiluria. Symptoms usually appear 5 days to several weeks after use of an offending agent. Drugs cause the majority of AIN cases; examples include antibiotics (eg, penicillins, cephalosporins, trimethoprim, rifampin), nonsteroidal anti-inflammatory drugs, and diuretics. Less commonly, AIN may be caused by infectious agents (eg, *Legionella*, *Mycobacterium tuberculosis*, *Streptococcus*).

(Choice A) Disseminated gonococcemia causes a vesicopustular rash. Renal failure with WBC casts is not usually seen.

(Choice C) The mucocutaneous manifestations of lupus include malar rash and discoid rash, but this patient has a maculopapular rash. Lupus nephritis is characterized by hypertension, mild proteinuria, and red blood cell (RBC) casts.

(Choice D) Postinfectious glomerulonephritis produces hematuria, mild proteinuria, RBC casts, and fluid retention resulting in hypertension and periorbital edema. It usually occurs 1-2 weeks after an episode of streptococcal pharyngitis/skin infection and not after a urinary infection.

(Choice E) Pyelonephritis can complicate untreated cystitis, but manifests with chills, fever, and tenderness in the flanks and/or the costovertebral angle. Disseminated rash is not usually seen.

Educational objective:
Drug-induced interstitial nephritis is usually caused by antibiotics (eg, penicillins, cephalosporins, trimethoprim, rifampin), nonsteroidal anti-inflammatory drugs, and diuretics. Patients present with a fever, maculopapular rash, and renal failure. Urinalysis may reveal white blood cell casts and, less frequently, eosinophils.

A 61-year-old man comes to the office due to nocturnal urinary frequency, occasional dribbling, and a weak urinary stream for the past 3 months. He was diagnosed with type 2 diabetes 16 years ago and has recently well-controlled blood sugars. His other medical problems include hypertension, myocardial infarction 2 years ago, and moderately decreased visual acuity. Blood pressure is 160/100 mm Hg and pulse is 70/min. Examination shows left-sided carotid bruit and trace bilateral ankle edema. Post-void bladder residual volume is 40 mL. Dipstick urinalysis shows 3+ protein (900 mg/dL) and no blood. The patient's serum creatinine level is 2.1 mg/dL. Which of the following is the most likely cause of his chronic kidney disease?

- A. Ascending infection
- B. Cystic kidney disease
- C. Fibromuscular dysplasia
- D. Microangiopathy
- E. Obstructive uropathy

Submit

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- A. Ascending infection [0%]
- B. Cystic kidney disease [1%]
- C. Fibromuscular dysplasia [4%]
- ✔ D. Microangiopathy [69%]
- E. Obstructive uropathy [23%]

Omitted
Correct answer
D

69%
Answered correctly

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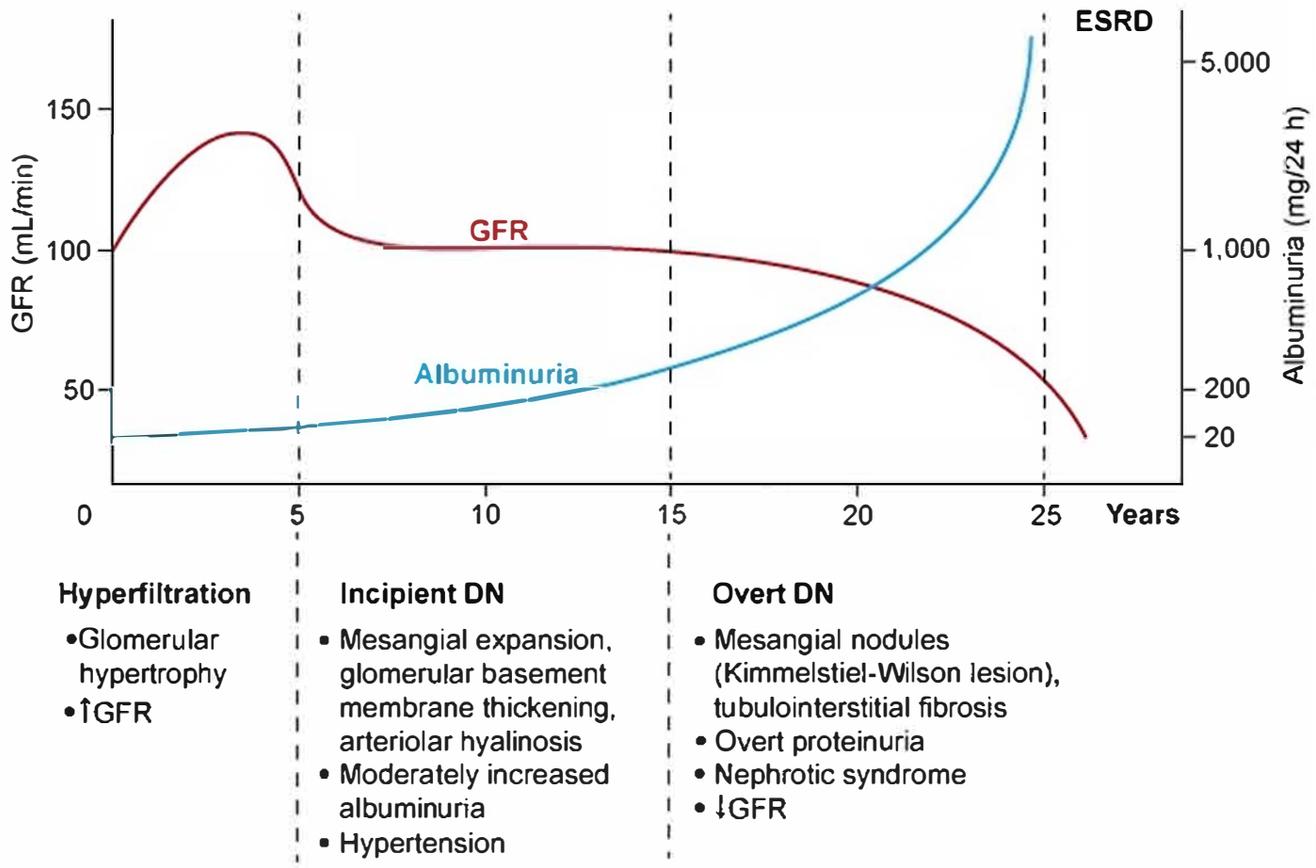
11/05/2018
Last Updated

Explanation

Natural history of diabetic nephropathy

Explanation

Natural history of diabetic nephropathy



DN = diabetic nephropathy; ESRD = end-stage renal disease; GFR = glomerular filtration rate.
©UWorld

In this patient with multiple comorbidities including diabetes, elevated serum creatinine, and proteinuria, the most likely cause of

In this patient with multiple comorbitidies including diabetes, elevated serum creatinine, and proteinuria, the most likely cause of **chronic kidney disease** is diabetic glomerulosclerosis. Diabetes is the most common cause of CKD and end-stage renal disease (ESRD) requiring dialysis in North America. Risk factors for **diabetic nephropathy** (DN) include poor glycemic control, elevated blood pressure, cigarette smoking, increasing age, and ethnicity (eg, African American, Mexican American). Most patients also have evidence of retinopathy or neuropathy.

DN typically occurs 10-15 years after onset of type 1 and 2 diabetes. Hyperglycemia leads to microvascular damage (microangiopathy). The advanced glycation end products and other inflammatory mediators damage the glomerulus, resulting in **proteinuria**, overt glomerular nephropathy, and ESRD. Up to 50% of type 1 and 2 diabetics can develop initial mild proteinuria (microalbuminuria), defined as urinary albumin excretion of 30-300 mg/day. However, the degree of albuminuria does not always correlate with the severity of disease progression. Histologic changes in the glomerulus include mesangial expansion, glomerular basement membrane thickening, and glomerular sclerosis.

Diabetics may also have albuminuria due to other causes. Clinical clues suggesting albuminuria due to nondiabetic renal disease include onset of proteinuria <5 years after disease onset, active urine sediment (eg, red cells, cellular casts), and >30% reduction of glomerular filtration rate within 2-3 months of starting ACE inhibitors or angiotensin receptor blockers. This patient's clinical findings (decreased visual acuity, elevated blood pressure, diabetes >15 years, and no active urinary sediment) favor DN as a cause of his CKD. Good glycemic control may help control the progression of DN but is unlikely to completely prevent its development.

(Choice A) Chronic ascending urinary tract infections can lead to scarring and CKD. Although this patient presents with lower urinary tract symptoms, he has no other signs of infection (eg, dysuria, fever, chills, flank pain, pyuria).

(Choice B) Adult polycystic kidney disease can cause hypertension and progressive renal insufficiency, but the associated proteinuria is usually minimal or mild. In addition, these patients usually have flank and/or abdominal pain and hematuria.

(Choice C) Fibromuscular dysplasia (FMD) can cause arterial stenosis, aneurysm, or dissection. FMD involving the renal arteries leads to hypertension. Involvement of the cerebrovascular arteries (eg, carotid, vertebral) can cause transient ischemic attack, stroke,

tract symptoms, he has no other signs of infection (eg, dysuria, fever, chills, flank pain, pyuria).

(Choice B) Adult polycystic kidney disease can cause hypertension and progressive renal insufficiency, but the associated proteinuria is usually minimal or mild. In addition, these patients usually have flank and/or abdominal pain and hematuria.

(Choice C) Fibromuscular dysplasia (FMD) can cause arterial stenosis, aneurysm, or dissection. FMD involving the renal arteries leads to hypertension. Involvement of the cerebrovascular arteries (eg, carotid, vertebral) can cause transient ischemic attack, stroke, or nonspecific symptoms (eg, headache, pulsatile tinnitus, dizziness). Although FMD could cause some of this patient's findings (hypertension, carotid bruit, CKD from chronic renal hypoperfusion), it usually does not cause significant proteinuria.

(Choice E) Bladder outlet obstruction can cause nocturnal urinary frequency, dribbling, and weak urinary stream. This is most commonly due to benign prostatic hyperplasia (BPH) in men age >50. Post-void residual volume is usually >50 mL in significant bladder outlet obstruction. However, BPH does not typically cause substantial proteinuria. In addition, it does not usually cause significant kidney disease unless there is complete obstruction. Although this patient has BPH symptoms (eg, weak stream, dribbling), he also was found to have likely diabetic CKD, which is often asymptomatic in early stages and commonly found on routine studies.

Educational objective:
Patients with diabetes for >10 years can develop diabetic microangiopathy, nephropathy, and glomerulosclerosis. Risk factors include poor glycemic control, elevated blood pressure, smoking, increasing age, and ethnicity (eg, African American, Mexican American). Clinical findings include mild to moderate proteinuria and chronic kidney disease with elevated creatinine.

References

- Pathogenesis and novel treatment from the mouse model of type 2 diabetic nephropathy

• Hypertension

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The urine dipstick for proteinuria (albumin)	
Trace	Between 15 and 30 mg/dL
1+	Between 30 and 100 mg/dL
2+	Between 100 and 300 mg/dL
3+	Between 300 and 1000 mg/dL
4+	>1000 mg/dL

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A 20-year-old man is brought to the emergency department with a one-day history of fever, headache, and altered mental status. He has no history of medical illness. Cerebral spinal fluid analysis shows lymphocytic pleocytosis, elevated protein level, and normal glucose level. The patient is started on high-dose intravenous acyclovir. Two days later, his neurologic status improves. Polymerase chain reaction assay for herpes simplex virus DNA comes back positive. However, the patient complains of nausea and abdominal discomfort. He is afebrile, normotensive without orthostasis, and has normal oxygen saturation. The rest of the physical examination is unremarkable. Laboratory results are as follows:

Serum sodium	140 mEq/L
Serum potassium	4.5 mEq/L
Serum creatinine	2.8 mg/dL
Blood urea nitrogen	38 mg/dL

His admission creatinine level was 0.8 mg/dL. Which of the following is the most likely cause of acute kidney injury in this patient?

A. Acute interstitial nephritis
B. Bladder neck obstruction
C. Glomerular injury
D. Prerenal azotemia
E. Renal tubular obstruction

Submit

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Blood urea nitrogen	38 mg/dL

His admission creatinine level was 0.8 mg/dL. Which of the following is the most likely cause of acute kidney injury in this patient?

- A. Acute interstitial nephritis [49%]
- B. Bladder neck obstruction [1%]
- C. Glomerular injury [12%]
- D. Prerenal azotemia [8%]
- ✔ E. Renal tubular obstruction [28%]

Omitted
Correct answer
E

28%
Answered correctly

3 Seconds
Time Spent

11/19/2018
Last Updated

Explanation

Clinical features of crystal-induced acute kidney injury	
Common etiologies	• Acyclovir • Sulfonamides • Methotrexate • Ethylene glycol • Protease inhibitors • Uric acid (tumor lysis syndrome)
Clinical presentation	• Usually asymptomatic • AKI ≤7 days of starting drug • UA: Hematuria, pyuria & crystals • Increased risk with volume depletion, CKD
Management	• Discontinuation of drug • Volume repletion • Loop diuretic

AKI = acute kidney injury; **CKD** = chronic kidney disease; **UA** = urinalysis.

AKI = acute kidney injury; **CKD** = chronic kidney disease; **UA** = urinalysis.

The patient's presentation is most likely consistent with crystal-induced acute kidney injury (AKI) due to intravenous acyclovir. The kidney rapidly excretes acyclovir into the urine, but the drug has low urine solubility. As a result, it easily precipitates in renal tubules, causing intratubular obstruction and direct renal tubular toxicity. Crystal-induced AKI is more common with large intravenous doses of acyclovir and occurs only rarely with oral acyclovir. Intravenous acyclovir less commonly causes AKI through acute tubular necrosis or acute interstitial nephritis.

Risk factors for crystal-induced AKI include underlying volume depletion or chronic kidney disease. Most patients develop AKI within 24-48 hours after drug exposure and can be asymptomatic or develop nonspecific symptoms (eg, nausea, flank/abdominal pain). Urinalysis can show hematuria, pyuria, and crystals visualized with a polarizing microscope. Treatment involves discontinuing the drug and providing volume repletion. Administration of intravenous fluids for adequate hydration while giving the drug can also prevent AKI.

(Choice A) Acute interstitial nephritis (AIN) is usually associated with exposure to medications such as beta lactams and proton pump inhibitors. However, AIN usually occurs 7-10 days after drug exposure. Patients can develop findings that include skin rash, eosinophilia, eosinophiluria, and pyuria. This patient's onset of AKI (2 days) makes AIN less likely.

(Choice B) Bladder neck obstruction due to conditions including benign prostatic hyperplasia or prostate cancer can cause post-renal AKI. However, this occurs more commonly in older men and presents with obstructive voiding symptoms. This patient's younger age and absence of symptoms of urinary obstruction make this unlikely.

(Choice C) Glomerular injury is seen in glomerulonephritis, which usually causes an abnormal urinalysis (eg, proteinuria, hematuria, red blood cell casts). Glomerulonephritis can occur after certain infections (eg, streptococcal) but is not usually associated with viral meningitis. In addition, acyclovir is more commonly associated with renal tubular than glomerular injury.

(Choice D) Pre-renal azotemia is often due to volume depletion with hypoperfusion to the kidneys. Laboratory studies typically show a blood urea nitrogen to creatinine ratio >20:1. This patient's normal blood pressure without orthostasis and ratio <20:1 make this less likely.

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Educational objective:
High-dose intravenous acyclovir can cause crystalluria with renal tubular obstruction. Administering intravenous fluids concurrently with the drug can help reduce the risk of acute kidney injury.

References

- Acyclovir nephrotoxicity: a case report highlighting the importance of prevention, detection, and treatment of acyclovir-induced nephropathy.

A 73-year-old man with dementia is brought to the emergency department by nursing home staff because he has been moaning continuously and gripping his lower abdomen for the past 36 hours. The patient is unable to give any history, but staff members say that he refused oral intake the previous day. He has had no vomiting or diarrhea. His last bowel movement was 2 days ago. The patient uses adult diapers, which were last changed 2 days ago. He has a history of benign prostatic hyperplasia, external hemorrhoids, hypertension, and hyperlipidemia. One year ago, he was hospitalized for diverticular bleeding that resolved spontaneously. His medications review shows that his primary care physician started amitriptyline 8 days ago for chronic neck pain. On physical examination, the patient is afebrile. His blood pressure is 160/70 mm Hg and his pulse is 100/min. The mucous membranes are moist. His lung fields are clear to auscultation. Palpation of the abdomen shows fullness and tenderness along the midline below the umbilicus without guarding or rigidity. Bowel sounds are heard in all 4 quadrants. Which of the following is the best initial management for this patient?

- A. Abdominal CT scan
- B. Barium enema
- C. Intravenous fluids, analgesics, and observation
- D. Upright abdominal x-ray
- E. Urgent surgical consultation
- F. Urinary catheterization

Submit

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- A. Abdominal CT scan [10%]
- B. Barium enema [2%]
- C. Intravenous fluids, analgesics, and observation [5%]
- D. Upright abdominal x-ray [17%]
- E. Urgent surgical consultation [1%]
- ✔ F. Urinary catheterization [62%]

Omitted
Correct answer
F

62%
Answered correctly

3 Seconds
Time Spent

11/14/2018
Last Updated

Explanation

This patient's abdominal pain and suprapubic fullness are consistent with amitriptyline-induced urinary retention. Amitriptyline is a tricyclic antidepressant with anticholinergic properties. The bladder empties under muscarinic control with both detrusor muscle contraction and internal urethral sphincter relaxation. Anticholinergic agents can cause urinary retention by reducing detrusor contraction and preventing urethral sphincter relaxation. In addition, elderly male patients often have some degree of underlying benign prostatic hyperplasia and are at risk for urinary retention. Both the anticholinergic properties of amitriptyline and pressure from urinary retention could have caused this patient's constipation as well.

This patient's clinical presentation is sufficient to initiate prompt urinary catheterization for suspected urinary retention. Bedside ultrasound or bladder scan (if available) can also help in diagnosis but should not delay urinary catheterization. Urinary catheterization can document a postvoid residual bladder volume (>50 mL is considered diagnostic for urinary retention) and provides symptomatic relief by draining urine from the distended bladder. This patient should also discontinue amitriptyline therapy.

(Choice A) An abdominal CT scan would reveal a distended bladder in this patient and may also show hydronephrosis and hydroureter. However, CT scans are more expensive and time-consuming than urinary catheterization and will not provide symptomatic relief.

(Choice B) Barium enemas are used to diagnose luminal abnormalities of the colon, such as colon cancer or diverticulosis.

(Choice C) Intravenous fluids, analgesics, and observation are the treatment for nephrolithiasis (kidney stones). Patients with kidney stones typically present with intense flank pain and hematuria instead of suprapubic fullness. Intravenous fluids could potentially worsen this patient's obstructive urinary symptoms.

(Choice D) Upright abdominal x-ray is not as reliable for evaluating urinary retention as it may not show a distended bladder (unless obstructed by a bladder stone). Abdominal x-rays are more useful for diagnosing ileus or small-bowel obstruction. Amitriptyline may cause ileus, but these patients typically develop nausea, vomiting, hypoactive bowel sounds, distended abdomen, diffuse mild

hydroureter. However, CT scans are more expensive and time-consuming than urinary catheterization and will not provide symptomatic relief.

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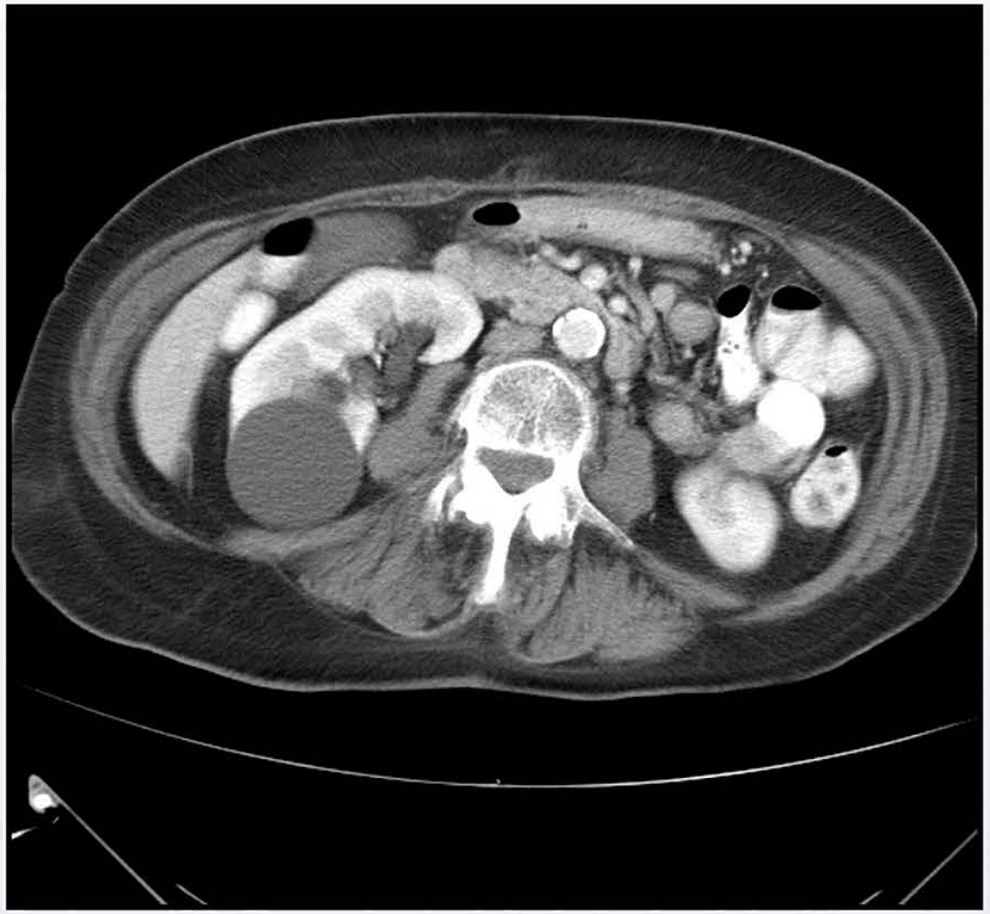
(Choice E) Surgical consultation is indicated for suspected acute abdomen (eg, bowel perforation, mechanical obstruction). Obstruction typically presents with vomiting, moderate-to-severe abdominal pain, hyperactive bowel sounds, possible peritoneal signs (eg, guarding), and dilated bowel loops with air fluid levels on imaging. Perforation usually has absent bowel sounds. This patient's absence of guarding/rigidity or gastrointestinal symptoms (eg, vomiting) makes these less likely.

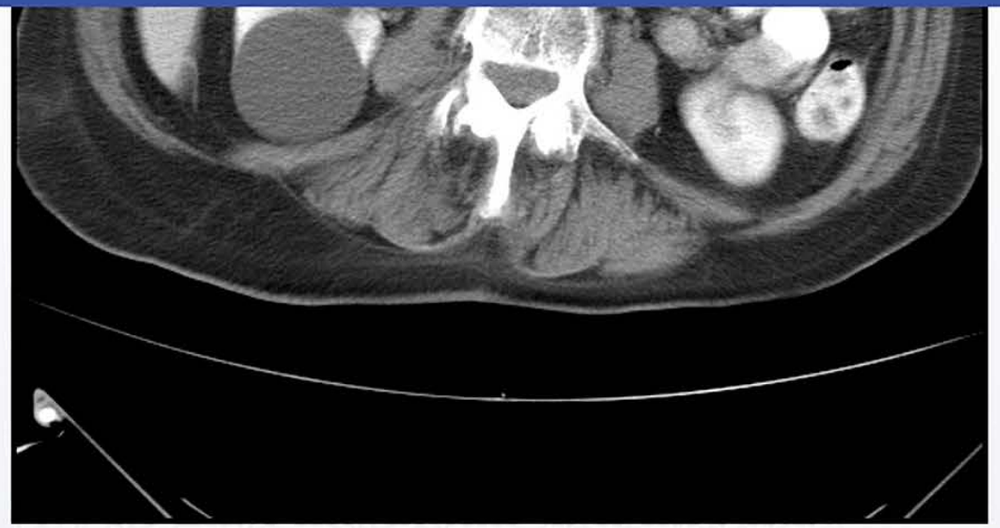
Educational objective:
Drugs with anticholinergic properties can cause acute urinary retention by preventing detrusor muscle contraction and urinary sphincter relaxation. The treatment involves urinary catheterization and discontinuing the medication.

References

- Management of acute urinary retention.

A 54-year-old man comes to the emergency department with cramping lower abdominal pain, mild nausea, and 2 episodes of watery diarrhea. He has no fever, vomiting, or urinary symptoms. His past medical history is unremarkable. The patient's father died at age 60 from abdominal aortic aneurysm rupture. He is a lifetime nonsmoker. His vital signs are normal. CT scan of the abdomen with contrast is shown in the image below.





The abdominal pain resolves in 2 hours without any intervention and the patient wants to go home. Which of the following is the most appropriate management for the renal findings on the CT scan?

- A. Antibiotics
- B. Percutaneous aspiration
- C. Reassurance only
- D. Surgical excision
- E. Ureteral stent placement

Submit

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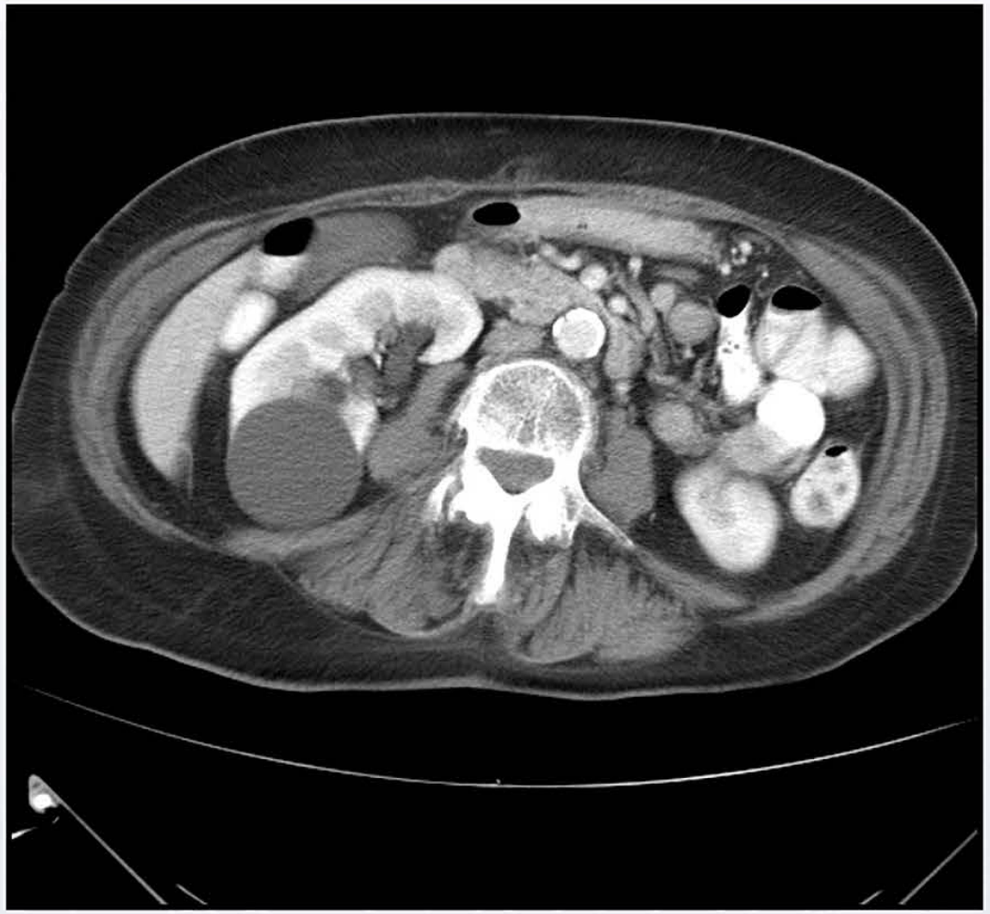

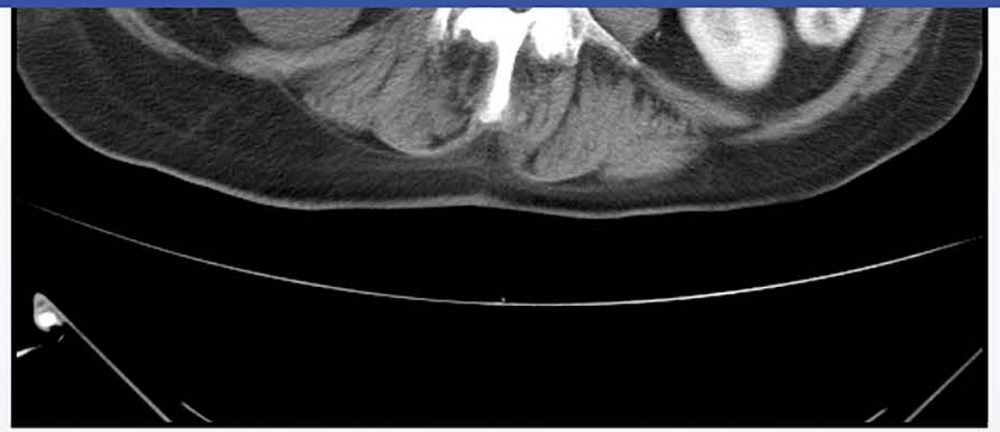

The abdominal pain resolves in 2 hours without any intervention and the patient wants to go home. Which of the following is the most appropriate management for the renal findings on the CT scan?

- A. Antibiotics [2%]
- B. Percutaneous aspiration [22%]
- ✔ C. Reassurance only [39%]
- D. Surgical excision [28%]
- E. Ureteral stent placement [6%]

Omitted
Correct answer
C

39%
Answered correctly

5 Seconds
Time Spent

10/29/2018
Last Updated

Explanation

Characteristics of renal cysts	
Simple renal cyst	**Malignant cystic mass**
Thin, smooth, regular wall	**Thick, irregular wall**
Unilocular	Multilocular
No septae	Multiple **septae**, occasionally thick & calcified
Homogenous content	Heterogenous content (solid & cystic)
Absence of contrast enhancement on CT/MRI	Presence of **contrast enhancement** on CT/MRI
Usually asymptomatic	May cause pain, hematuria, or hypertension
No follow-up needed	Requires follow-up imaging & urological evaluation for malignancy

This patient's abdominal CT scan shows a **simple renal cyst**. Such cysts are most commonly seen in patients age >50. They are benign and often discovered incidentally by radiological examination. Most of the time, cysts do not cause hypertension, flank pain, hematuria, or proteinuria; infection occurs rarely.

When a renal cyst is found on imaging, it is often possible to differentiate between a simple cyst and a malignant cystic mass based on the radiological features. Features suggesting malignancy include irregular or multilocular structure with multiple septations, heterogeneous content, and contrast enhancement on CT or MRI. Incidentally discovered cysts with benign features require no additional follow-up evaluation or imaging, and the patient may be reassured.

Usually asymptomatic	May cause pain, hematuria, or hypertension
No follow-up needed	Requires follow-up imaging & urological evaluation for malignancy

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(Choice A) Antibiotics are not indicated as there is no evidence of bacterial infection (either gastrointestinal or genitourinary).

(Choice B) Percutaneous aspiration of the cyst is not indicated as the patient has no symptoms from it. Percutaneous aspiration may be considered if the cyst is large and painful or infected with purulent material.

(Choice D) Surgical excision or nephrectomy is not indicated as this patient has a simple benign cyst with good prognosis.

(Choice E) Ureteral stent placement is indicated when there is urinary obstruction at the ureter or renal pelvis. Dilation of the renal pelvis **(hydronephrosis)** or ureter (hydroureter) is a typical sign of urinary obstruction. This patient has no symptoms of urinary obstruction or radiological signs of obstruction on CT scan.

Educational objective:
Simple renal cysts are almost always benign and do not require further evaluation. Features concerning for malignant renal mass include a multilocular mass, irregular walls, thickened septae, and contrast enhancement.

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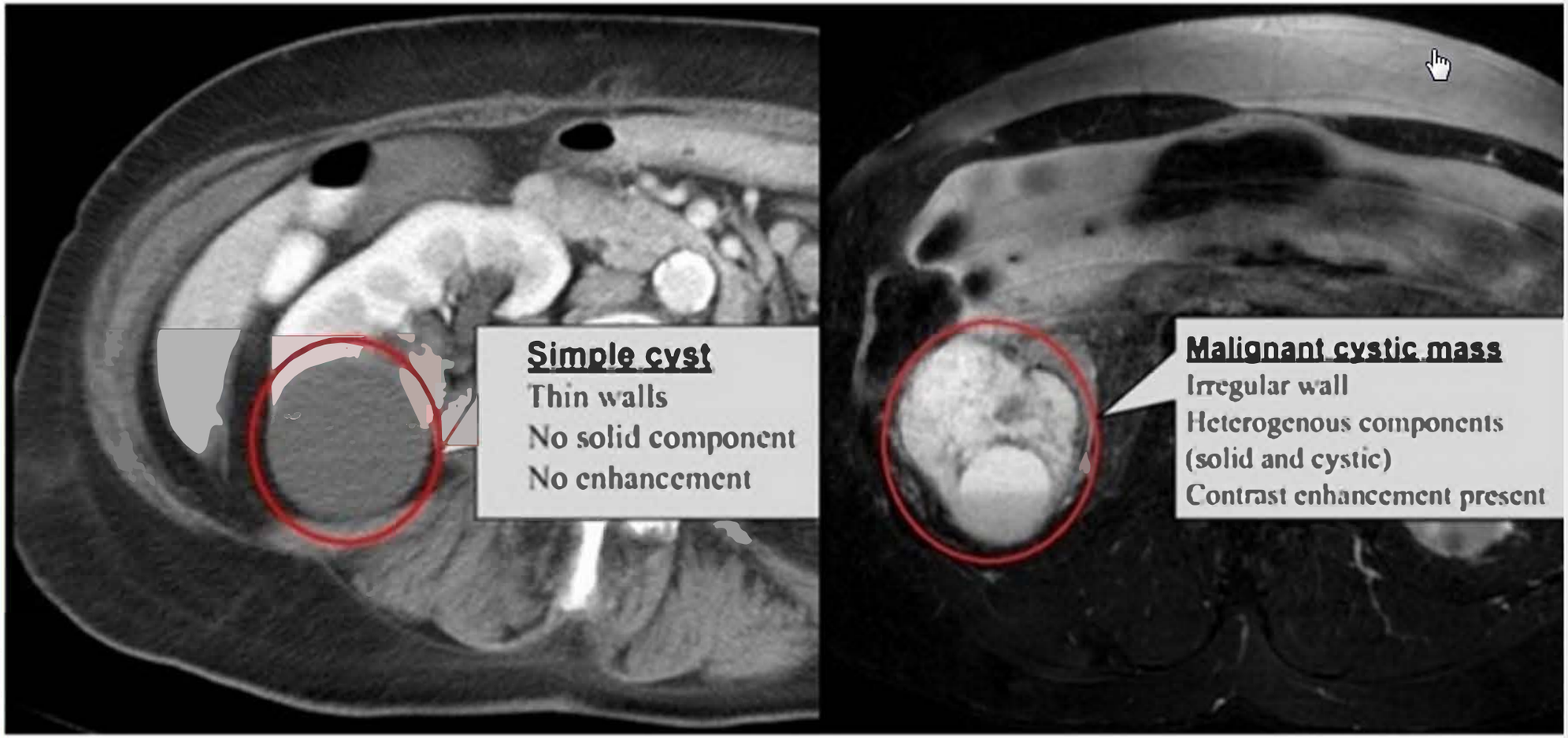

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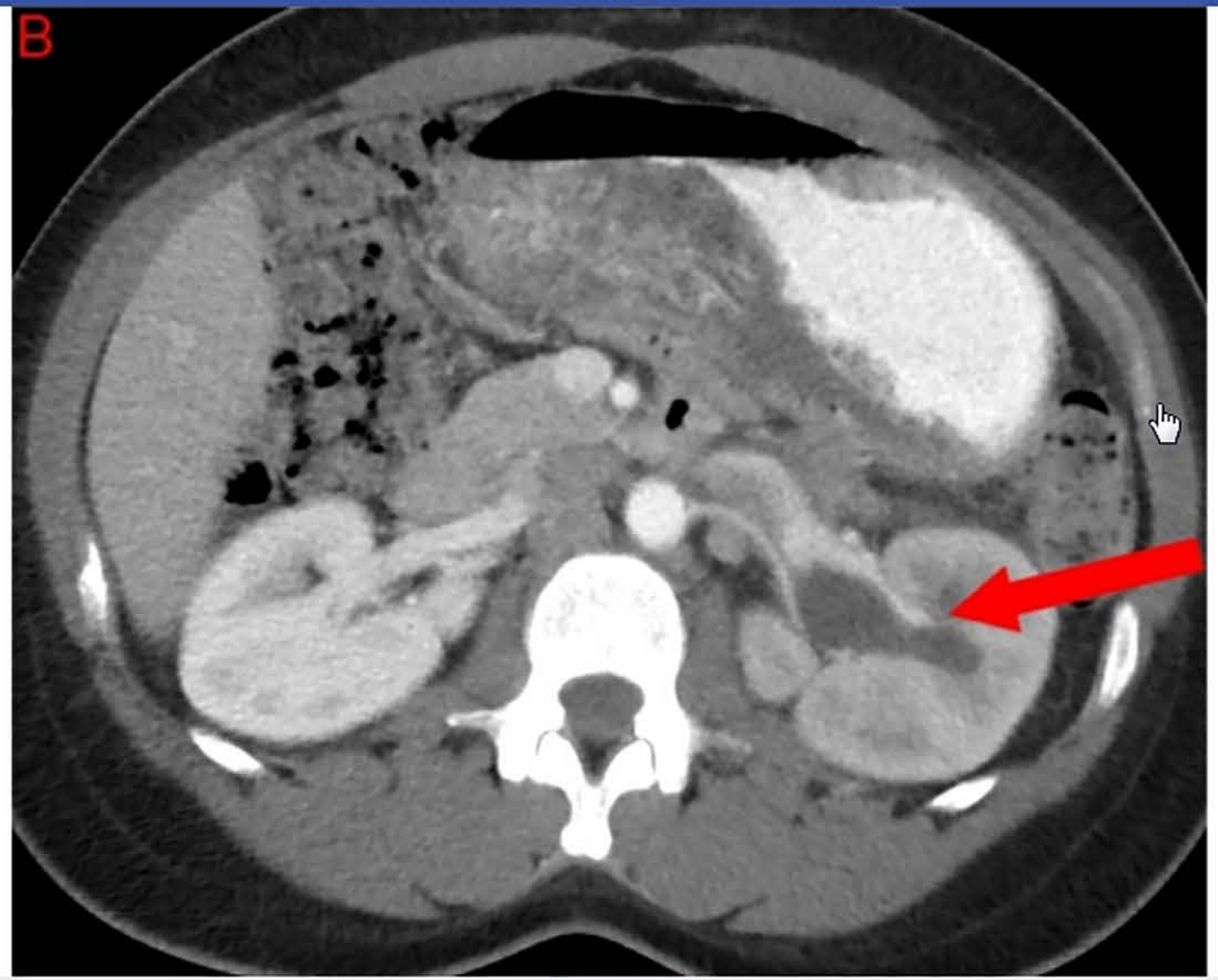

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A 70-year-old woman is brought to the emergency department by her daughter due to lethargy. The patient has a history of mild cognitive impairment but is able to perform daily activities by herself and lives alone. She experienced a minor febrile illness with upper respiratory infection several days ago, and her primary care physician advised symptomatic treatment. Since then, the patient has reported generalized weakness and decreased appetite to the daughter. Today, the patient did not answer her phone and the daughter found her lethargic and confused. The patient takes no medications and does not use tobacco or alcohol. Temperature is 37.2 C (99 F), blood pressure is 92/50 mm Hg, pulse is 110/min, and respirations are 18/min. Physical examination shows dry oral mucosa. Lung auscultation reveals right-sided bronchial breath sounds and crackles. Laboratory results are as follows:

Hemoglobin	9.8 g/dL
Leukocytes	14,000/mm^3
Serum sodium	147 mEq/L
Blood urea nitrogen	70 mg/dL
Serum creatinine	1.8 mg/dL
Serum albumin	2.8 g/dL

Chest radiography reveals a right lower lobe consolidation, and empiric antibiotics are started. Which of the following is the most appropriate next step in management of this patient's renal insufficiency?

A. Albumin infusion
B. Blood transfusion
C. Dopamine infusion
D. Intravenous furosemide

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A. Albumin infusion
B. Blood transfusion
C. Dopamine infusion
D. Intravenous furosemide
E. Intravenous normal saline
F. Urgent hemodialysis

Submit

A 70-year-old woman is brought to the emergency department by her daughter due to lethargy. The patient has a history of mild cognitive impairment but is able to perform daily activities by herself and lives alone. She experienced a minor febrile illness with upper respiratory infection several days ago, and her primary care physician advised symptomatic treatment. Since then, the patient has reported generalized weakness and decreased appetite to the daughter. Today, the patient did not answer her phone and the daughter found her lethargic and confused. The patient takes no medications and does not use tobacco or alcohol. Temperature is 37.2 C (99 F), blood pressure is 92/50 mm Hg, pulse is 110/min, and respirations are 18/min. Physical examination shows dry oral mucosa. Lung auscultation reveals right-sided bronchial breath sounds and crackles. Laboratory results are as follows:

Hemoglobin	9.8 g/dL
Leukocytes	14,000/mm^3
Serum sodium	147 mEq/L
Blood urea nitrogen	70 mg/dL
Serum creatinine	1.8 mg/dL
Serum albumin	2.8 g/dL

Chest radiography reveals a right lower lobe consolidation, and empiric antibiotics are started. Which of the following is the most appropriate next step in management of this patient's renal insufficiency?

A. Albumin infusion [1%]
B. Blood transfusion [0%]
C. Dopamine infusion [0%]
D. Intravenous furosemide [1%]

Hemoglobin	9.8 g/dL
Leukocytes	14,000/mm³
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- A. Albumin infusion [1%]
- B. Blood transfusion [0%]
- C. Dopamine infusion [0%]
- D. Intravenous furosemide [1%]
- ✔ E. Intravenous normal saline [87%]
- F. Urgent hemodialysis [8%]

Omitted
Correct answer
E

87%
Answered correctly

6 Seconds
Time Spent

12/28/2018
Last Updated

Explanation

Prerenal acute kidney injury	

Etiology	• Decreased renal perfusion
	  ◦ True volume depletion
	  ◦ Decreased EABV (eg, heart failure, cirrhosis)
	  ◦ Displacement of intravascular fluid (eg, sepsis, pancreatitis)
	  ◦ Renal artery stenosis
	  ◦ Afferent arteriole vasoconstriction (eg, NSAIDs)

Clinical features	• Increase in serum creatinine (eg, 50% from baseline) • Decreased urine output • Blood urea nitrogen/creatinine ratio >20:1 • Fractional excretion of sodium <1% • Unremarkable ("bland") urine sediment
Treatment	• Restoration of renal perfusion

EABV = effective arterial blood volume; **NSAIDs** = nonsteroidal anti-inflammatory drugs.

This patient's clinical presentation - altered mental status, tachycardia, hypotension, leukocytosis, and evidence of right lower lobe consolidation on chest x-ray - is consistent with **sepsis** due to **community-acquired pneumonia**. The systemic inflammatory response associated with sepsis leads to **hypotension** (due to peripheral vasodilation) and **intravascular volume depletion** (due to vascular leakage). Although the effects of sepsis on the kidneys are complex, much of this patient's impaired renal function most likely represents **prerenal acute kidney injury** (AKI) due to **decreased renal perfusion**.

vascular leakage). Although the effects of sepsis on the kidneys are complex, much of this patient's impaired renal function most likely represents **prerenal acute kidney injury** (AKI) due to **decreased renal perfusion**.

Renal hypoperfusion leads to a decrease in glomerular filtration rate and a consequent increase in serum creatinine. Passive resorption of urea is increased (due to an increase in renal tubular sodium and water resorption), leading to the typical finding of **blood urea nitrogen/creatinine ratio >20:1**. There is no intrinsic kidney damage in pure prerenal AKI; however, if renal hypoperfusion persists, renal ischemia will lead to acute tubular necrosis. Therefore, patients require volume resuscitation with **intravenous normal saline** as soon as possible to restore adequate renal perfusion.

(Choice A) Albumin infusion is indicated for intravascular volume expansion in specific circumstances (eg, prevention of prerenal AKI following large-volume paracentesis in patients with decompensated cirrhosis). However, due to similar efficacy and lower cost, isotonic saline is preferred over albumin for volume resuscitation in patients with hypovolemia or sepsis.

(Choice B) Although maintaining adequate oxygen delivery to the kidneys is important, red blood cell transfusion is generally not recommended in septic patients with a hemoglobin ≥7 g/dL and may result in inferior outcomes (eg, increased mortality).

(Choice C) Low-dose dopamine infusion primarily acts to stimulate dopamine-1 receptors, resulting in renal arteriole dilation and increased renal perfusion. However, volume resuscitation is the best initial step in this septic patient.

(Choice D) Diuretics (eg, furosemide) are contraindicated in this patient as they will worsen intravascular volume depletion and potentially lead to circulatory collapse.

(Choice F) This patient is likely to demonstrate improved renal function following administration of normal saline; therefore, hemodialysis should not be considered at this time.

Educational objective:
Sepsis leads to hypotension and intravascular volume depletion, and is a common cause of prerenal acute kidney injury. Volume resuscitation with intravenous normal saline is needed to restore renal perfusion and prevent development of acute tubular necrosis.

A 50-year-old male presents with polyuria and polydipsia. He has smoked 1 pack of cigarettes daily for the past 30 years. He denies having any past medical problems. His mother and one maternal uncle are diabetic. His height is 5'8" (172 cm), weight is 180 lbs (81.6 kg), temperature is 37° C (98.6° F), pulse is 75/min, blood pressure is 150/90 mm Hg, and respirations are 15/min. Examination of all the systems is unremarkable. Chemistry panel shows:

Sodium	140 mEq/L
Potassium	4.1 mEq/L
Bicarbonate	26 mEq/L
Blood glucose	210 mg/dL
BUN	12 mg/dL
Creatinine	0.9 mg/dL

The patient is diagnosed with type 2 diabetes mellitus. He is advised exercise and dietary modification. He is referred to an ophthalmologist and appropriately screened for diabetic retinopathy. What is the most sensitive test to screen for nephropathy in this patient?

A. Creatinine clearance
B. Dipstick testing of urine for protein
C. Random urine for microalbumin/creatinine ratio
D. Renal ultrasound
E. Oral glucose tolerance test

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The patient is diagnosed with type 2 diabetes mellitus. He is advised exercise and dietary modification. He is referred to an ophthalmologist and appropriately screened for diabetic retinopathy. What is the most sensitive test to screen for nephropathy in this patient?

- A. Creatinine clearance [5%]
- B. Dipstick testing of urine for protein [14%]
- ✔ C. Random urine for microalbumin/creatinine ratio [76%]
- D. Renal ultrasound [1%]
- E. Oral glucose tolerance test [1%]

Sodium	140 mEq/L
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- E. Oral glucose tolerance test [1%]

Omitted
Correct answer
C

76%
Answered correctly

4 Seconds
Time Spent

10/10/2018
Last Updated

Explanation

Development of nephropathy is preceded by development of excessive protein excretion, the initial stages of which is termed microalbuminuria. Patients with microalbuminuria typically have a urine albumin excretion value between 30-300 mg/24 hr. (Normal urine protein excretion is < 30 mg/24 hr). Spot urine collection and timed urine collection for the measurement of urine microalbumin to creatinine ratio are generally accepted as good screening methods for microalbuminuria. Although 24-hour urine collection is slightly more accurate in screening for microalbuminuria, its inconvenience to patients makes it less preferred by physicians.

(Choice A) During the initial phases of diabetic nephropathy, there is glomerular hyperfiltration and an increase in creatinine clearance. Creatinine clearance then declines with the progression of diabetic nephropathy; however, it can still be relatively normal with proteinuria (micro and macroalbuminuria) due to high initial values. Low creatinine clearance occurs when the renal damage is fairly advanced. Due to these reasons, creatinine clearance is not used as a screening tool for diabetic nephropathy.

(Choice B) Routine dipstick testing is not recommended during the initial stages of nephropathy. Dipsticks can only detect excessive urinary protein excretion when the level is > 300 mg/24 hr (macroalbuminuria).

(Choice D) Ultrasound is not useful as a screening tool for diabetic nephropathy. The kidney size is relatively preserved until advanced renal failure occurs. Ultrasound should be considered if non-diabetic renal damage (e.g., obstructive uropathy) is suspected clinically.

(Choice E) Oral glucose tolerance testing has no role in screening patients for diabetic nephropathy.

Educational Objective:
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A 23-month-old girl is brought to the emergency department with fever and abdominal pain. She was in her usual state of health until 2 days earlier, when she cried while urinating. Today her parents saw a small amount of blood in the urine, which they said smelled foul. The patient's temperature is 38.9 C (102 F), blood pressure is 96/62 mm Hg, pulse is 130/min, and respirations are 20/min. Physical examination shows suprapubic tenderness and left costovertebral angle tenderness. Complete blood count shows leukocytosis. Serum creatine and electrolytes are normal. Catheterized urinalysis results are as follows:

Specific gravity	1.013
Protein	None
Blood	Moderate
Glucose	Negative
Ketones	Negative
Leukocyte esterase	Positive
Nitrites	Positive
Bacteria	Moderate
White blood cells	50+/hpf
Red blood cells	20-30/hpf
Casts	None
Crystals	None

A urine culture grows 100,000 colony-forming units/mL of *Escherichia coli*. The girl is started on antibiotics, and her fever and pain

Nitrites	Positive
Bacteria	Moderate
White blood cells	50+/hpf
Red blood cells	20-30/hpf
Casts	None
Crystals	None

A urine culture grows 100,000 colony-forming units/mL of *Escherichia coli*. The girl is started on antibiotics, and her fever and pain resolve on the second day of hospitalization. In addition to completing the current course of antibiotics, which of the following is the most appropriate next step in management of this patient?

- A. No further studies
- B. Renal and bladder ultrasound
- C. Repeat complete blood count
- D. Repeat urine culture
- E. Start daily antibiotic prophylaxis
- F. Voiding cystourethrogram

Submit

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Crystals	None

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- A. No further studies [20%]
- ✔ B. Renal and bladder ultrasound [48%]
- C. Repeat complete blood count [1%]
- D. Repeat urine culture [8%]
- E. Start daily antibiotic prophylaxis [0%]
- F. Voiding cystourethrogram [21%]

Omitted
Correct answer
B

48%
Answered correctly

5 Seconds
Time Spent

07/15/2018
Last Updated

Explanation

Indications for renal & bladder ultrasound

Explanation

Indications for renal & bladder ultrasound

- Infants and children age < 24 months with a first febrile UTI
- Recurrent febrile UTIs in children of any age
- UTI in a child of any age with a family history of renal or urologic disease, hypertension, or poor growth
- Children who do not respond to appropriate antibiotic treatment

This child's clinical presentation and laboratory studies are consistent with her first urinary tract infection (UTI). The fever, dysuria, and suprapubic/flank pain are suggestive of renal involvement. **Pyuria** (≥5 white blood cells/hpf) and **bacteriuria** (50,000 colony-forming units/mL from a catheterized specimen) confirm the infection. Children **age <2 years** are at increased risk of complications from UTI and should be treated with 1-2 weeks of antibiotics. In addition, all children age <2 years with a first febrile UTI should undergo a **renal and bladder ultrasound** to evaluate for any anatomic abnormalities that might predispose the child to UTIs. Ideally, the ultrasound is performed after improvement of fever and symptoms to minimize false positive results from acute inflammation. If the patient has persistent or worsening symptoms, an ultrasound should be performed immediately to assess for renal abscess.

(Choice A) Older children and adults generally do not need further evaluation of a first-time UTI due to lower likelihood of predisposing anatomic issues, lower risk of complications, and lower risk of recurrent UTI.

(Choices C and D) Repeat blood work and urine culture should be performed only in children who fail to improve after 2-3 days of appropriate antibiotics. These studies are not indicated to prove a "cure" in children with obvious symptomatic improvement.

(Choice E) Daily prophylactic antibiotics can be considered in patients with recurrent UTIs or evidence of high-grade vesicoureteral

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(Choice E) Daily prophylactic antibiotics can be considered in patients with recurrent UTIs or evidence of high-grade vesicoureteral reflux. They are generally not indicated in children with a first febrile UTI.

(Choice F) A voiding cystourethrogram can be considered if hydronephrosis or scarring is seen in renal ultrasound. It is also indicated in newborns age <1 month and children age <2 years with recurrent UTIs or a first UTI from an organism other than *Escherichia coli*.

Educational objective:
Children age <2 years with a first febrile urinary tract infection (UTI) should be treated with 1-2 weeks of antibiotics. A renal and bladder ultrasound should be ordered to evaluate for abnormalities that lead to recurrent UTIs. Voiding cystourethrogram is generally not indicated for a first febrile UTI unless there are abnormalities on renal ultrasound or if the patient is a neonate.

References

A 68-year-old man with no previous history of renal disease is evaluated in the postoperative unit due to decreased urine output. The patient was admitted due to severe abdominal pain, fever, and vomiting. Evaluation revealed perforated sigmoid diverticulitis, and the patient underwent urgent sigmoid resection. Postoperatively, he has received maintenance intravenous fluids and piperacillin/tazobactam. In the day since surgery, the patient has had 200 mL of urine output. Temperature is 37.1 C (98.8 F), blood pressure is 100/70 mm Hg, and pulse is 96/min. The lungs are clear to auscultation, and heart sounds are normal. The abdomen shows postoperative changes with sluggish bowel sounds. The patient has no skin rash. Laboratory results are as follows:

Hemoglobin	10.5 g/dL
Leukocytes	13,000/mm^3
Sodium	138 mg/dL
Potassium	5.1 mg/dL
Glucose	108 mg/dL
Creatinine	1.9 mg/dL
Blood urea nitrogen	82 mg/dL

Urinalysis reveals no sediment, and bladder scanning reveals no urine. Which of the following is the best next step in management of this patient?

A. Bolus of isotonic saline
B. Discontinuation of antibiotics
C. Dopamine infusion
D. Intravenous furosemide

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- E. Intravenous mannitol
- F. Intravenous urography

Submit

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Urinalysis reveals no sediment, and bladder scanning reveals no urine. Which of the following is the best next step in management of this patient?

- A. Bolus of isotonic saline [70%]
- B. Discontinuation of antibiotics [4%]
- C. Dopamine infusion [2%]
- D. Intravenous furosemide [5%]

Glucose	108 mg/dL
Creatinine	1.9 mg/dL
Blood urea nitrogen	82 mg/dL

Urinalysis reveals no sediment, and bladder scanning reveals no urine. Which of the following is the best next step in management of this patient?

- A. Bolus of isotonic saline [70%] ✔
- B. Discontinuation of antibiotics [4%]
- C. Dopamine infusion [2%]
- D. Intravenous furosemide [5%]
- E. Intravenous mannitol [1%]
- F. Intravenous urography [14%]

Omitted
Correct answer
A

70%
Answered correctly

4 Seconds
Time Spent

12/09/2018
Last Updated

Explanation

Explanation

Prerenal acute kidney injury	

Etiology	• Decreased renal perfusion
	  ◦ True volume depletion
	  ◦ Decreased EABV (eg, heart failure, cirrhosis)
	  ◦ Displacement of intravascular fluid (eg, sepsis, pancreatitis)
	  ◦ Renal artery stenosis
	  ◦ Afferent arteriole vasoconstriction (eg, NSAIDs)

Clinical features	• Increase in serum creatinine (eg, 50% from baseline) • Decreased urine output • Blood urea nitrogen/creatinine ratio >20:1 • Fractional excretion of sodium <1% • Unremarkable ("bland") urine sediment
Treatment	• Restoration of renal perfusion

EABV = effective arterial blood volume; **NSAIDs** = nonsteroidal anti-inflammatory drugs.

This patient with elevated serum creatinine and no underlying renal disease likely has **prerenal acute kidney injury (AKI)** due to **intravascular volume depletion**. Preoperative infection and intraoperative blood loss predispose patients to volume depletion, and volume depletion is further suggested by tachycardia. There is no intrinsic damage to the kidneys in prerenal AKI, but decreased renal perfusion leads to a decreased glomerular filtration rate and increased serum creatinine. The kidneys increase tubular sodium and water resorption, resulting in markedly increased passive resorption of urea. Therefore, a **blood urea nitrogen/creatinine ratio**

This patient with elevated serum creatinine and no underlying renal disease likely has **prerenal acute kidney injury (AKI)** due to **intravascular volume depletion**. Preoperative infection and intraoperative blood loss predispose patients to volume depletion, and volume depletion is further suggested by tachycardia. There is no intrinsic damage to the kidneys in prerenal AKI, but decreased renal perfusion leads to a decreased glomerular filtration rate and increased serum creatinine. The kidneys increase tubular sodium and water resorption, resulting in markedly increased passive resorption of urea. Therefore, a **blood urea nitrogen/creatinine ratio >20:1** (43:1 in this patient) is highly suggestive of a prerenal state. **Oliguria** (<500 mL of urine/24 hours) is often present, as is **unremarkable urine sediment** (absence of casts, cells, or protein).

Although heart failure can lead to prerenal AKI in the setting of hypervolemia (due to decreased effective arterial blood volume), most prerenal AKI is due to a volume-depleted state. In the absence of clear signs of volume overload (eg, elevated jugular venous pressure, lung crackles), patients with suspected prerenal AKI should be treated with **intravenous isotonic fluid** (eg, normal saline) to restore renal perfusion.

(Choice B) Beta-lactam antibiotics (eg, piperacillin/tazobactam) are a common cause of intrinsic AKI due to acute interstitial nephritis (AIN). AIN necessitates discontinuation of the offending drug; however, no leukocytes on urinalysis and absence of skin rash make AIN unlikely.

(Choice C) Low-dose dopamine acts primarily as a dopamine-1 receptor agonist, resulting in renal arteriole dilation and increased renal blood flow. However, fluid bolus is the best treatment in this patient with evidence of hypovolemia.

(Choices D and E) Loop diuretics (eg, furosemide) or osmotic diuretics (eg, mannitol) would worsen this patient's volume depletion and possibly lead to intrinsic renal injury (eg, acute tubular necrosis) due to a further decrease in renal perfusion.

(Choice F) Intravenous urography visualizes radiopaque contrast filling the urinary tract and is useful in diagnosing ureteral injury. Iatrogenic ureteral injury can occur following abdominal surgery; however, unilateral injury does not result in AKI, and bilateral injury (a potential cause of postrenal AKI) is extremely unlikely.

Educational objective:

(Choice C) Low-dose dopamine acts primarily as a dopamine-1 receptor agonist, resulting in renal arteriole dilation and increased renal blood flow. However, fluid bolus is the best treatment in this patient with evidence of hypovolemia.

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Educational objective:
Intravascular volume depletion is a common cause of prerenal acute kidney injury (AKI). Patients typically have an elevated blood urea nitrogen/creatinine ratio (>20:1), oliguria, and unremarkable urine sediment. Administration of intravenous fluid restores renal perfusion and corrects the AKI.

References

- Clinical scenarios in acute kidney injury: pre-renal acute kidney injury.