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Tutorial

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A 44-year-old man is evaluated in the clinic for occasional chest discomfort that is not consistently related to exertion. The patient's past medical history is significant for hypertension and hyperlipidemia. His grandfather experienced a myocardial infarction at age 50. Coronary CT angiography reveals several nonobstructive atherosclerotic plaques in the coronary arteries. One plaque in the proximal left anterior descending artery appears extensive, has a large hypodense core, and occupies 40% of the lumen. No intervention is performed. One year later, the patient comes to the emergency department with acute severe chest pain and is found to have thrombotic occlusion of the proximal left anterior descending artery. High intraplaque activity of which of the following enzymes most likely resulted in this patient's myocardial infarction?

A. Hydroxymethylglutaryl CoA reductase

B. Lysyl oxidase

C. Metalloproteinases

D. Procollagen peptidases

E. Prolyl hydroxylase

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A. Hydroxymethylglutaryl CoA reductase [16%]

B. Lysyl oxidase [10%]

C. Metalloproteinases [59%]

D. Procollagen peptidases [9%]

E. Prolyl hydroxylase [4%]

Omitted

Correct answer
C

59%
Answered correctly

17 Seconds
Time Spent

10/03/2018
Last Updated

Explanation

Stable angina

Acute coronary syndrome

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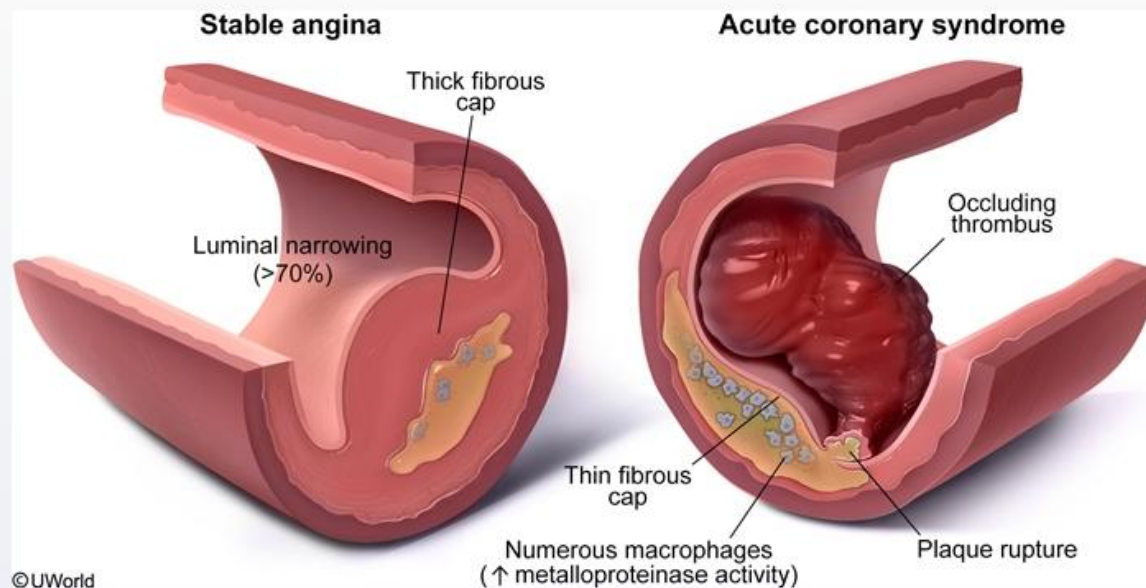
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Windows Taskbar

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Explanation



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This patient with coronary atherosclerosis has likely developed plaque rupture with thrombotic occlusion of the left anterior descending artery, resulting in acute coronary syndrome (unstable angina, myocardial infarction).

Patients with coronary **atherosclerosis** can be asymptomatic but typically develop symptoms of stable angina (exertional chest pain relieved with rest/nitroglycerin) if progressive enlargement of the atherosclerotic plaque causes >70% luminal stenosis. **Acute coronary syndrome** usually occurs due to **plaque rupture**, which leads to superimposed **thrombosis** and vessel occlusion. The likelihood of plaque rupture is typically related to plaque stability rather than plaque size or degree of luminal narrowing. Plaque stability largely depends on the mechanical strength of

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This patient with coronary atherosclerosis has likely developed plaque rupture with thrombotic occlusion of the left anterior descending artery, resulting in acute coronary syndrome (unstable angina, myocardial infarction).

Patients with coronary **atherosclerosis** can be asymptomatic but typically develop symptoms of stable angina (exertional chest pain relieved with rest/nitroglycerin) if progressive enlargement of the atherosclerotic plaque causes >70% luminal stenosis. **Acute coronary syndrome** usually occurs due to **plaque rupture**, which leads to superimposed **thrombosis** and vessel occlusion. The likelihood of plaque rupture is typically related to plaque stability rather than plaque size or degree of luminal narrowing. Plaque stability largely depends on the mechanical strength of the fibrous cap: Thin-cap fibroatheromas are generally unstable and more vulnerable to rupture.

During the chronic inflammatory progression of an atheroma, the fibrous cap is continually being remodeled. The balance of collagen synthesis and degradation determines the mechanical strength of the cap. **Thin-cap fibroatheromas** are characterized by a large necrotic core covered by a thin fibrous cap. Activated macrophages infiltrating the atheroma contribute to the breakdown of extracellular matrix proteins (eg, collagen) by secreting **metalloproteinases**. Ongoing intimal inflammation can destabilize the mechanical integrity of the plaque through release of these metalloproteinases, resulting in plaque rupture and consequent acute coronary syndrome.

(Choice A) Hydroxymethylglutaryl (HMG) CoA reductase is the rate-limiting enzyme in cholesterol synthesis and is mostly expressed in the liver, intestine, adrenal cortex, and reproductive tissues. Patients with hyperlipidemia are typically treated with statins (eg, atorvastatin), which competitively inhibit HMG CoA reductase.

(Choice B) Lysyl oxidase strengthens extracellular collagen fibers by mediating cross-link formation between lysine and hydroxylysine residues (requires copper). Therefore, a high activity of lysyl oxidase would favor atheroma stabilization by strengthening collagen in the fibrous cap.

(Choice D) Procollagen peptidase cleaves the terminal regions of procollagen molecules exocytosed by fibroblasts or smooth muscle cells, transforming procollagen into insoluble tropocollagen. Tropocollagen then aggregates to form collagen fibrils.

(Choice E) Prolyl hydroxylase is responsible for the hydroxylation of proline on procollagen chains (requires vitamin C), which is a necessary step in the formation of a stable collagen triple helix.

Educational objective:

The likelihood of plaque rupture is related to plaque stability rather than plaque size or the degree of luminal narrowing. Plaque stability largely

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and degradation determines the mechanical strength of the cap. **Thin-cap fibroatheromas** are characterized by a large necrotic core covered by a thin fibrous cap. Activated macrophages infiltrating the atheroma contribute to the breakdown of extracellular matrix proteins (eg, collagen) by secreting **metalloproteinases**. Ongoing intimal inflammation can destabilize the mechanical integrity of the plaque through release of these metalloproteinases, resulting in plaque rupture and consequent acute coronary syndrome.

(Choice A) Hydroxymethylglutaryl (HMG) CoA reductase is the rate-limiting enzyme in cholesterol synthesis and is mostly expressed in the liver, intestine, adrenal cortex, and reproductive tissues. Patients with hyperlipidemia are typically treated with statins (eg, atorvastatin), which competitively inhibit HMG CoA reductase.

(Choice B) Lysyl oxidase strengthens extracellular collagen fibers by mediating cross-link formation between lysine and hydroxylysine residues (requires copper). Therefore, a high activity of lysyl oxidase would favor atheroma stabilization by strengthening collagen in the fibrous cap.

(Choice D) Procollagen peptidase cleaves the terminal regions of procollagen molecules exocytosed by fibroblasts or smooth muscle cells, transforming procollagen into insoluble tropocollagen. Tropocollagen then aggregates to form collagen fibrils.

(Choice E) Prolyl hydroxylase is responsible for the hydroxylation of proline on procollagen chains (requires vitamin C), which is a necessary step in the formation of a stable collagen triple helix.

Educational objective:

The likelihood of plaque rupture is related to plaque stability rather than plaque size or the degree of luminal narrowing. Plaque stability largely depends on the mechanical strength of the fibrous cap. Inflammatory macrophages in the intima may reduce plaque stability by secreting metalloproteinases, which degrade extracellular matrix proteins (eg, collagen).

References

- Evidence for increased collagenolysis by interstitial collagenases-1 and -3 in vulnerable human atheromatous plaques.
- Inflammation in atherosclerosis.

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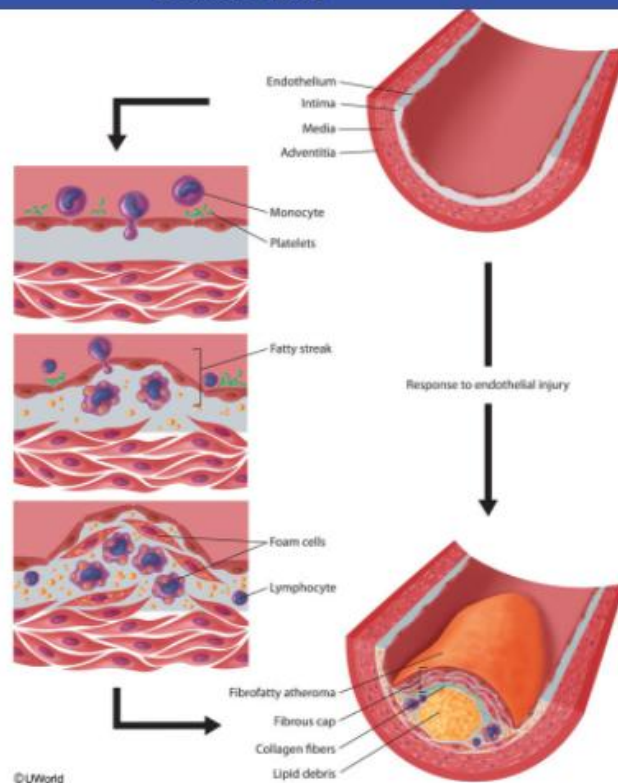
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Windows Taskbar

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Exhibit Display



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Zoom In

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(Choice A) Hydroxymethylglutaryl (HMG) CoA reductase is the rate limiting enzyme in cholesterol synthesis and is mostly expressed in the liver.

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An 82-year-old man comes to the office due to progressive dyspnea and fatigue over the last year, which now limit his daily activities. He has also noticed bilateral swelling of his feet. The patient has hypertension, which is controlled with amlodipine. His blood pressure is 122/72 mm Hg and pulse is 55/min. Physical examination reveals elevated jugular venous pressure with rapid 'y' descent and a prominent S4. Abdominal examination shows moderate ascites. The patient has 3+ bilateral lower extremity pitting edema. Echocardiogram reveals left atrial enlargement with marked left ventricular hypertrophy and normal left ventricular ejection fraction. Complete blood count, basic metabolic panel, and serum and urine protein electrophoresis are within normal limits. Endomyocardial biopsy findings are shown below.

Which of the following is the most likely diagnosis for this patient?

A. Cardiac myxoma

B. Cardiac sarcoidosis

C. Chagas disease

D. Hemochromatosis

E. Senile amyloidosis

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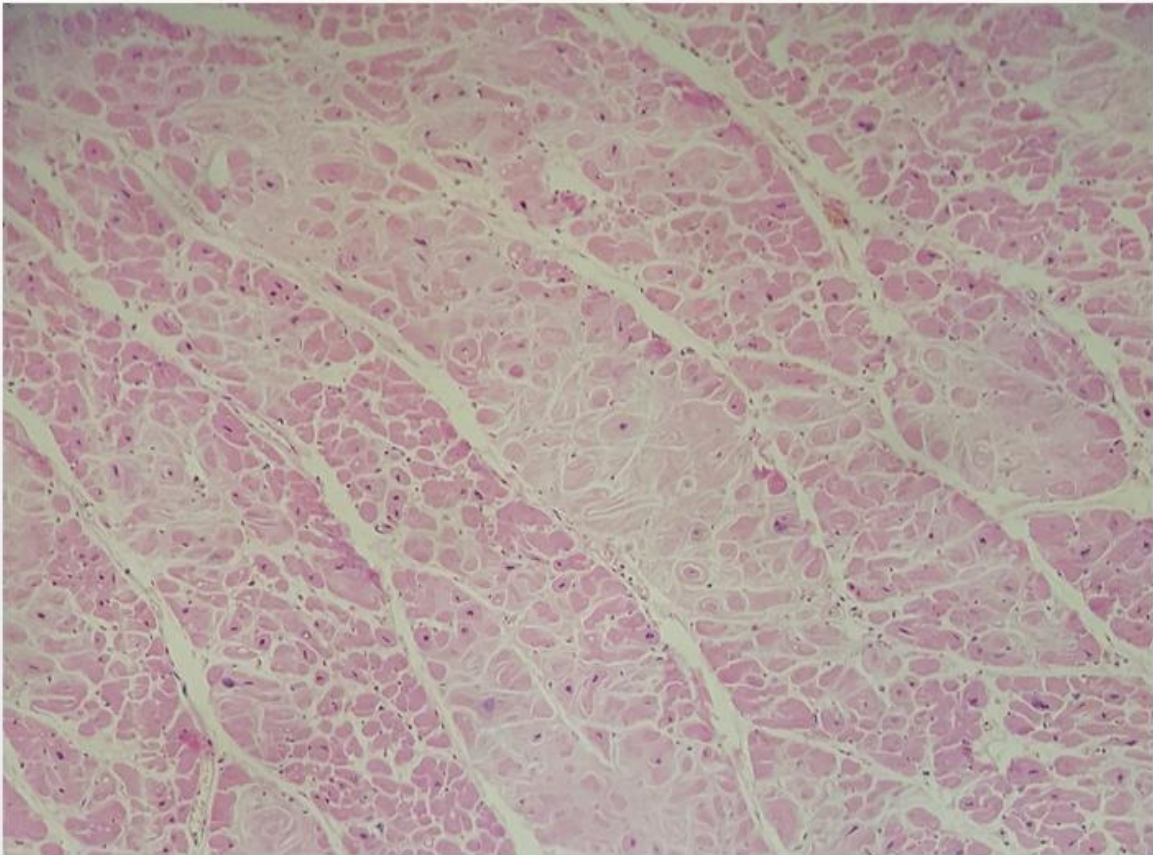
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with marked left ventricular hypertrophy and normal left ventricular ejection fraction. Complete blood count, basic metabolic panel, and serum and urine protein electrophoresis are within normal limits. Endomyocardial biopsy findings are shown below.



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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Store, Mail, Teams, Chrome, Firefox, and OneDrive.

System tray showing network, volume, and time (2:48 PM 2/9/2019).

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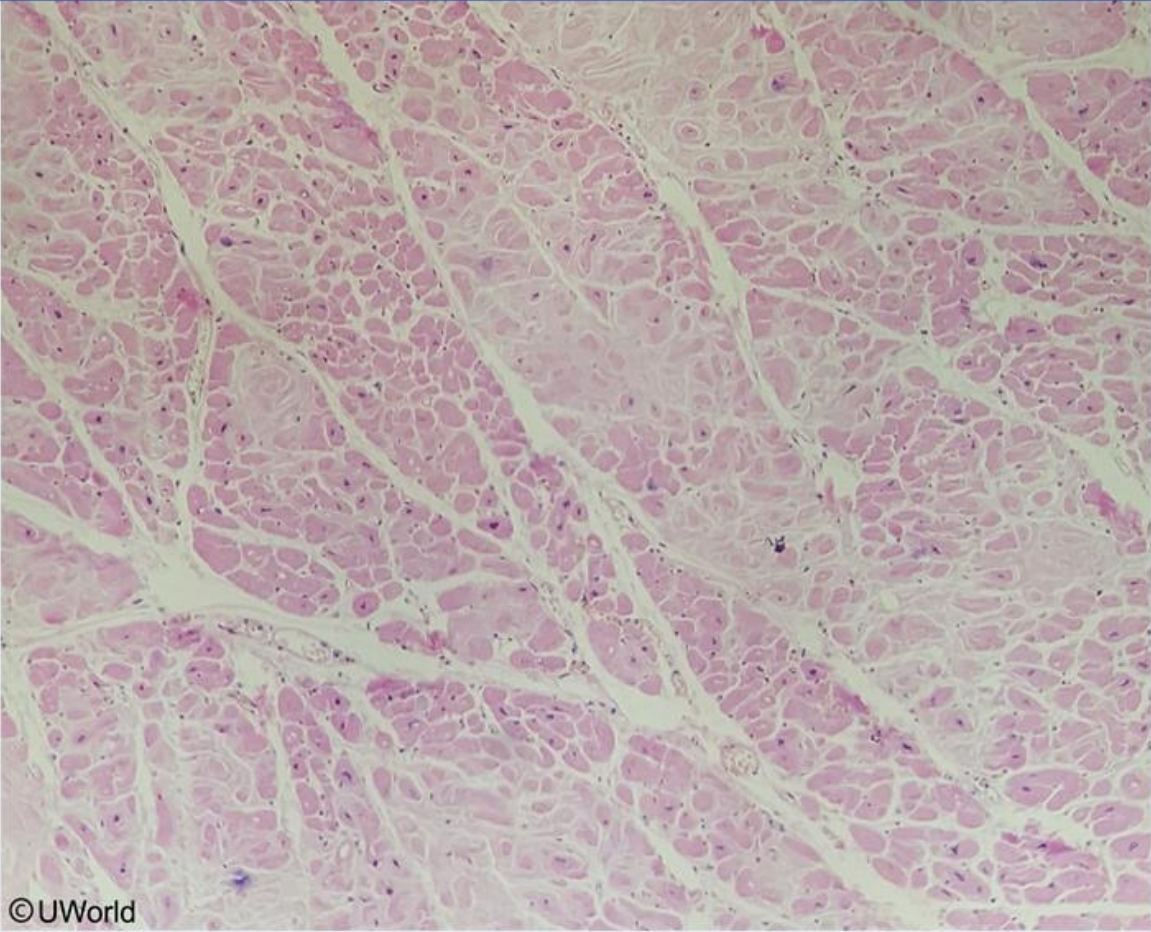
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Block Time Remaining: 00:03:05

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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Store, Mail, Teams, Chrome, Word, and OneDrive.

System tray showing network, volume, and battery status, along with the time 2:48 PM on 2/9/2019.

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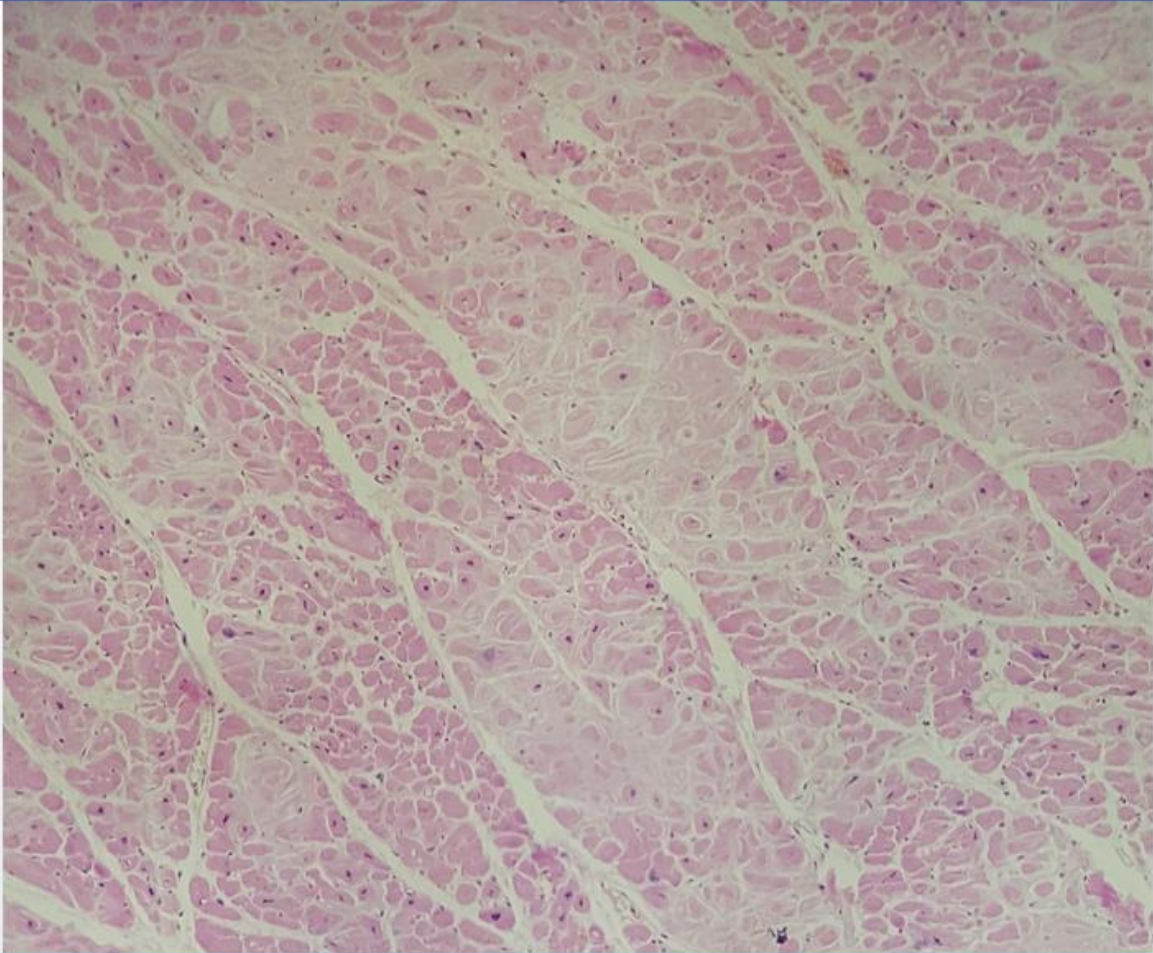
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Tutorial

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Text Zoom

An 82-year-old man comes to the office due to progressive dyspnea and fatigue over the last year, which now limit his daily activities. He has also noticed bilateral swelling of his feet. The patient has hypertension, which is controlled with amlodipine. His blood pressure is 122/72 mm Hg and pulse is 55/min. Physical examination reveals elevated jugular venous pressure with rapid 'y' descent and a prominent S4. Abdominal examination shows moderate ascites. The patient has 3+ bilateral lower extremity pitting edema. Echocardiogram reveals left atrial enlargement with marked left ventricular hypertrophy and normal left ventricular ejection fraction. Complete blood count, basic metabolic panel, and serum and urine protein electrophoresis are within normal limits. Endomyocardial biopsy findings are shown below.

Which of the following is the most likely diagnosis for this patient?

A. Cardiac myxoma

B. Cardiac sarcoidosis

C. Chagas disease

D. Hemochromatosis

E. Senile amyloidosis

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Which of the following is the most likely diagnosis for this patient?

☐ A. Cardiac myxoma [14%]

☐ B. Cardiac sarcoidosis [8%]

☐ C. Chagas disease [6%]

☐ D. Hemochromatosis [6%]

☒ E. Senile amyloidosis [63%]

Omitted

Correct answer
E

63%

Answered correctly

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Explanation

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Explanation

This patient's clinical presentation (eg, progressive exertional dyspnea, edema, ascites, elevated jugular venous pressure with rapid 'y' descent, prominent S4) and echocardiogram findings (eg, left atrial enlargement, left ventricular hypertrophy, normal ejection fraction) are consistent with **diastolic heart failure** due to restrictive cardiomyopathy. **Restrictive cardiomyopathy** can be idiopathic or caused by infiltrative diseases (eg, amyloidosis, sarcoidosis, hemochromatosis), radiation fibrosis, or endomyocardial fibrosis. Although most cases are associated with normal ventricular wall thickness, infiltrative conditions such as cardiac amyloidosis may lead to significant **ventricular hypertrophy**. Infiltrative diseases can also cause conduction system abnormalities (eg, bradycardia) that may ultimately require pacemaker implantation.

Cardiac amyloidosis results from abnormal extracellular deposition of insoluble proteins such as monoclonal light chains (AL amyloidosis), mutated transthyretin (familial ATTR amyloidosis), or wild-type transthyretin (**senile systemic amyloidosis**) in myocardial tissue. **Endomyocardial biopsy** typically reveals cross-sections of normal myocardial cells (blue arrow) with other areas of myocardium infiltrated by an **amorphous and acellular pink material** (black arrow). **Congo red stain** classically shows apple-green birefringence under polarized light microscopy.

(Choice A) Cardiac myxomas are the most common primary cardiac neoplasms, with approximately 80% originating in the left atrium. Left atrial myxomas can obstruct blood flow across the mitral valve, resulting in syncope and symptoms of heart failure. Endomyocardial biopsy typically reveals numerous bland stellate cells in a background of myxoid ground substance.

(Choice B) Cardiac sarcoidosis can present with restrictive or dilated cardiomyopathy along with conduction defects and atrial or ventricular arrhythmias; sudden death can also occur. Microscopic examination of the myocardium reveals the presence of noncaseating granulomas containing giant cells.

(Choice C) Chagas disease occurs secondary to *Trypanosoma cruzi* infection and is a common cause of myopericarditis and dilated cardiomyopathy in Latin America. Microscopic examination typically shows multiple parasitic protozoa within the myocardial fibers with a dense cellular infiltrate and myofiber destruction.

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microscopy.

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(Choice B) **Cardiac sarcoidosis** can present with restrictive or dilated cardiomyopathy along with conduction defects and atrial or ventricular arrhythmias; sudden death can also occur. Microscopic examination of the myocardium reveals the presence of noncaseating granulomas containing giant cells.

(Choice C) **Chagas disease** occurs secondary to *Trypanosoma cruzi* infection and is a common cause of myopericarditis and dilated cardiomyopathy in Latin America. Microscopic examination typically shows multiple parasitic protozoa within the myocardial fibers with a dense cellular infiltrate and myofiber destruction.

(Choice D) **Hemochromatosis** leads to excessive iron deposition in the myocardium and can cause conduction abnormalities and restrictive or dilated cardiomyopathy. Endomyocardial biopsy typically reveals excessive iron deposition in the myocardium, which can be confirmed with Prussian blue staining.

Educational objective:

Restrictive cardiomyopathy can be caused by infiltrative diseases (eg, amyloidosis, sarcoidosis, hemochromatosis) and often results in diastolic heart failure due to ventricular hypertrophy with impaired ventricular filling. Cardiac amyloidosis is characterized histologically by areas of myocardium infiltrated by an amorphous and acellular pink material (amyloid).

References

- Cardiac transthyretin amyloidosis.

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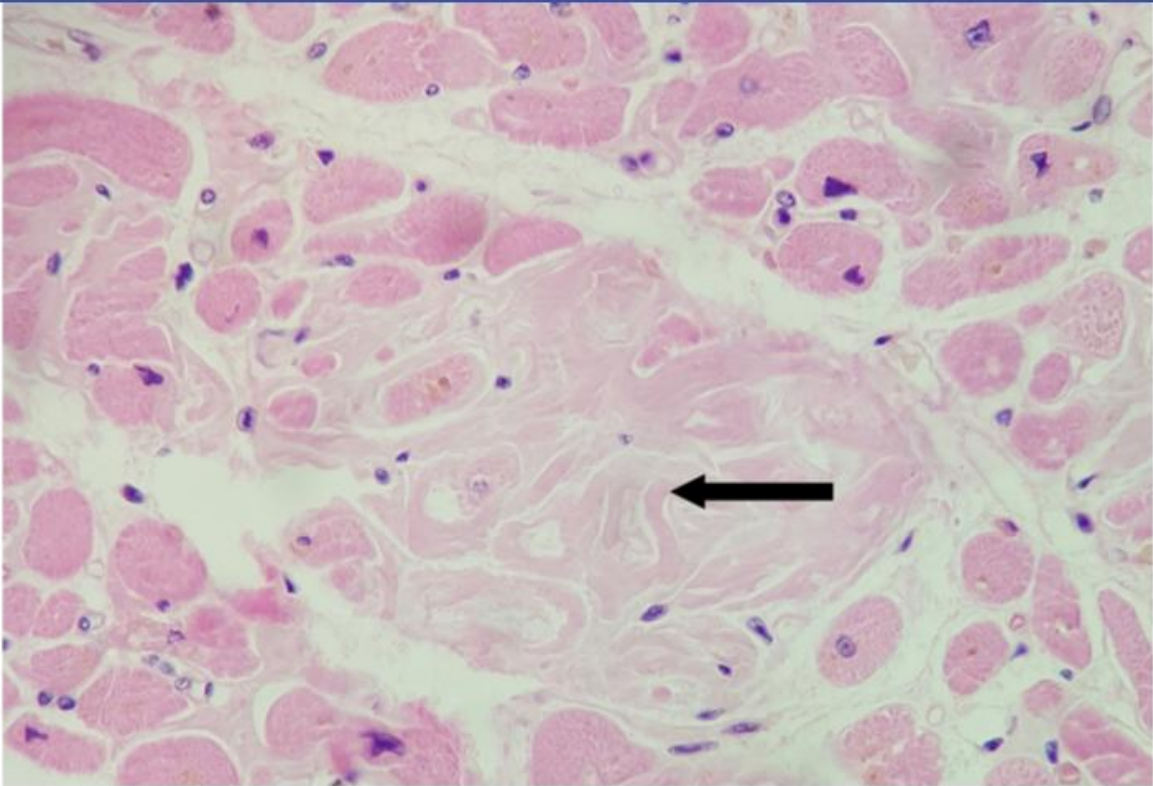
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A 62-year-old obese woman is brought to the hospital with acute-onset chest pain and shortness of breath. Medical history is significant for hyperlipidemia, diet-controlled type 2 diabetes mellitus, and peripheral artery disease. Two years ago, she had an ischemic stroke but had no residual neurologic deficit. The patient is a current smoker with a 30-pack-year smoking history. After initial evaluation, she is taken to the cardiac catheterization laboratory where she has a cardiac arrest due to ventricular arrhythmia and cannot be resuscitated. Autopsy shows thrombotic occlusion of the left anterior descending artery. In addition, it shows 95% atherosclerotic narrowing of the proximal right renal artery. Compared with the right kidney, this patient's left kidney is more likely to demonstrate which of the following findings?

A. Atrophic tubules

B. Crowded glomeruli

C. Interstitial fibrosis

D. Significant arteriolosclerosis

E. Smaller size

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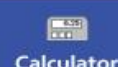
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A 62-year-old obese woman is brought to the hospital with acute-onset chest pain and shortness of breath. Medical history is significant for hyperlipidemia, diet-controlled type 2 diabetes mellitus, and peripheral artery disease. Two years ago, she had an ischemic stroke but had no residual neurologic deficit. The patient is a current smoker with a 30-pack-year smoking history. After initial evaluation, she is taken to the cardiac catheterization laboratory where she has a cardiac arrest due to ventricular arrhythmia and cannot be resuscitated. Autopsy shows thrombotic occlusion of the left anterior descending artery. In addition, it shows 95% atherosclerotic narrowing of the proximal right renal artery. Compared with the right kidney, this patient's left kidney is more likely to demonstrate which of the following findings?

- ☐ A. Atrophic tubules [4%]
- ☐ B. Crowded glomeruli [60%]
- ☐ C. Interstitial fibrosis [5%]
- ☒ D. Significant arteriosclerosis [16%]
- ☐ E. Smaller size [12%]

Omitted

Correct answer
D16%
Answered correctly5 Seconds
Time Spent11/12/2018
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Explanation

The most common cause of **renal artery stenosis** (RAS), especially in the elderly, is atherosclerosis. Rare alternate etiologies of RAS include

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Explanation

The most common cause of **renal artery stenosis** (RAS), especially in the elderly, is atherosclerosis. Rare alternate etiologies of RAS include fibromuscular dysplasia in younger women and Takayasu arteritis in individuals of Far Eastern descent. Regardless of the cause, RAS-induced hypoperfusion stimulates renin release by juxtaglomerular apparatus cells. Renin converts angiotensinogen to angiotensin I, which is then converted to angiotensin II by angiotensin-converting enzyme. As a potent vasoconstrictor, angiotensin II increases glomerular filtration pressure by promoting **systemic hypertension**.

RAS causes morphologic changes that markedly differ in the affected kidney and the contralateral kidney. The narrowing of the renal artery to the affected kidney protects the organ parenchyma from high systemic pressure. Morphologic changes in the affected kidney are instead related to hypoperfusion and include diffuse cortical thinning and atrophy, diffuse tubular atrophy, glomerular crowding, and juxtaglomerular apparatus enlargement.

In contrast, the contralateral **nonstenotic kidney** is exposed to high blood pressure and therefore demonstrates typical signs of hypertensive nephrosclerosis, including arterial intimal fibroplasia and hyalinization of the arterioles (**hyaline arteriolosclerosis**). In cases of severe hypertension, hyperplastic arteriosclerosis (onion-skinning) and fibrinoid necrosis may occur.

(Choices A, B, and E) A kidney affected by stenosis typically has atrophic tubules and is small in size. The glomeruli also appear crowded due to diffuse tubular atrophy.

(Choice C) Renal interstitial fibrosis is characteristic of the ischemic kidney and various progressive renal diseases. This finding is seen in advanced RAS in the involved (not contralateral) kidney.

Educational objective:

In unilateral renal artery stenosis, the affected kidney is protected from high blood pressure by the narrowing of its renal artery and may suffer ischemic damage. In contrast, the contralateral well-perfused kidney typically shows changes of hypertensive nephropathy (eg, hyaline or

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The most common cause of **renal artery stenosis** (RAS), especially in the elderly, is atherosclerosis. Rare alternate etiologies of RAS include fibromuscular dysplasia in younger women and Takayasu arteritis in individuals of Far Eastern descent. Regardless of the cause, RAS-induced hypoperfusion stimulates renin release by juxtaglomerular apparatus cells. Renin converts angiotensinogen to angiotensin I, which is then converted to angiotensin II by angiotensin-converting enzyme. As a potent vasoconstrictor, angiotensin II increases glomerular filtration pressure by promoting **systemic hypertension**.

RAS causes morphologic changes that markedly differ in the affected kidney and the contralateral kidney. The narrowing of the renal artery to the affected kidney protects the organ parenchyma from high systemic pressure. Morphologic changes in the affected kidney are instead related to hypoperfusion and include diffuse cortical thinning and atrophy, diffuse tubular atrophy, glomerular crowding, and juxtaglomerular apparatus enlargement.

In contrast, the contralateral **nonstenotic kidney** is exposed to high blood pressure and therefore demonstrates typical signs of hypertensive nephrosclerosis, including arterial intimal fibroplasia and hyalinization of the arterioles (**hyaline arteriolosclerosis**). In cases of severe hypertension, hyperplastic arteriosclerosis (onion-skinning) and fibrinoid necrosis may occur.

(Choices A, B, and E) A kidney affected by stenosis typically has atrophic tubules and is small in size. The glomeruli also appear crowded due to diffuse tubular atrophy.

(Choice C) Renal interstitial fibrosis is characteristic of the ischemic kidney and various progressive renal diseases. This finding is seen in advanced RAS in the involved (not contralateral) kidney.

Educational objective:

In unilateral renal artery stenosis, the affected kidney is protected from high blood pressure by the narrowing of its renal artery and may suffer ischemic damage. In contrast, the contralateral well-perfused kidney typically shows changes of hypertensive nephropathy (eg, hyaline or hyperplastic arteriolosclerosis).

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Text Zoom

A 34-year-old woman is evaluated in the emergency department due to 1-day of chest and left shoulder pain. The patient describes the pain as constant and worse in certain positions. A month ago, she was treated for a skin rash and joint pain. Her medical history is otherwise unremarkable. She is a lifetime nonsmoker and does not use alcohol or illicit drugs. Her father has hypertension and had a stroke at age 64. Temperature is 37.5 C (99.5 F), blood pressure is 122/70 mm Hg, and pulse is 97/min and regular. A triphasic, scratchy sound is heard over the left sternal border with the patient sitting up. The lungs are clear on auscultation. ECG shows sinus rhythm with ST-segment elevation in all leads except avR and V1. Which of the following is the most likely diagnosis?

A. Eosinophilic myocarditis

B. Fibrinous pericarditis

C. Hemorrhagic pericarditis

D. Nonbacterial endocarditis

E. Purulent pericarditis

Submit

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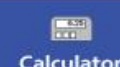
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2/7/2019



A 34-year-old woman is evaluated in the emergency department due to 1-day of chest and left shoulder pain. The patient describes the pain as constant and worse in certain positions. A month ago, she was treated for a skin rash and joint pain. Her medical history is otherwise unremarkable. She is a lifetime nonsmoker and does not use alcohol or illicit drugs. Her father has hypertension and had a stroke at age 64. Temperature is 37.5 C (99.5 F), blood pressure is 122/70 mm Hg, and pulse is 97/min and regular. A triphasic, scratchy sound is heard over the left sternal border with the patient sitting up. The lungs are clear on auscultation. ECG shows sinus rhythm with ST-segment elevation in all leads except avR and V1. Which of the following is the most likely diagnosis?

- ☐ A. Eosinophilic myocarditis [7%]
- ☒ B. Fibrinous pericarditis [67%]
- ☐ C. Hemorrhagic pericarditis [2%]
- ☐ D. Nonbacterial endocarditis [11%]
- ☐ E. Purulent pericarditis [11%]

Omitted

Correct answer
B67%
Answered correctly4 Seconds
Time Spent11/27/2018
Last Updated

Explanation

Acute pericarditis

Block Time Remaining: 00:00:38

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Explanation

Acute pericarditis	
Etiology	- Viral or idiopathic - Autoimmune disease (eg, SLE) - Uremia (acute or chronic renal failure) - Post myocardial infarction - Early: peri-infarction pericarditis - Late: Dressler syndrome
Clinical features & diagnosis	- Pleuritic chest pain (↓ when sitting up) ± fever - Pericardial friction rub - ECG: diffuse ST elevation - Pericardial effusion on echocardiogram

SLE = systemic lupus erythematosus.

This patient with pleuritic-type chest pain radiating to the shoulder and a recent history of skin rash and joint pain likely has acute **fibrinous pericarditis** due to **systemic lupus erythematosus**. Fibrinous pericarditis is the most common type of pericarditis and consists of pericardial inflammation with serous fluid and **fibrin-containing exudate** in the pericardial space.

Patients typically have pleuritic (sharp, worse with breathing or movement) anterior chest pain that can radiate to the left shoulder or posteriorly to the bilateral scapulae. Fibrin deposition causes roughening of the visceral and parietal pericardium, often heard as a **triphasic friction rub** (occurring during atrial systole, ventricular systole, and early ventricular diastole) on cardiac auscultation; however, the rub can be absent if significant pericardial effusion is present. **ECG** typically shows **diffuse ST elevation** due to inflammation of the ventricular myocardium.

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Patients typically have pleuritic (sharp, worse with breathing or movement) anterior chest pain that can radiate to the left shoulder or posteriorly to the bilateral scapulae. Fibrin deposition causes roughening of the visceral and parietal pericardium, often heard as a **triphasic friction rub** (occurring during atrial systole, ventricular systole, and early ventricular diastole) on cardiac auscultation; however, the rub can be absent if significant pericardial effusion is present. **ECG** typically shows **diffuse ST elevation** due to inflammation of the ventricular myocardium.

Other causes of fibrinous pericarditis include viral infection, myocardial infarction, uremia, and other rheumatologic disease (eg, rheumatoid arthritis). If acute fibrinous pericarditis goes without treatment, chronic constrictive pericarditis can develop in some patients.

(Choice A) Eosinophilic myocarditis can occur with malignancy, parasitic infection, or drug hypersensitivity and often presents with symptoms of heart failure. A friction rub on cardiac auscultation is not expected.

(Choices C and E) Hemorrhagic and purulent pericarditis are both less common than fibrinous pericarditis. Hemorrhagic pericarditis consists of blood mixed with fibrinous exudate; it most commonly occurs in the setting of malignancy but may also occur due to tuberculosis, following cardiac surgery, or in patients with underlying coagulopathy. Purulent pericarditis results from active bacterial infection (eg, *Staphylococcus aureus*, *Streptococcus* species) in the pericardial space, which may occur due to hematogenous spread or direct extension of pneumonia or endocarditis.

(Choice D) Nonbacterial endocarditis can occur in systemic lupus erythematosus and involves deposition of sterile, platelet-rich thrombi on the mitral or aortic valve. It would not explain a friction rub or diffuse ST elevation on ECG.

Educational objective:

Fibrinous pericarditis is the most common type of pericarditis and is characterized by pericardial inflammation with a serous, fibrin-containing exudate in the pericardial space. Pleuritic chest pain and a triphasic friction rub are frequently seen. Common causes include viral infection, myocardial infarction, uremia, and rheumatologic disease (eg, systemic lupus erythematosus, rheumatoid arthritis).

References

- Acute pericarditis.

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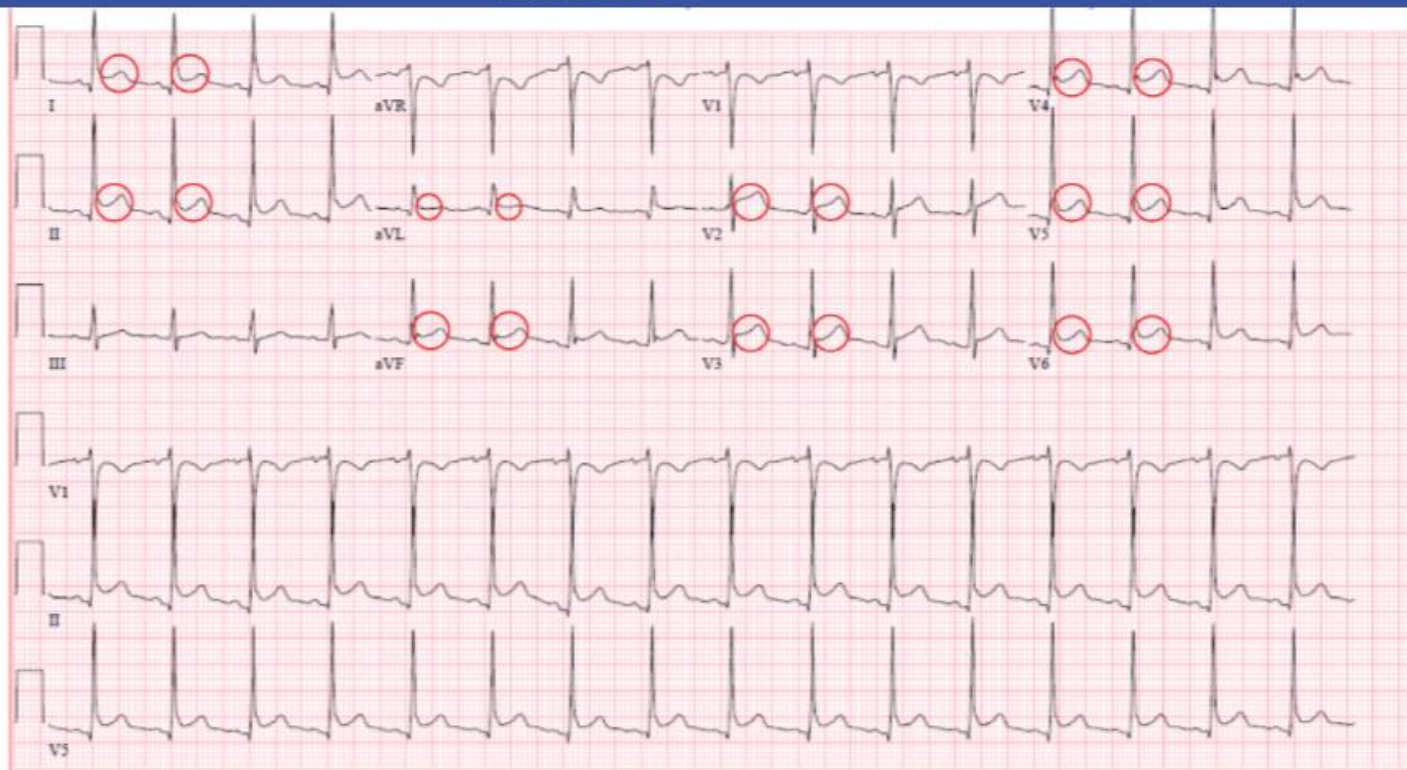


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Exhibit Display



Zoom In

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Add To Flash Card

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Question Id: 232

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Text Zoom

A 42-year-old woman is brought to the hospital due to right-sided weakness and difficulty speaking. She has a longstanding history of a diastolic murmur, but her medical follow-up has been poor. She does not use tobacco, alcohol, or illicit drugs. A CT scan of the brain reveals a large ischemic stroke involving the left middle cerebral artery distribution. The patient dies 2 days later due to progressive neurologic deterioration. At autopsy, exploration of the left atrium shows diffuse fibrous thickening and distortion of the mitral valve leaflets, commissural fusion at the leaflet edges, and narrowing of the mitral valve orifice. This finding is most likely the result of which of the following conditions?

☐ A. Congenital heart disease

☐ B. Degenerative valvular calcinosis

☐ C. Infective endocarditis

☐ D. Late syphilis

☐ E. Rheumatic fever

☐ F. Rheumatoid arthritis

☐ G. Rupture of the papillary muscle

Submit

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TUTOR

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Question Id: 232

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Text Zoom

A 42-year-old woman is brought to the hospital due to right-sided weakness and difficulty speaking. She has a longstanding history of a diastolic murmur, but her medical follow-up has been poor. She does not use tobacco, alcohol, or illicit drugs. A CT scan of the brain reveals a large ischemic stroke involving the left middle cerebral artery distribution. The patient dies 2 days later due to progressive neurologic deterioration. At autopsy, exploration of the left atrium shows diffuse fibrous thickening and distortion of the mitral valve leaflets, commissural fusion at the leaflet edges, and narrowing of the mitral valve orifice. This finding is most likely the result of which of the following conditions?

☐

A. Congenital heart disease [3%]

☐

B. Degenerative valvular calcinosis [7%]

☐

C. Infective endocarditis [12%]

☐

D. Late syphilis [1%]

☒

E. Rheumatic fever [72%]

☐

F. Rheumatoid arthritis [0%]

☐

G. Rupture of the papillary muscle [1%]

Omitted

Correct answer
E

72%

Answered correctly

3 Seconds

Time Spent

11/14/2018

Last Updated

Explanation

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Explanation

This patient's presentation suggests **mitral stenosis** (MS) due to rheumatic heart disease. Fibrous thickening and fusion of the valve leaflets in chronic rheumatic heart disease following acute **rheumatic fever** is the most common cause of MS, accounting for up to 99% of cases. There is often a latency period of 10-20 years between the initial episode of rheumatic fever and symptomatic MS, with most patients manifesting during the fourth or fifth decade of life.

Mitral stenosis can cause atrial enlargement, which may lead to **atrial fibrillation** and/or atrial **mural thromboses**. Thrombi dislodged from the wall of the left atrium may later cause an embolic stroke, which likely occurred in this patient. Cardiac auscultation in **mitral stenosis** often reveals a loud first heart sound (S1), an early diastolic sound (opening snap), followed by a **mid-diastolic murmur** from turbulent flow across the mitral valve.

(Choice A) Congenital endocardial cushion defects can result in mitral regurgitation. However, congenital mitral valve stenosis is rare and untreated newborns and infants typically have a grim prognosis.

(Choice B) Degenerative calcific deposits most commonly develop in the mitral valve annulus in women over age 60 and generally do not impair valve function. This patient's autopsy did not show any peripherally located nodular calcific deposits.

(Choice C) Embolization of vegetations due to infective endocarditis can cause a stroke or septic infarct resulting in a brain abscess. However, infective endocarditis is usually associated with large friable vegetations on the valve cusps along with destruction (rather than fibrosis) of the valve leaflets.

(Choice D) Tertiary syphilis may result in aortitis, aortic aneurysm, and/or aortic regurgitation. Mitral valve lesions are not commonly observed.

(Choice F) Although cardiac involvement is uncommon in rheumatoid arthritis, patients can develop pericarditis and myocarditis. Rheumatoid nodules can also develop in the cardiac tissues (including valvular structures), but these are rarely symptomatic.

(Choice G) Rupture of papillary muscle (whether due to bacterial endocarditis, a connective tissue disease, or myocardial infarction) leads to prolapse of the valve leaflets and mitral regurgitation (holosystolic or early systolic murmur).

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Question Id: 442

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Text Zoom

A 60-year-old man comes to the office with a 6-month history of exertional chest pain that remits with rest. His other medical conditions include hypertension, diabetes mellitus, and hypercholesterolemia. An exercise stress test is positive for inducible ischemia. Cardiac catheterization shows 80% occlusion of the right coronary artery and 60% occlusion of the left coronary artery. The first step in the pathogenesis of this patient's coronary artery disease most likely involved which of the following cell types?

A. Endothelial cells

B. Interstitial fibroblasts

C. Macrophages

D. Mast cells

E. Pericytes

F. Platelets

G. Smooth muscle cells

Submit

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TUTOR

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Feedback

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Windows Taskbar

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A 60-year-old man comes to the office with a 6-month history of exertional chest pain that remits with rest. His other medical conditions include hypertension, diabetes mellitus, and hypercholesterolemia. An exercise stress test is positive for inducible ischemia. Cardiac catheterization shows 80% occlusion of the right coronary artery and 60% occlusion of the left coronary artery. The first step in the pathogenesis of this patient's coronary artery disease most likely involved which of the following cell types?

- ☒ A. Endothelial cells [76%]
- ☐ B. Interstitial fibroblasts [0%]
- ☐ C. Macrophages [16%]
- ☐ D. Mast cells [0%]
- ☐ E. Pericytes [0%]
- ☐ F. Platelets [2%]
- ☐ G. Smooth muscle cells [2%]

Omitted

Correct answer
A



76%
Answered correctly



3 Seconds
Time Spent



11/05/2018
Last Updated

Explanation

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Endothelium

Intima

Media

Adventitia

Monocyte

Platelets

Fatty streak

Response to endothelial injury

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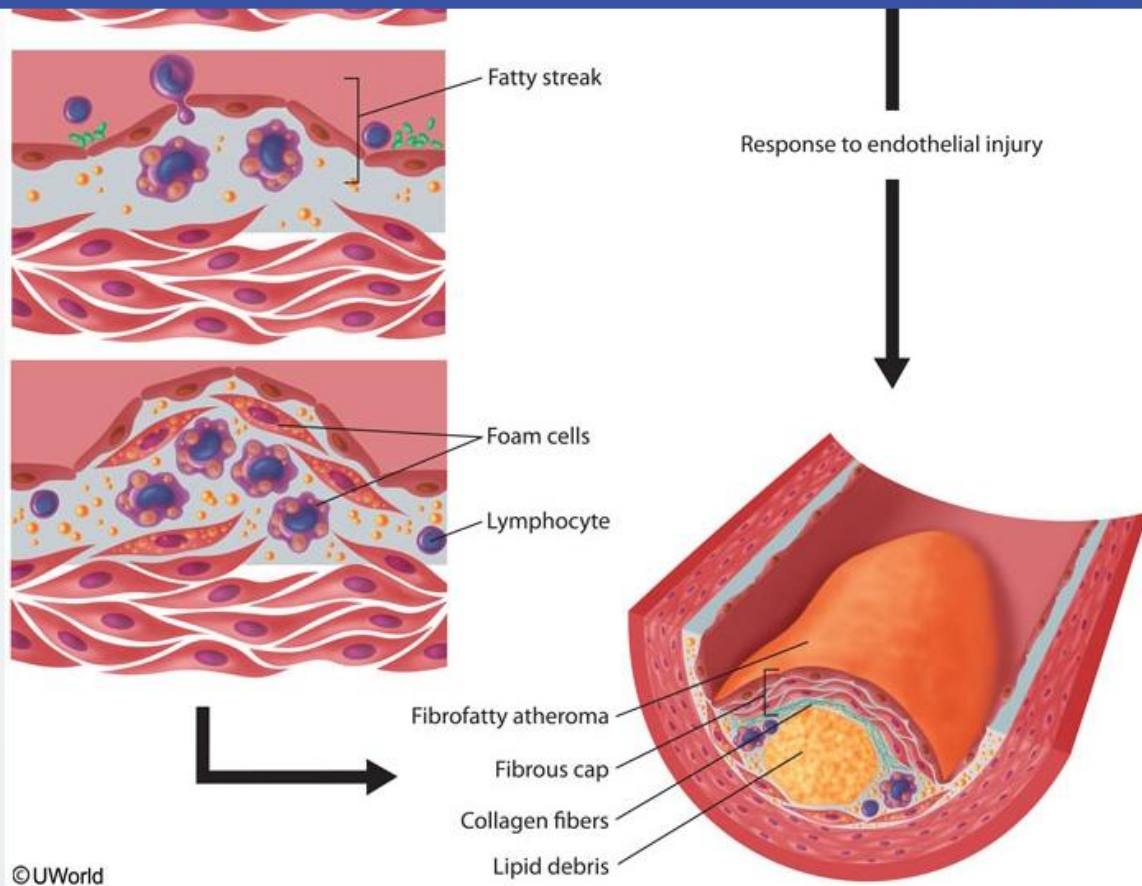
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This patient's presentation is consistent with coronary artery **atherosclerosis**. The pathogenesis of atherosclerosis likely begins with **endothelial cell injury**. Chronic endothelial cell injury may result from hypertension (and related hemodynamic factors), hyperlipidemia, smoking, and



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Lipid debris

This patient's presentation is consistent with coronary artery **atherosclerosis**. The pathogenesis of atherosclerosis likely begins with **endothelial cell injury**. Chronic endothelial cell injury may result from hypertension (and related hemodynamic factors), hyperlipidemia, smoking, and diabetes mellitus. Such injury leads to endothelial cell dysfunction and/or exposure of subendothelial collagen (endothelial cell denudation).

Endothelial cell dysfunction results in monocyte and lymphocyte adhesion and migration into the intima (**Choice C**), while exposure of subendothelial collagen promotes platelet adhesion (**Choice F**). Growth factors produced by monocytes and platelets stimulate medial smooth muscle cell (SMC) migration into the intima (**Choice G**). At the same time, increased vascular permeability allows LDL cholesterol into the intima, where it is phagocytosed by the accumulating macrophages and SMCs to produce lipid-laden foam cells (**fatty streak**).

The continued release of cytokines and growth factors results in a chronic inflammatory state within the underlying intima. This promotes further deposition of LDL cholesterol within the intima and stimulates SMC proliferation with increased production of collagen and proteoglycans. Necrosis of foam cells results in release of toxic oxidized LDL into the extracellular matrix, perpetuating a cycle of injury. The lesion eventually organizes into a core of lipid debris surrounded by monocytes and lymphocytes covered by a fibrous cap with intermixed SMCs (**fibrofatty atheroma**).

(**Choice B**) Fibroblasts do not contribute significantly to atheroma formation. The fibrous tissue, including the fibrous cap, of atheromas is synthesized by SMCs that have migrated to the intimal layer in which the plaque forms.

(**Choice D**) Mast cells are not significantly involved in the pathogenesis of atherosclerosis.

(**Choice E**) Pericytes are pluripotent cells that surround the smallest blood vessels (especially postcapillary venules). Because atherosclerosis affects the large elastic arteries and large- and medium-sized muscular arteries, these cells do not play a significant role.

Educational objective:

Atherosclerosis is initiated by repetitive endothelial cell injury, which leads to a chronic inflammatory state in the underlying intima of large elastic arteries as well as in large- and medium-sized muscular arteries.

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Text Zoom

A 56-year-old man comes to the emergency department with acute, severe chest pain. His electrocardiogram is within normal limits. Contrast-enhanced computed tomography of his chest is shown below.

Which of the following is the single most important risk factor for this patient's current condition?

☐ A. Diabetes mellitus

☐ B. High cholesterol

☐ C. Hypertension

☐ D. Limited physical activity

☐ E. Smoking

☐ F. Spirochete infection

Submit

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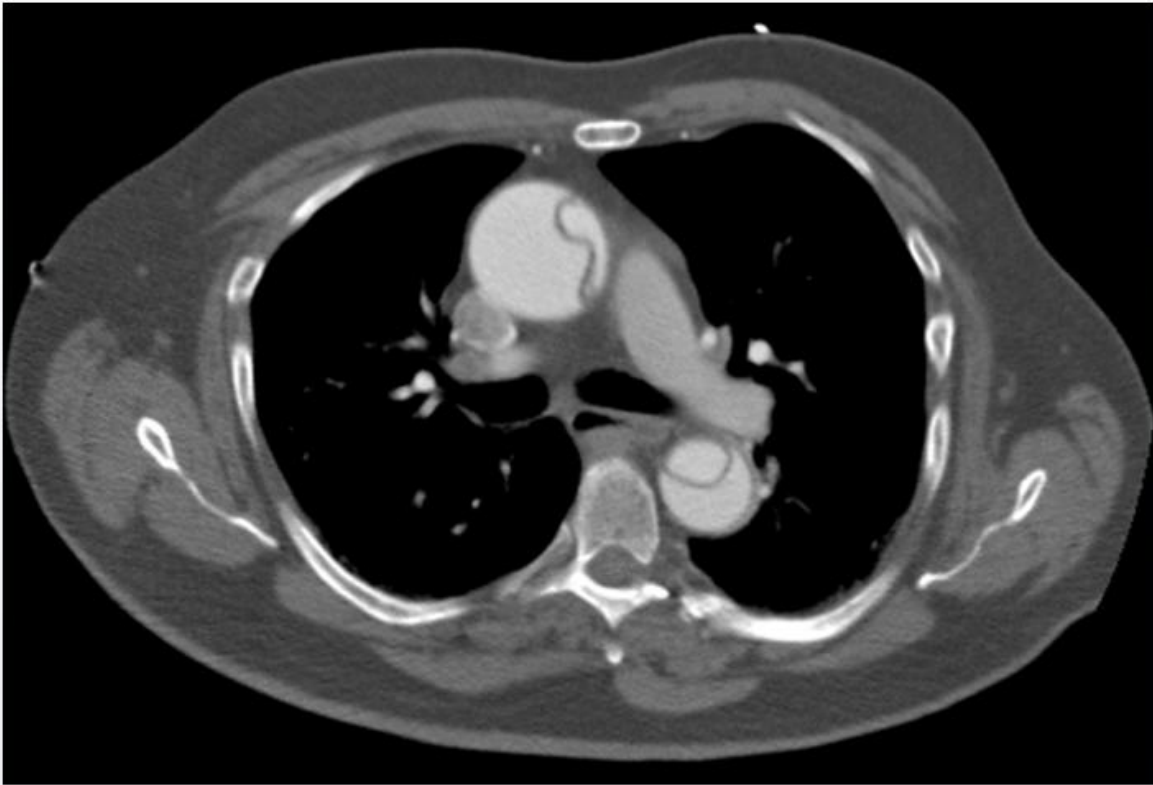
Notes

Calculator

Reverse Color

Text Zoom

A 56-year-old man comes to the emergency department with acute, severe chest pain. His electrocardiogram is within normal limits. Contrast-enhanced computed tomography of his chest is shown below.



Which of the following is the single most important risk factor for this patient's current condition?

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Text Zoom

A 56-year-old man comes to the emergency department with acute, severe chest pain. His electrocardiogram is within normal limits. Contrast-enhanced computed tomography of his chest is shown below.

Which of the following is the single most important risk factor for this patient's current condition?

A. Diabetes mellitus

B. High cholesterol

C. Hypertension

D. Limited physical activity

E. Smoking

F. Spirochete infection

Submit

Block Time Remaining: 00:00:45

TUTOR

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Feedback

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2/7/2019

A 56-year-old man comes to the emergency department with acute, severe chest pain. His electrocardiogram is within normal limits. Contrast-enhanced computed tomography of his chest is shown below.

Which of the following is the single most important risk factor for this patient's current condition?

- ☐ A. Diabetes mellitus [1%]
- ☐ B. High cholesterol [6%]
- ☒ C. Hypertension [64%]
- ☐ D. Limited physical activity [2%]
- ☐ E. Smoking [15%]
- ☐ F. Spirochete infection [9%]

Omitted

Correct answer
C



64%
Answered correctly



2 Seconds
Time Spent



10/10/2018
Last Updated

Explanation

This patient's chest CT scan reveals a widened, ascending thoracic aorta with a nonenhancing septum dividing the lumen of the ascending and descending aorta. The "septum" is actually the tunica intima of the aorta, which has been torn from the remainder of the aortic wall. A tear in the tunica intima is thought to be the primary event in the process leading to aortic dissection. A space and space island is formed through the tear.

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This patient's chest CT scan reveals a widened, ascending thoracic aorta with a nonenhancing septum dividing the lumen of the ascending and descending aorta. The "septum" is actually the tunica intima of the aorta, which has been torn from the remainder of the aortic wall. A tear in the tunica intima is thought to be the primary event in the process leading to aortic dissection. As more and more blood is forced through the tear, an intramural hematoma begins to develop between the intimal flap and medial wall, creating a false lumen. The dissection can extend both proximally toward the heart and distally, sometimes extending all the way to the iliac and femoral arteries. **Hypertension** is the single most important risk factor for the development of intimal tears leading to **aortic dissection**. Cystic medial degeneration, which may be seen in connective tissue diseases such as Marfan syndrome, also predisposes patients (especially younger ones) to aortic dissection.

(Choice A) Diabetes mellitus is an important risk factor for the development of atherosclerosis and hypertension. It can increase the relative risk of aortic dissection by promoting hypertension, which is the single most important risk factor for aortic dissection.

(Choice B) High cholesterol is a risk factor for atherosclerosis. Atherosclerosis predisposes more to **aortic aneurysm** formation (especially abdominal aortic aneurysms) than to aortic dissection.

(Choice D) A sedentary lifestyle is a risk factor for cardiovascular disease (eg, stroke, myocardial infarction) but does not appear to be directly linked to aortic dissection.

(Choice E) Smoking contributes significantly to the risk of atherosclerosis and thromboangiitis obliterans (Buerger disease). Thromboangiitis obliterans is a vasculitis of medium and small arteries, principally the tibial and radial arteries.

(Choice F) Tertiary syphilis can result in spirochetal endarteritis that affects the vasa vasorum within the adventitia of the ascending thoracic aorta. This weakens the aortic wall and predisposes infected patients to ascending aortic aneurysms. Aortic dissection is only rarely caused by syphilitic aneurysms.

Educational objective:

Hypertension is the single most important risk factor for the development of intimal tears leading to aortic dissection. Hypertension, smoking, diabetes mellitus, and hypercholesterolemia are all major risk factors for atherosclerosis, which predisposes more to aortic aneurysm formation than aortic dissection.

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Text Zoom

A group of forensic pathologists are analyzing tissue samples of adolescents age 13-18 to study the aging process. Autopsy of a 14-year-old boy who died in a motor vehicle accident shows several minimally raised yellow spots on the inner surface of the abdominal aorta. The rest of the cardiovascular findings during the autopsy are unremarkable. He had no known medical problems. There was no family history of cardiovascular disease or sudden cardiac death. Which of the following is most likely to be the predominant cell type in these lesions on light microscopy?

A. Eosinophils

B. Fibroblasts

C. Macrophages

D. Mast cells

E. Neutrophils

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Text Zoom

A group of forensic pathologists are analyzing tissue samples of adolescents age 13-18 to study the aging process. Autopsy of a 14-year-old boy who died in a motor vehicle accident shows several minimally raised yellow spots on the inner surface of the abdominal aorta. The rest of the cardiovascular findings during the autopsy are unremarkable. He had no known medical problems. There was no family history of cardiovascular disease or sudden cardiac death. Which of the following is most likely to be the predominant cell type in these lesions on light microscopy?

A. Eosinophils [0%]

B. Fibroblasts [13%]

C. Macrophages [80%]

D. Mast cells [0%]

E. Neutrophils [5%]

Omitted

Correct answer
C

80%

Answered correctly

2 Seconds

Time Spent

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Last Updated

Explanation



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Endothelium

Intima

Media

Adventitia

Monocyte

Platelets

Fatty streak

Response to endothelial injury

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Platelets

Fatty streak

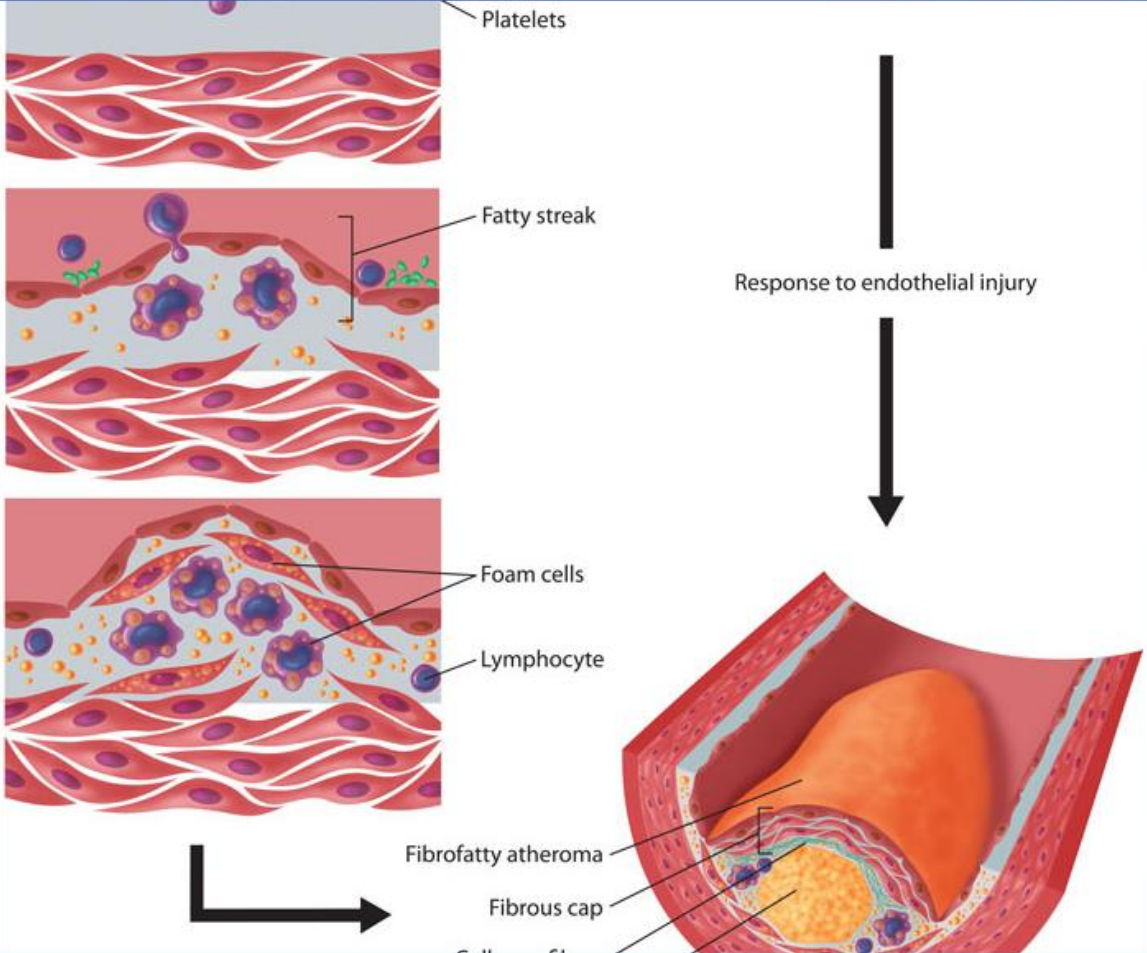
Foam cells

Lymphocyte

Response to endothelial injury

Fibrofatty atheroma

Fibrous cap



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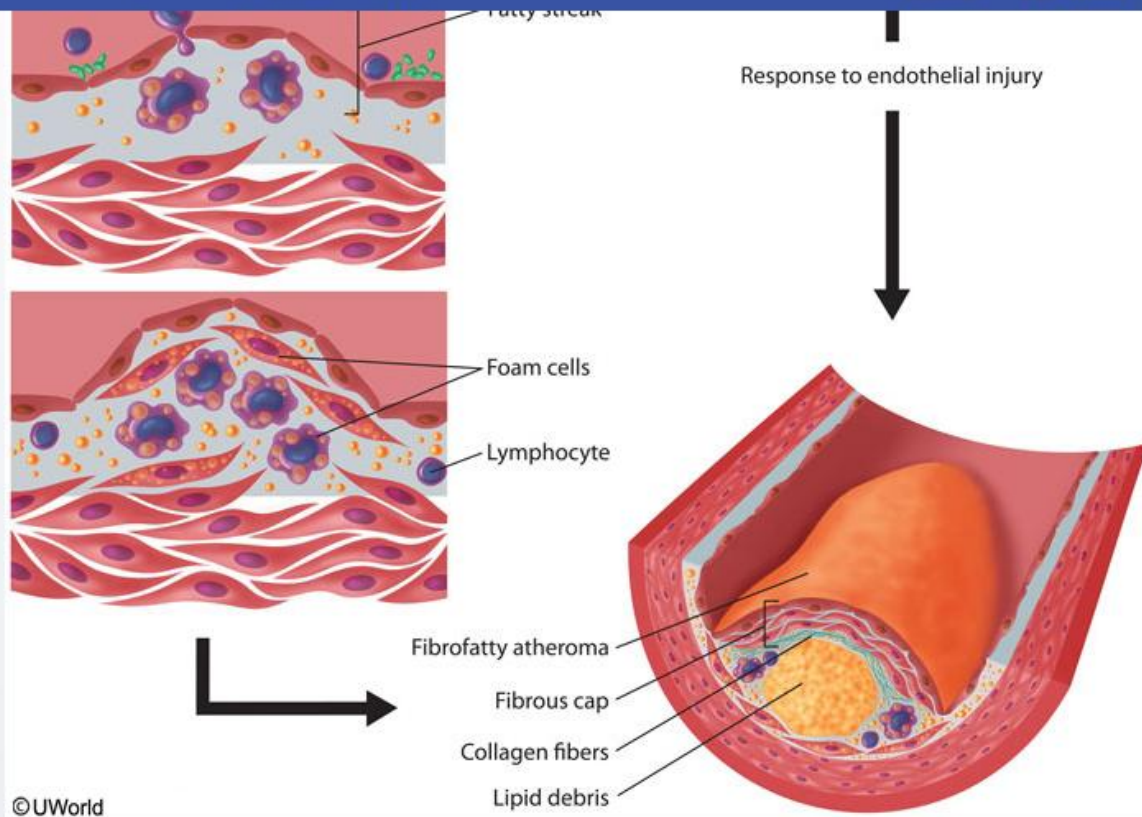
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The lesions described during autopsy on the inner surface of the abdominal aorta are consistent with **fatty streaks**. These are the earliest lesions in the progression to atherosclerosis and can be seen as early as the second decade of life. Atherosclerosis is a pathophysiologic process that begins in childhood with the development of focal intimal thickening and lipid accumulation. It progresses in the following sequence:

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Collagen fibers

Lipid debris

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The lesions described during autopsy on the inner surface of the abdominal aorta are consistent with **fatty streaks**. These are the earliest lesions in the progression to atherosclerosis and can be seen as early as the second decade of life. Atherosclerosis is a pathophysiologic process that begins in childhood with the development of focal intimal thickening and lipid accumulation. It progresses in the following sequence:

- **Endothelial injury/dysfunction** causing increased vascular permeability and enhanced leukocyte adhesion
- Accumulation of lipoproteins (LDL) within the vessel wall
- **Monocyte adhesion** to the endothelium, followed by migration and transformation into lipid-laden **macrophages (foam cells)**
- Release of growth factors by adherent platelets, activated macrophages, and endothelial cells
- Recruitment of T cells and **smooth muscle cell proliferation**, which lead to extracellular matrix production

With advancing age, the chronic inflammatory process initiated by endothelial injury transitions from fatty streaks within the intima (composed mainly of lipid-laden foam cells) into atherosclerotic plaques, such as fibrous cap atheromas and fibrous plaques.

(Choices A, B, D, and E) Eosinophils, fibroblasts, mast cells, and neutrophils are not the predominant cell types found in fatty streaks.

Educational objective:

Fatty streaks are the earliest lesions of atherosclerosis and can be seen as early as the second decade of life. They appear as a collection of lipid-laden macrophages (foam cells) in the intima that can eventually progress to atherosclerotic plaques.

References

- [Pathogenesis of atherosclerosis.](#)

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A 34-year-old woman comes to the emergency department because of sharp chest pain that radiates to the left shoulder. The pain increases with inspiration and is partially relieved by sitting up and leaning forward. Review of her outpatient medical records shows that she was seen for a facial rash 6 months ago. She is also being evaluated for proteinuria that was identified during her last clinic appointment. Which of the following is the most likely cause of this patient's chest pain?

☐ A. Aortic dissection

☐ B. Cardiac tamponade

☐ C. Intimal hyperplasia of pulmonary arteries

☐ D. Non-infectious cardiac valve vegetations

☐ E. Pericardial inflammation

Submit

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A 34-year-old woman comes to the emergency department because of sharp chest pain that radiates to the left shoulder. The pain increases with inspiration and is partially relieved by sitting up and leaning forward. Review of her outpatient medical records shows that she was seen for a facial rash 6 months ago. She is also being evaluated for proteinuria that was identified during her last clinic appointment. Which of the following is the most likely cause of this patient's chest pain?

☐ A. Aortic dissection [1%]

☐ B. Cardiac tamponade [3%]

☐ C. Intimal hyperplasia of pulmonary arteries [1%]

☐ D. Non-infectious cardiac valve vegetations [9%]

☒ E. Pericardial inflammation [83%]

Omitted

Correct answer
E

83%

Answered correctly

3 Seconds

Time Spent

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Explanation

Manifestations of systemic lupus erythematosus

- **Constitutional:** fever, fatigue & weight loss
- Symmetric, migratory arthritis

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Explanation

Manifestations of systemic lupus erythematosus

Clinical symptoms

- **Constitutional:** fever, fatigue & weight loss
- Symmetric, migratory **arthritis**
- Skin: **butterfly rash** & **photosensitivity**
- **Serositis:** pleurisy, pericarditis & peritonitis
- Thromboembolic events (due to vasculitis & antiphospholipid antibodies)
- Neurologic: cognitive dysfunction & seizures

Laboratory findings

- Hemolytic anemia, thrombocytopenia & leukopenia
- **Hypocomplementemia** (C3 & C4)
- Antibodies:
 - **ANA** (sensitive)
 - **Anti-dsDNA** & **anti-Smith** (specific)
- Renal involvement: **proteinuria** & elevated creatinine

ANA = antinuclear antibodies; dsDNA = double-stranded DNA.

This patient's chest pain is characteristic of **acute pericarditis**, which along with her facial rash and proteinuria is suggestive of underlying **systemic lupus erythematosus (SLE)**. SLE is a chronic autoimmune disease predominantly affecting women age 20-40 that causes **constitutional** and **multisystemic** symptoms. **Serosal inflammation** is common in SLE and most often manifests as pleuritis or pericarditis.

Pericarditis presents with severe and constant middle or left chest pain that may radiate to the neck and shoulders (particularly the trapezius ridge). The pain increases on **inspiration** (pleuritic) and is relieved by **sitting up and leaning forward** (postural). Auscultation of the chest reveals a scratchy sound called a **pericardial friction rub** that is best heard when the patient is leaning forward or lying prone. Additional

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systemic lupus erythematosus (SLE). SLE is a chronic autoimmune disease predominantly affecting women age 20-40 that causes **constitutional** and **multisystemic** symptoms. **Serosal inflammation** is common in SLE and most often manifests as pleuritis or pericarditis.

Pericarditis presents with severe and constant middle or left chest pain that may radiate to the neck and shoulders (particularly the trapezius ridge). The pain increases on **inspiration** (pleuritic) and is relieved by **sitting up and leaning forward** (postural). Auscultation of the chest reveals a scratchy sound called a **pericardial friction rub** that is best heard when the patient is leaning forward or lying prone. Additional cardiovascular manifestations in SLE include pericardial effusion, verrucous (Libman-Sacks) endocarditis, and increased risk of coronary artery disease.

(Choice A) Aortic dissection occurs in patients with long-standing hypertension and those with Marfan and Ehlers-Danlos syndromes (the 2 latter conditions cause cystic medial necrosis of the aortic wall). It presents with abrupt-onset, severe, tearing chest pain that radiates to the back.

(Choice B) Cardiac tamponade is caused by accumulation of fluid in the pericardial space that prevents the heart from filling properly in diastole. It presents with dyspnea and tachypnea. Physical examination shows distended neck veins, hypotension, diminished heart sounds, and pulsus paradoxus (drop in systolic blood pressure ≥ 10 mm Hg on inspiration).

(Choice C) Intimal hyperplasia of the pulmonary arteries is characteristic of both primary and secondary pulmonary hypertension. It presents with dyspnea, malaise, and findings associated with right ventricular failure (eg, jugular venous distention, pedal edema, hepatomegaly). Severe symptomatic pulmonary hypertension is a rare complication of SLE.

(Choice D) Non-bacterial endocarditis (verrucous or Libman-Sacks endocarditis) refers to small, wart-like fibrinous lesions and generalized thickening that can affect the heart valves of patients with SLE. These are typically asymptomatic but may lead to valvular insufficiency or embolism.

Educational objective:

Pericarditis is the most common cardiovascular manifestation associated with systemic lupus erythematosus. It presents with sharp pleuritic chest pain that is relieved by sitting up and leaning forward.

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A 42-year-old man is found dead at home. His medical problems included hypertension and dyslipidemia, but he had been noncompliant with his medications. The patient had a lengthy smoking history and, despite constant urging from his physicians to stop smoking, he had only quit recently. An autopsy is requested by the family. Pathological examination shows complete thrombotic occlusion of the left main coronary artery and diffuse atherosclerotic vascular disease characterized by multiple atheromas. Along with a lipid core, these atheromas have a fibrous cap formed from dense deposition of collagen. Which of the following cells are directly responsible for synthesizing this fibrous cap?

- ☐ A. Endothelial cells
- ☐ B. Interstitial fibroblasts
- ☐ C. Macrophages
- ☐ D. Smooth muscle cells
- ☐ E. T lymphocytes

Submit

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TUTOR

A 42-year-old man is found dead at home. His medical problems included hypertension and dyslipidemia, but he had been noncompliant with his medications. The patient had a lengthy smoking history and, despite constant urging from his physicians to stop smoking, he had only quit recently. An autopsy is requested by the family. Pathological examination shows complete thrombotic occlusion of the left main coronary artery and diffuse atherosclerotic vascular disease characterized by multiple atheromas. Along with a lipid core, these atheromas have a fibrous cap formed from dense deposition of collagen. Which of the following cells are directly responsible for synthesizing this fibrous cap?

- ☐ A. Endothelial cells [7%]
- ☐ B. Interstitial fibroblasts [45%]
- ☐ C. Macrophages [13%]
- ☒ D. Smooth muscle cells [33%]
- ☐ E. T lymphocytes [0%]

Omitted

Correct answer
D



33%
Answered correctly



3 Seconds
Time Spent

02/01/2019
Last Updated

Explanation

Atherosclerosis is a chronic inflammatory and fibroproliferative disease. Endothelial cells, vascular smooth muscle cells (VSMCs), and leukocytes play an important role in the development and progression of this disease. The process is initiated by chronic hemodynamic stress and

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Explanation

Atherosclerosis is a chronic inflammatory and fibroproliferative disease. Endothelial cells, vascular smooth muscle cells (VSMCs), and leukocytes play an important role in the development and progression of this disease. The process is initiated by chronic hemodynamic stress and hyperlipidemia, which cause **endothelial cell injury**. This leads to increased expression of surface vascular cell-adhesion molecules (VCAMs) that allow adherence and migration of **monocytes** and T lymphocytes into the intima. The infiltrating leukocytes and dysfunctional endothelium release cytokines and growth factors (eg, platelet-derived growth factor, fibroblast growth factor, endothelin-1, interleukin-1) that promote **migration and proliferation** of **VSMCs** within the intima. VSMCs are also stimulated to synthesize extracellular matrix proteins (eg, **collagen**, elastin, proteoglycans) that form the **fibrous cap** typical of mature atheromas. Disruption of the fibrous cap with luminal thrombosis (**atherosclerotic plaque rupture**) has catastrophic clinical consequences and is responsible for the majority of acute coronary syndrome cases and sudden cardiac deaths.

(Choice A) Endothelial cell dysfunction is the earliest pathophysiologic change that precedes the formation of atherosclerotic lesions. Endothelial injury results in increased lipid permeability, leukocyte (monocytes, T cells) and platelet recruitment, and migration and proliferation of VSMC from media to the intima. However, the VSMCs are directly responsible for the synthesis of new collagen.

(Choice B) Fibroblasts are found infrequently in the tunica intima of blood vessels and are not significantly involved in atherosclerosis pathogenesis.

(Choices C and E) Activated macrophages and T lymphocytes secrete growth factors that recruit the VSMCs responsible for forming the fibrous cap, but they are not directly responsible for the dense deposition of fibrillar collagen. Macrophages also produce matrix metalloproteinases and tissue factors that degrade the extracellular matrix, causing the formation of a large, soft lipid-rich core with thinning of the fibrous cap. Such vulnerable plaques have an increased propensity for rupture.

Educational objective:
Vascular smooth muscle cells (VSMCs) are the only cells within the atherosclerotic plaque capable of synthesizing structurally important collagen

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hyperlipidemia, which cause **endothelial cell injury**. This leads to increased expression of surface vascular cell-adhesion molecules (VCAMs) that allow adherence and migration of **monocytes** and T lymphocytes into the intima. The infiltrating leukocytes and dysfunctional endothelium release cytokines and growth factors (eg, platelet-derived growth factor, fibroblast growth factor, endothelin-1, interleukin-1) that promote **migration and proliferation** of **VSMCs** within the intima. VSMCs are also stimulated to synthesize extracellular matrix proteins (eg, **collagen**, elastin, proteoglycans) that form the **fibrous cap** typical of mature atheromas. Disruption of the fibrous cap with luminal thrombosis (**atherosclerotic plaque rupture**) has catastrophic clinical consequences and is responsible for the majority of acute coronary syndrome cases and sudden cardiac deaths.

(Choice A) Endothelial cell dysfunction is the earliest pathophysiologic change that precedes the formation of atherosclerotic lesions. Endothelial injury results in increased lipid permeability, leukocyte (monocytes, T cells) and platelet recruitment, and migration and proliferation of VSMC from media to the intima. However, the VSMCs are directly responsible for the synthesis of new collagen.

(Choice B) Fibroblasts are found infrequently in the tunica intima of blood vessels and are not significantly involved in atherosclerosis pathogenesis.

(Choices C and E) Activated macrophages and T lymphocytes secrete growth factors that recruit the VSMCs responsible for forming the fibrous cap, but they are not directly responsible for the dense deposition of fibrillar collagen. Macrophages also produce matrix metalloproteinases and tissue factors that degrade the extracellular matrix, causing the formation of a large, soft lipid-rich core with thinning of the fibrous cap. Such vulnerable plaques have an increased propensity for rupture.

Educational objective:

Vascular smooth muscle cells (VSMCs) are the only cells within the atherosclerotic plaque capable of synthesizing structurally important collagen isoforms and other matrix components. Progressive enlargement of the plaque results in remodeling of the extracellular matrix and VSMC death, promoting development of vulnerable plaques with an increased propensity for rupture.

References

- The role of vascular smooth muscle cells on the pathogenesis of atherosclerosis

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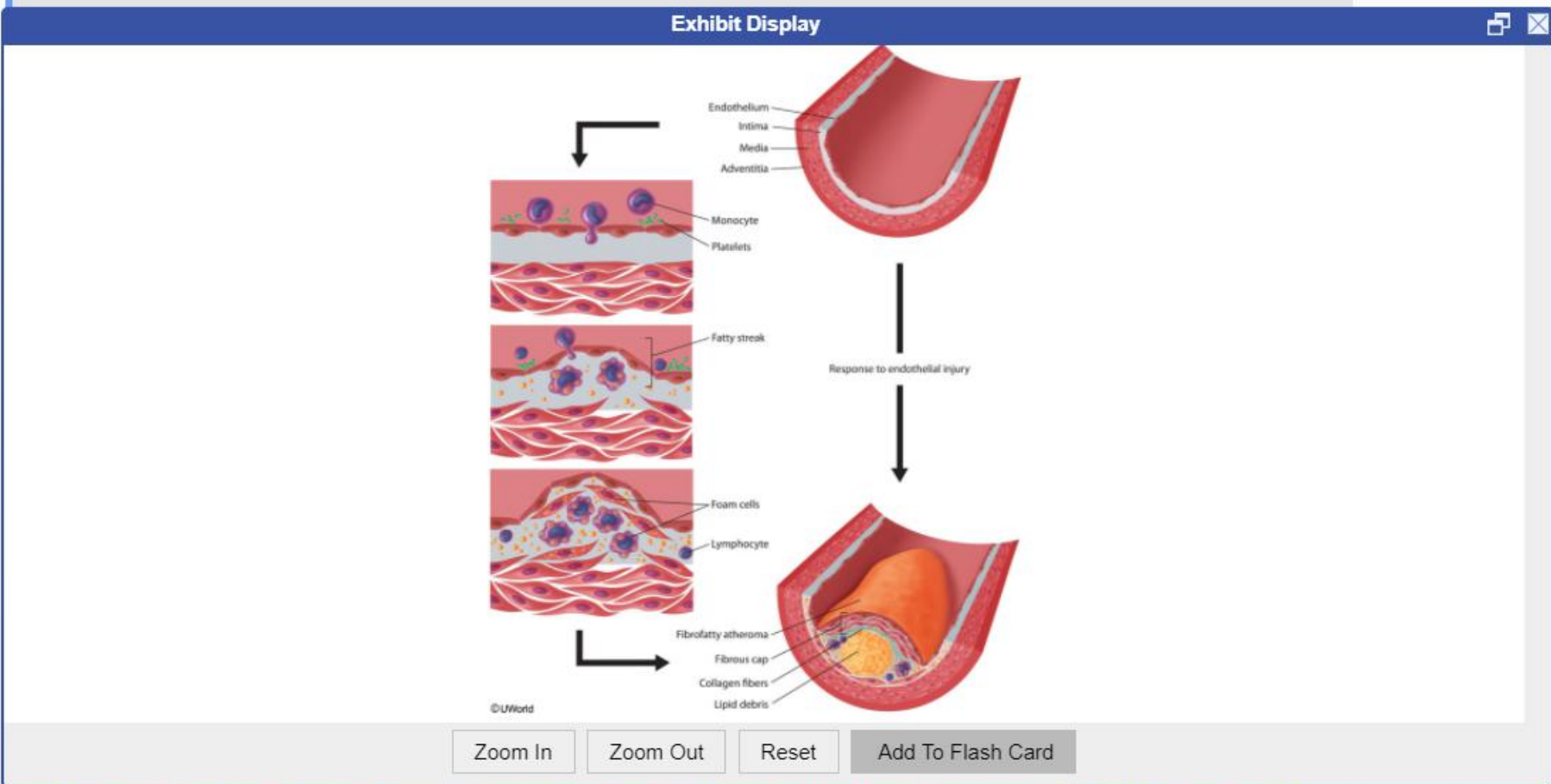
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tissue factors that degrade the extracellular matrix, causing the formation of a large, soft lipid-rich core with thinning of the fibrous cap. Such

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A 56-year-old woman with unknown medical history is brought to the emergency department in an unresponsive state. Paramedics were called for sudden-onset, left-sided weakness, and when they arrived at the patient's house she was having a generalized tonic-clonic seizure. Antiseizure medications were given and the patient was subsequently intubated. Evaluation shows a comatose woman with left hemiplegia. Neuroimaging reveals a large infarction in the right middle cerebral artery territory with cerebral edema. Despite aggressive interventions, the patient dies from brain herniation. Autopsy reveals multiple small, nondestructive masses attached to the edges of the mitral valve leaflets. Microscopy shows that these masses are composed of platelet-rich thrombi, but no organisms are present. Which of the following conditions is most closely associated with this patient's heart valve findings?

- ☐ A. Advanced malignancy
- ☐ B. Chronic liver failure
- ☐ C. Dilated cardiomyopathy
- ☐ D. End-stage renal disease
- ☐ E. Large-vessel vasculitis

Submit

Block Time Remaining: 00:00:55

TUTOR



A 56-year-old woman with unknown medical history is brought to the emergency department in an unresponsive state. Paramedics were called for sudden-onset, left-sided weakness, and when they arrived at the patient's house she was having a generalized tonic-clonic seizure. Antiseizure medications were given and the patient was subsequently intubated. Evaluation shows a comatose woman with left hemiplegia. Neuroimaging reveals a large infarction in the right middle cerebral artery territory with cerebral edema. Despite aggressive interventions, the patient dies from brain herniation. Autopsy reveals multiple small, nondestructive masses attached to the edges of the mitral valve leaflets. Microscopy shows that these masses are composed of platelet-rich thrombi, but no organisms are present. Which of the following conditions is most closely associated with this patient's heart valve findings?

- ☒ A. Advanced malignancy [40%]
☐ B. Chronic liver failure [5%]
☐ C. Dilated cardiomyopathy [16%]
☐ D. End-stage renal disease [21%]
☐ E. Large-vessel vasculitis [17%]

Omitted

Correct answer
A40%
Answered correctly3 Seconds
Time Spent12/30/2018
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Explanation

The autopsy finding of **sterile platelet-rich thrombi** attached to the mitral valve leaflets is characteristic of **nonbacterial thrombotic**

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Explanation

The autopsy finding of **sterile platelet-rich thrombi** attached to the mitral valve leaflets is characteristic of **nonbacterial thrombotic endocarditis** (NBTE) (marantic endocarditis). The pathogenesis of NBTE is thought to begin with valvular endothelial injury caused by inflammatory cytokines, which triggers platelet deposition in the presence of an underlying **hypercoagulable state**. NBTE is most commonly associated with **advanced malignancy** (especially mucinous adenocarcinoma) and systemic lupus erythematosus (Libman-Sacks endocarditis); less commonly, it can occur with antiphospholipid syndrome, disseminated intravascular coagulation, and extensive burns.

Histologically, NBTE vegetations consist of bland thrombus with strands of fibrin, immune complexes, and mononuclear cells (white thrombus). The vegetations typically affect the left-sided heart valves (mitral or aortic) and are often asymptomatic; however, **systemic embolization** (eg, stroke, acute limb ischemia) can occur and is the most common presentation of NBTE.

(Choice B) Chronic liver failure (ie, cirrhosis) can be complicated by portal vein thrombosis; however, it is not associated with NBTE.

(Choice C) Dilated cardiomyopathy is characterized by ventricular enlargement and reduced systolic function. It can occasionally lead to left ventricular mural thrombus with risk of systemic embolization, but it is not associated with NBTE.

(Choice D) End-stage renal disease can accelerate aortic valve calcification, but it is not associated with NBTE.

(Choice E) Temporal (giant cell) arteritis, a large-vessel vasculitis that typically presents with headaches and fever, is associated with an increased risk of venous thromboembolic disease. Although associated NBTE has been reported, it is quite rare.

Educational objective:

Nonbacterial thrombotic endocarditis is a form of noninfectious endocarditis characterized by valvular deposition of sterile platelet-rich thrombi. It likely results from valvular damage due to inflammatory cytokines in the setting of an underlying hypercoagulable state, and it is most commonly seen with advanced malignancy (especially mucinous adenocarcinoma) or systemic lupus erythematosus.

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A 54-year-old man comes to the office due to 2 days of redness and pain in his right arm. He was recently diagnosed with superficial thrombophlebitis involving the left lower and upper extremities. His symptoms then subsided within days of nonsteroidal anti-inflammatory drug therapy only. The patient has no other past medical history. He has some abdominal discomfort that he attributes to gastroesophageal reflux disease. Temperature is 37.6 C (99.7 F). On examination, there is erythema and tenderness extending linearly from the right forearm to just anterior to the right antecubital fossa. No fluid collection is palpable. Complete blood count shows mild leukocytosis. The patient's symptoms may indicate the presence of which of the following?

A. Celiac sprue

B. Chlamydial infection

C. Hyperthyroidism

D. Polycystic kidneys

E. Visceral cancer

Submit

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2/7/2019



A 54-year-old man comes to the office due to 2 days of redness and pain in his right arm. He was recently diagnosed with superficial thrombophlebitis involving the left lower and upper extremities. His symptoms then subsided within days of nonsteroidal anti-inflammatory drug therapy only. The patient has no other past medical history. He has some abdominal discomfort that he attributes to gastroesophageal reflux disease. Temperature is 37.6 C (99.7 F). On examination, there is erythema and tenderness extending linearly from the right forearm to just anterior to the right antecubital fossa. No fluid collection is palpable. Complete blood count shows mild leukocytosis. The patient's symptoms may indicate the presence of which of the following?

- ☐ A. Celiac sprue [22%]
- ☐ B. Chlamydial infection [8%]
- ☐ C. Hyperthyroidism [3%]
- ☐ D. Polycystic kidneys [5%]
- ☒ E. Visceral cancer [60%]

Omitted

Correct answer
E60%
Answered correctly2 Seconds
Time Spent10/27/2018
Last Updated

Explanation

Migratory thrombophlebitis should raise suspicion for **cancer**. Hypercoagulability is a very common paraneoplastic syndrome seen most frequently in **visceral** adenocarcinomas of the **pancreas**, colon, and lung. **Hypercoagulability** develops because adenocarcinomas produce a

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Explanation

Migratory thrombophlebitis should raise suspicion for **cancer**. Hypercoagulability is a very common paraneoplastic syndrome seen most frequently in **visceral** adenocarcinomas of the **pancreas**, colon, and lung. **Hypercoagulability** develops because adenocarcinomas produce a thromboplastin-like substance capable of causing chronic intravascular coagulations that can disseminate and tend to migrate.

Migratory superficial thrombophlebitis, known as **Trousseau syndrome**, is named after Armand Trousseau, a well-respected French physician who first described the association with cancer. Trousseau later diagnosed his own visceral cancer after developing the syndrome.

Trousseau also described the Trousseau sign (hand/forearm muscle spasms on sphygmomanometric measurement) associated with hypocalcemia.

(Choice A) Celiac sprue has been associated with a hemorrhagic diathesis (rather than abnormal thrombosis). The malabsorption resulting from celiac sprue can result in vitamin K deficiency.

(Choices B and D) Polycystic kidney disease is not among the conditions commonly associated with an acquired prothrombotic state. Disseminated chlamydia infections are classically associated with reactive arthritis (not thrombophlebitis), and infections in general can create a systemic inflammatory state that favors thrombosis. However, malignancy would be a more likely explanation for migratory superficial thrombophlebitis accompanied by vague abdominal pain.

(Choice C) Hyperthyroidism has been associated with a hypercoagulable state and is occasionally reported in cases of cerebral venous thrombosis, but there is no evidence of a significant association with venous thrombotic events.

Educational objective:

A paraneoplastic syndrome of hypercoagulability may be seen in some patients with cancer, especially adenocarcinomas of the pancreas, colon, or lung. Superficial venous thromboses may therefore appear in one site and then resolve, only to recur in another site. This is known as Trousseau syndrome (migratory superficial thrombophlebitis), an indication of visceral cancer.

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TUTOR

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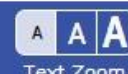
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Windows Taskbar

System Tray



Autopsy of a 78-year-old man demonstrates decreased left ventricular cavity size and a sigmoid-shaped ventricular septum. Light microscopy shows increased collagen content within the ventricular wall. Some myocardial cells also have brownish perinuclear cytoplasmic inclusions. The changes described are most consistent with which of the following conditions?

- ☐ A. Dilated cardiomyopathy
- ☐ B. Hemolytic anemia
- ☐ C. Hypertrophic cardiomyopathy
- ☐ D. Ischemic heart disease
- ☐ E. Long-standing hypertension
- ☐ F. Normal aging

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TUTOR





Autopsy of a 78-year-old man demonstrates decreased left ventricular cavity size and a sigmoid-shaped ventricular septum. Light microscopy shows increased collagen content within the ventricular wall. Some myocardial cells also have brownish perinuclear cytoplasmic inclusions. The changes described are most consistent with which of the following conditions?

- ☐ A. Dilated cardiomyopathy [2%]
- ☐ B. Hemolytic anemia [1%]
- ☐ C. Hypertrophic cardiomyopathy [18%]
- ☐ D. Ischemic heart disease [11%]
- ☐ E. Long-standing hypertension [13%]
- ☒ F. Normal aging [53%]

Omitted

Correct answer
F53%
Answered correctly2 Seconds
Time Spent01/09/2019
Last Updated

Explanation

The morphologic changes described are consistent with **normal aging** of the heart. Aging is associated with **decreased left ventricular chamber size**, particularly in the apex-to-base dimension. This decrease in chamber length causes the ventricular septum to acquire a **sigmoid shape**, with the basilar portion bulging into the left ventricular outflow tract. Atrophy of the myocardium results in **increased interstitial**

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Text Zoom

The morphologic changes described are consistent with **normal aging** of the heart. Aging is associated with **decreased left ventricular chamber size**, particularly in the apex-to-base dimension. This decrease in chamber length causes the ventricular septum to acquire a **sigmoid shape**, with the basilar portion bulging into the left ventricular outflow tract. Atrophy of the myocardium results in **increased interstitial connective tissue**, often with concomitant extracellular amyloid deposition. Within the cardiomyocytes, there is a progressive accumulation of cytoplasmic granules containing brownish **lipofuscin pigment** (the result of indigestible byproducts of subcellular membrane lipid oxidation).

(Choice A) Dilated cardiomyopathy causes an increase in the left ventricular chamber size. There is an increased risk of mural thrombi with this condition due to contractile dysfunction.

(Choice B) Chronic hemolytic anemia may result in the formation of hemosiderin granules containing excess iron inside cardiac myocytes (hemosiderosis), which can be visualized with Prussian-blue stain. Excessive deposition of iron within the myocytes usually leads to dilated or restrictive cardiomyopathy.

(Choice C) Hypertrophic cardiomyopathy (idiopathic hypertrophic subaortic stenosis) causes asymmetric septal hypertrophy with disproportionate thickening of the ventricular septum compared to the left ventricular free wall. Microscopic examination shows extreme myocyte hypertrophy, disorganized myocyte bundles, and myofiber disarray.

(Choice D) Chronic ischemic heart disease tends to result in progressive congestive heart failure and ventricular dilation. Histologically, the major findings are diffuse subendocardial vacuolization and fibrosis.

(Choice E) Long-standing hypertension results in hypertensive heart disease. Although left ventricular chamber size may be reduced in this condition, the major morphologic change is concentric left ventricular hypertrophy.

Educational objective:

Normal morphological changes in the aging heart include a decrease in left ventricular chamber apex-to-base dimension, development of a sigmoid-shaped ventricular septum, myocardial atrophy with increased collagen deposition, and accumulation of cytoplasmic lipofuscin pigment within cardiomyocytes.

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TUTOR

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Windows Taskbar

System Tray

A 56-year-old man with a history of hypertension and hyperlipidemia is brought to the emergency department with chest pain, diaphoresis, and lightheadedness. His symptoms started an hour ago while he was stacking boxes in his garage. He describes the pain as a tight squeezing sensation in the center of his chest that radiates down his left arm. Electrocardiogram shows ST-segment elevation in leads II, III, and aVF. The tissues affected by this patient's acute condition will most likely develop which of the following histologic changes over the next few days?

- ☐ A. Caseous necrosis
- ☐ B. Coagulative necrosis
- ☐ C. Fat necrosis
- ☐ D. Fibrinoid necrosis
- ☐ E. Liquefactive necrosis
- ☐ F. Tissue calcification

Submit

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Question Id: 178

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Lab Values

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A 56-year-old man with a history of hypertension and hyperlipidemia is brought to the emergency department with chest pain, diaphoresis, and lightheadedness. His symptoms started an hour ago while he was stacking boxes in his garage. He describes the pain as a tight squeezing sensation in the center of his chest that radiates down his left arm. Electrocardiogram shows ST-segment elevation in leads II, III, and aVF. The tissues affected by this patient's acute condition will most likely develop which of the following histologic changes over the next few days?

A. Caseous necrosis [0%]

B. Coagulative necrosis [90%]

C. Fat necrosis [0%]

D. Fibrinoid necrosis [6%]

E. Liquefactive necrosis [1%]

F. Tissue calcification [0%]

Omitted

Correct answer B

90%

Answered correctly

2 Seconds

Time Spent

09/28/2018

Last Updated

Explanation

Patterns of tissue necrosis

Block Time Remaining: 00:01:03

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Explanation

Patterns of tissue necrosis		
Type	Etiology	Morphology
Liquefactive necrosis	- Severe bacterial infections (eg, gangrene) - CNS infarcts	- Necrotic tissue is digested by hydrolytic enzymes into a viscous fluid: - Infected abscess fluid is creamy yellow due to dead leukocytes (pus) - Brain infarcts resolve into CSF-filled spaces
Fibrinoid necrosis	- Hypertension - Vasculitis	- Damaged vessels leak fibrin/immune complexes - Eosinophilic layer of proteinaceous material in vessel walls
Fat necrosis	- Acute pancreatitis - Trauma (subcutaneous adipose tissue)	- Free fatty acids released by active enzymes (eg, lipases) or mechanical damage - Fatty acids combine with calcium, forming chalky-white deposits (saponification)
Caseous necrosis	- Mycobacterial infections - Fungal infections (eg, <i>Histoplasma</i>, <i>Cryptococcus</i>, <i>Coccidioides</i>)	- Friable, cheeselike material composed of cell fragments & amorphous proteinaceous debris - Surrounded by epithelioid macrophages & giant cells (granuloma)
Coagulative	Irreversible ischemic injury outside CNS	Tissue architecture is preserved due to denaturation of lytic enzymes:

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necrosis	Fungal infections (eg, <i>Histoplasma</i>, <i>Cryptococcus</i>, <i>Coccidioides</i>)	Surrounded by epithelioid macrophages & giant cells (granuloma)
Coagulative necrosis	Irreversible ischemic injury outside CNS	- Tissue architecture is preserved due to denaturation of lytic enzymes: - Cells are anucleate with eosinophilic cytoplasm - Leukocytes eventually infiltrate & digest necrotic tissue

CSF = cerebrospinal fluid.

This patient's electrocardiogram findings indicate acute myocardial injury due to transmural ischemia of the inferior wall of the heart. Complete cardiac muscle ischemia for >30 minutes is associated with progressive cardiomyocyte death and **coagulative necrosis** within the affected vascular territory.

Necrosis is an uncontrolled process of cell death that results in denaturation of cellular proteins and enzymatic digestion of cellular components. Unlike apoptosis, necrosis leads to a loss of membrane integrity and leakage of proinflammatory intracellular material that can injure the surrounding tissue. Necrosis can occur in a number of distinct morphologic patterns. However, in most tissues, ischemic injury results in coagulative necrosis because the digestive enzymes denature before they can significantly disrupt tissue integrity.

(Choice D) Fibrinoid necrosis is a histologic pattern of injury seen in the walls of blood vessels affected by vasculitis syndromes (eg, polyarteritis nodosa), malignant hypertension, and diabetes mellitus. It results from immune complexes and/or plasma proteins (eg, fibrin) leaking through the damaged intima and being deposited within the vessel wall. It appears histologically as a circumferential ring of pink, amorphous material surrounding the vascular lumen.

(Choice F) Dystrophic calcification frequently occurs in areas of necrosis that escape removal by phagocytes. However, it develops slowly over the months and years following the initial injury. Calcium salts can appear grossly as white granules and microscopically as amorphous basophilic material on H&E stain.

Educational objective:

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Settings

necrosis

• Irreversible ischemic injury outside CNS

- Cells are anucleate with eosinophilic cytoplasm
- Leukocytes eventually infiltrate & digest necrotic tissue

CSF = cerebrospinal fluid.

This patient's electrocardiogram findings indicate acute myocardial injury due to transmural ischemia of the inferior wall of the heart. Complete cardiac muscle ischemia for >30 minutes is associated with progressive cardiomyocyte death and **coagulative necrosis** within the affected vascular territory.

Necrosis is an uncontrolled process of cell death that results in denaturation of cellular proteins and enzymatic digestion of cellular components. Unlike apoptosis, necrosis leads to a loss of membrane integrity and leakage of proinflammatory intracellular material that can injure the surrounding tissue. Necrosis can occur in a number of distinct morphologic patterns. However, in most tissues, ischemic injury results in coagulative necrosis because the digestive enzymes denature before they can significantly disrupt tissue integrity.

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(Choice F) Dystrophic calcification frequently occurs in areas of necrosis that escape removal by phagocytes. However, it develops slowly over the months and years following the initial injury. Calcium salts can appear grossly as white granules and microscopically as amorphous basophilic material on H&E stain.

Educational objective:

Lethal tissue ischemia causes coagulative necrosis in most tissues, including the myocardium. The exception is ischemic cell death in the central nervous system, which causes liquefactive necrosis.

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necrosis

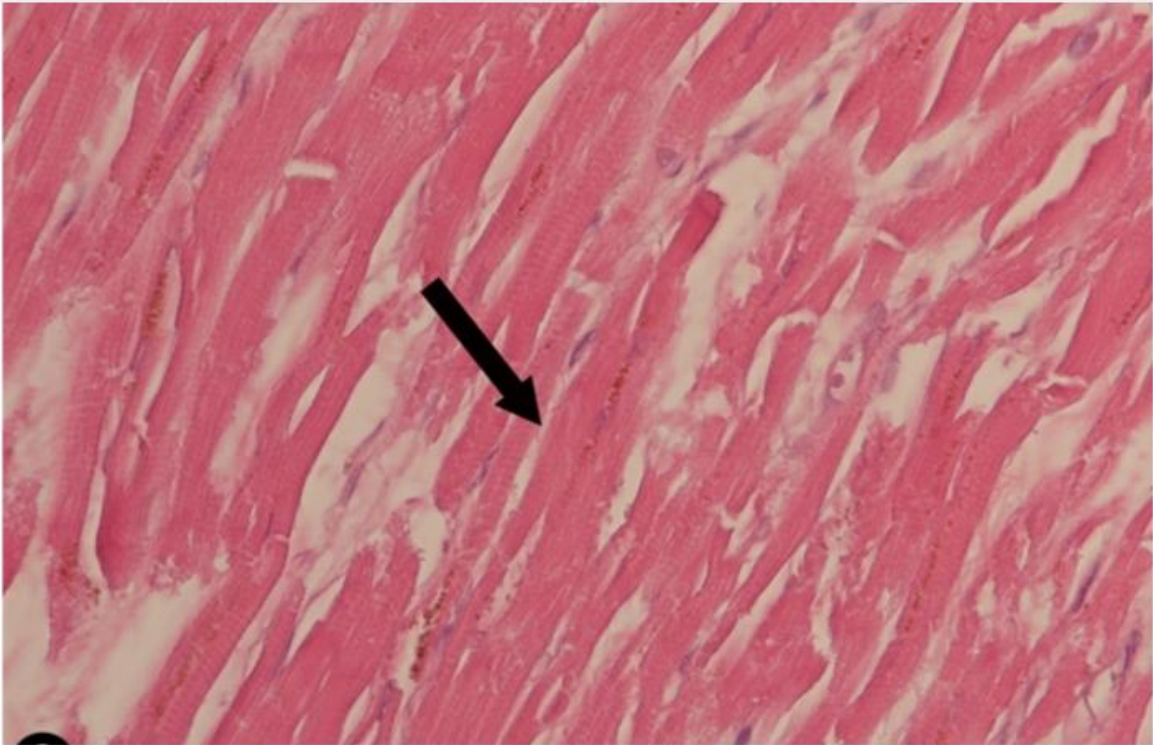
irreversible ischemic injury outside CNS

Cells are anucleate with eosinophilic cytoplasm

Exhibit Display

Myocardial coagulative necrosis

Myocardial coagulative necrosis



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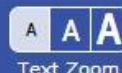
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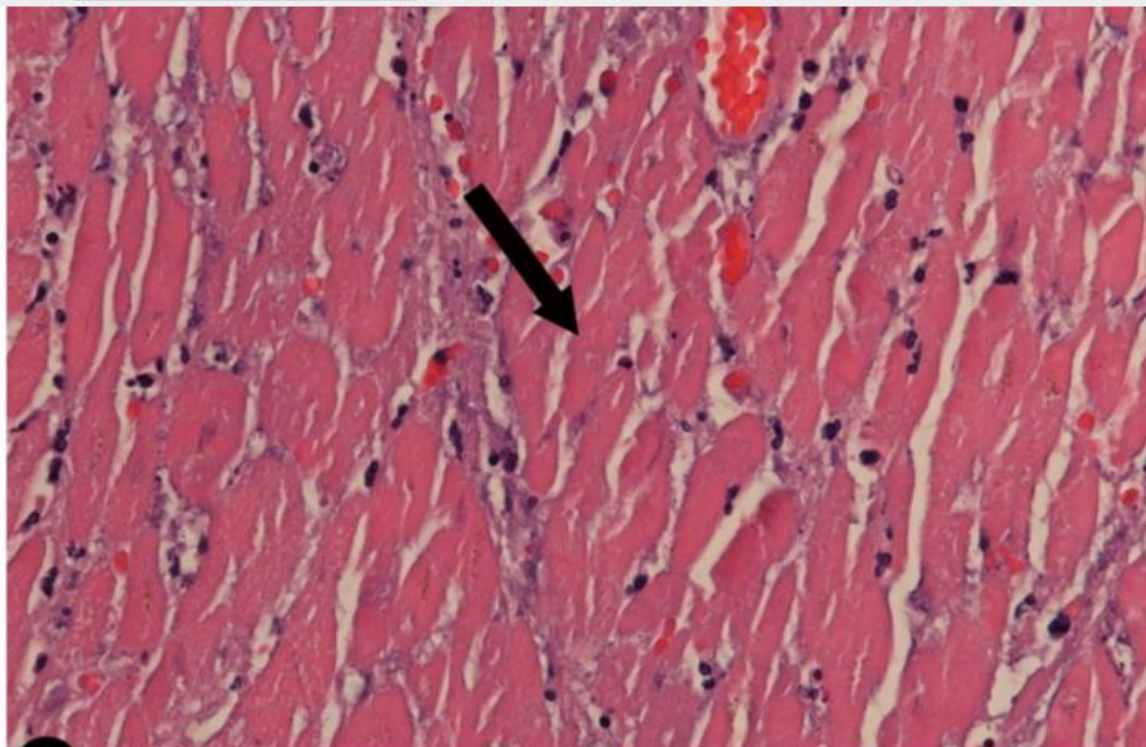


necrosis

• Irreversible ischemic injury outside CNS

• Cells are anucleate with eosinophilic cytoplasm

Exhibit Display

Myocardial coagulative necrosis [Myocardial coagulative necrosis](#)

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Question Id: 37

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Text Zoom

A 55-year-old man is brought to the emergency department due to severe substernal chest pain that is accompanied by sweating and mild dyspnea. The pain started as midline chest discomfort several hours ago but then progressively worsened such that the patient could not fall asleep. He received sublingual nitroglycerin in the ambulance without significant pain relief. His medical history includes hypertension, type 2 diabetes mellitus, and hyperlipidemia. ECG demonstrates normal sinus rhythm and ST-segment elevation in leads I, aVL, and V1-V4, with deep Q wave development over the next several hours. Cardiac catheterization in this patient would most likely show which of the following?

A. Atherosclerotic plaque obstructing 50% of the coronary artery lumen, no thrombus

B. Atherosclerotic plaque obstructing 80% of the coronary artery lumen, no thrombus

C. Ruptured atherosclerotic plaque with fully obstructive thrombus

D. Significant coronary artery vasospasm causing flow obstruction

E. Ulcerated atherosclerotic plaque with partially obstructive thrombus

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Text Zoom

A 55-year-old man is brought to the emergency department due to severe substernal chest pain that is accompanied by sweating and mild dyspnea. The pain started as midline chest discomfort several hours ago but then progressively worsened such that the patient could not fall asleep. He received sublingual nitroglycerin in the ambulance without significant pain relief. His medical history includes hypertension, type 2 diabetes mellitus, and hyperlipidemia. ECG demonstrates normal sinus rhythm and ST-segment elevation in leads I, aVL, and V1-V4, with deep Q wave development over the next several hours. Cardiac catheterization in this patient would most likely show which of the following?

A. Atherosclerotic plaque obstructing 50% of the coronary artery lumen, no thrombus [0%]

B. Atherosclerotic plaque obstructing 80% of the coronary artery lumen, no thrombus [9%]

C. Ruptured atherosclerotic plaque with fully obstructive thrombus [81%]

D. Significant coronary artery vasospasm causing flow obstruction [4%]

E. Ulcerated atherosclerotic plaque with partially obstructive thrombus [4%]

Omitted

Correct answer
C

81%

Answered correctly

3 Seconds

Time Spent

01/10/2019

Last Updated

Explanation

This patient with persistent substernal chest pain and ECG demonstrating ST elevation in the anterolateral leads has an **ST-elevation myocardial infarction** (STEMI). In the evolution of STEMI on ECG, peaked T waves in the affected leads are typically the first manifestation, followed by the

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Lab Values

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Text Zoom

Explanation

This patient with persistent substernal chest pain and ECG demonstrating ST elevation in the anterolateral leads has an **ST-elevation myocardial infarction (STEMI)**. In the evolution of STEMI on ECG, peaked T waves in the affected leads are typically the first manifestation, followed by the appearance of ST elevation within several minutes, then Q waves within hours to days. Patients with STEMI typically have sudden-onset substernal chest pain that may be accompanied by nausea, diaphoresis, or dyspnea; the pain does not improve with rest or the administration of short-acting nitrates (eg, nitroglycerin).

Myocardial infarction is usually caused by acute **atherosclerotic plaque rupture** with release of lipids, collagen, and tissue factor into the blood stream; because these contents are highly thrombogenic, there is rapid formation of **overlying thrombus**. In STEMI, the overlying thrombus is extensive enough to **fully occlude** the coronary artery lumen, completely blocking blood flow to a portion of the heart and leading to transmural (full-thickness) infarction of the myocardial wall. Partially obstructive thrombus (**Choice E**) is likely to produce non-STEMI or unstable angina. Non-STEMI is associated with impaired blood flow to only the subendocardial region; the symptomatic presentation is similar to STEMI, but ECG reveals ST segment and T wave abnormalities with no ST elevation.

(Choice A) Atherosclerotic plaque obstructing 50% of the coronary artery lumen is likely to be asymptomatic. Symptoms of stable angina do not typically become prominent until >70% of the cross-sectional area of the coronary artery lumen is occluded.

(Choice B) Atherosclerotic plaque obstructing 80% of the coronary artery lumen is likely to cause stable angina, characterized by substernal or left-sided chest pressure, tightness, or pain that is precipitated by exertion and relieved by rest or short-acting nitrates.

(Choice D) Coronary artery vasospasm (vasospastic or Prinzmetal angina) results from hyperactivity of intimal smooth muscle, usually in the absence of significant atherosclerotic coronary artery disease. Transient anginal chest pain with corresponding ST elevation on ECG can occur; however, the chest pain should rapidly resolve with nitroglycerin.

Educational objective:

ST-elevation myocardial infarction involves transmural (full-thickness) infarction of the myocardial wall, and usually results from acute

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stream; because these contents are highly thrombogenic, there is rapid formation of **overlying thrombus**. In STEMI, the overlying thrombus is extensive enough to **fully occlude** the coronary artery lumen, completely blocking blood flow to a portion of the heart and leading to transmural (full-thickness) infarction of the myocardial wall. Partially obstructive thrombus (**Choice E**) is likely to produce non-STEMI or unstable angina. Non-STEMI is associated with impaired blood flow to only the subendocardial region; the symptomatic presentation is similar to STEMI, but ECG reveals ST segment and T wave abnormalities with no ST elevation.

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(Choice B) Atherosclerotic plaque obstructing 80% of the coronary artery lumen is likely to cause stable angina, characterized by substernal or left-sided chest pressure, tightness, or pain that is precipitated by exertion and relieved by rest or short-acting nitrates.

(Choice D) Coronary artery vasospasm (vasospastic or Prinzmetal angina) results from hyperactivity of intimal smooth muscle, usually in the absence of significant atherosclerotic coronary artery disease. Transient anginal chest pain with corresponding ST elevation on ECG can occur; however, the chest pain should rapidly resolve with nitroglycerin.

Educational objective:

ST-elevation myocardial infarction involves transmural (full-thickness) infarction of the myocardial wall, and usually results from acute atherosclerotic plaque rupture with the development of overlying thrombus that fully occludes the coronary artery lumen. It classically presents with sudden-onset substernal chest pain that is not relieved by rest or short-acting nitrates. ECG demonstrates ST elevation in the affected leads with subsequent development of Q waves.

References

- Atherosclerotic plaque rupture—pathologic basis of plaque stability and instability.

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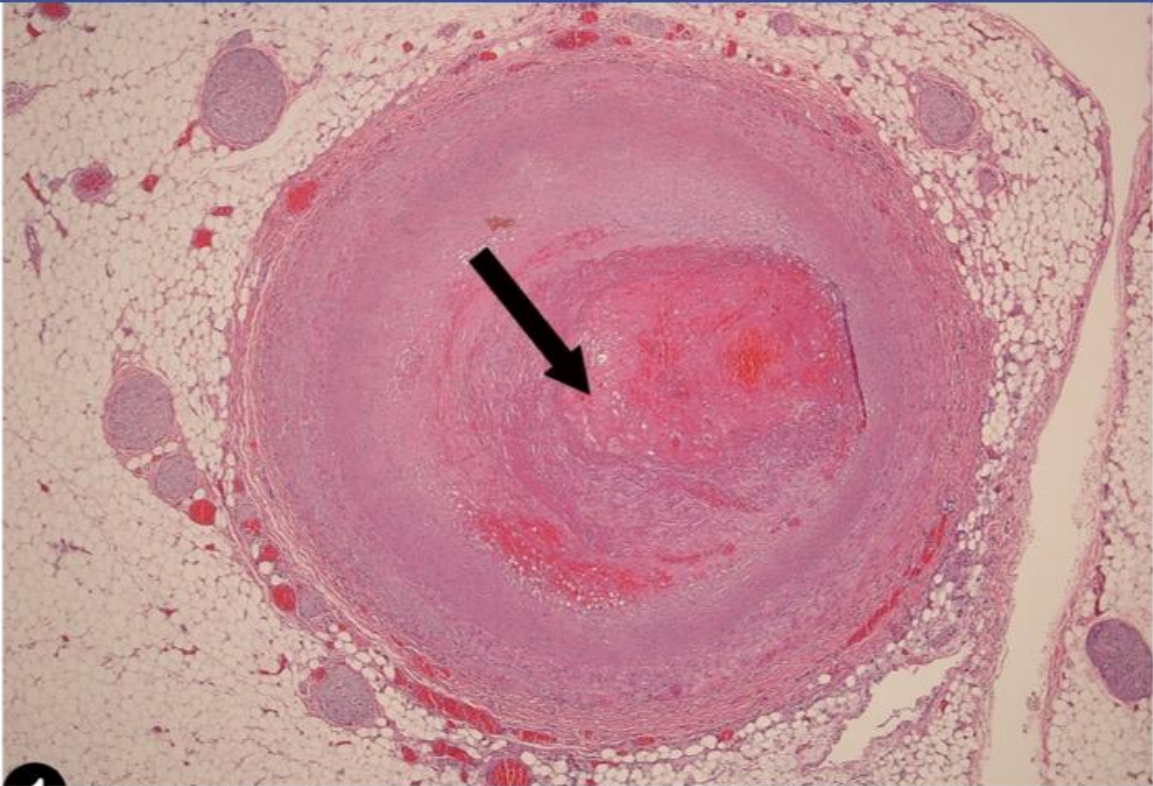
Notes

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Text Zoom

Exhibit Display

A histological section of a blood vessel, likely an artery, stained with hematoxylin and eosin (H&E). The vessel lumen is the central, clear area. A black arrow points to the lumen. The vessel wall is thick and shows a prominent internal elastic lamina. The surrounding tissue is composed of various cell types and connective tissue.

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TUTOR

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2/7/2019



A 34-year-old Asian female is hospitalized with progressive exertional dyspnea, lower extremity edema and cough. She also describes frequent nocturnal episodes of breathlessness and recent hoarseness. She does not use tobacco, alcohol or drugs. Auscultation reveals loud first and second heart sounds and a mid-diastolic rumble best heard at the cardiac apex. This patient's hoarseness is most likely caused by:

- ☐ A. Laryngeal edema
- ☐ B. Impaired arterial supply
- ☐ C. Nerve impingement
- ☐ D. Epithelial sloughing
- ☐ E. Vocal cord polyps

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A 34-year-old Asian female is hospitalized with progressive exertional dyspnea, lower extremity edema and cough. She also describes frequent nocturnal episodes of breathlessness and recent hoarseness. She does not use tobacco, alcohol or drugs. Auscultation reveals loud first and second heart sounds and a mid-diastolic rumble best heard at the cardiac apex. This patient's hoarseness is most likely caused by:

- ☐ A. Laryngeal edema [11%]
- ☐ B. Impaired arterial supply [6%]
- ☒ C. Nerve impingement [78%]
- ☐ D. Epithelial sloughing [1%]
- ☐ E. Vocal cord polyps [1%]

Omitted

Correct answer
C78%
Answered correctly3 Seconds
Time Spent02/06/2019
Last Updated

Explanation

In rare instances, the left recurrent laryngeal nerve may be compressed to the point of neurapraxia (failure of nerve conduction due to blunt injury) by enlargement of the left atrium and/or other structures in the vicinity of its course as it loops behind the ligamentum arteriosum, underneath and around the aortic arch, and back up alongside the trachea to the larynx. Mitral stenosis can cause left atrial dilatation sufficient to impinge on the left recurrent laryngeal nerve (Ortner syndrome).

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Lab Values

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Text Zoom

Explanation

In rare instances, the left recurrent laryngeal nerve may be compressed to the point of neurapraxia (failure of nerve conduction due to blunt injury) by enlargement of the left atrium and/or other structures in the vicinity of its course as it loops behind the ligamentum arteriosum, underneath and around the aortic arch, and back up alongside the trachea to the larynx. Mitral stenosis can cause left atrial dilatation sufficient to impinge on the left recurrent laryngeal nerve (Ortner syndrome).

The recurrent laryngeal nerves innervate all of the intrinsic muscles of the larynx except the cricothyroid muscle. Paresis of vocal cord muscles innervated by left recurrent laryngeal nerve can cause hoarseness.

(Choice A) Laryngeal edema can cause hoarseness but is commonly due to direct laryngeal inflammation (e.g. by an upper respiratory infection).

(Choice B) Vascular disease or injury resulting in ischemia of the vocal cords, the recurrent laryngeal nerves, and/or the vagal motor nuclei in the brain stem could result in laryngeal neuromuscular dysfunction, producing hoarseness. However, the patient's history and presentation make MS a more likely culprit.

(Choice D) Hoarseness is predominantly due to vocal cord disease (including mucosal inflammation or edema) or vocal cord malfunction. Epithelial sloughing could be associated with laryngeal mucosal disease and might be seen in asthma or airway exposure to noxious external gases or gastric acid. However, the other components of the patient's history and physical make severe MS more likely as an underlying pathogenic factor.

(Choice E) Vocal cord epithelial or mucosal polyps can also cause hoarseness, but are less likely etiologies here, given the patient's history.

Educational Objective:

Left atrial enlargement can sometimes cause left recurrent laryngeal nerve impingement. Neurapraxia resulting in left vocal cord paresis and hoarseness may result.

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A 64-year-old man dies suddenly while playing tennis. In the preceding months, the patient experienced fatigue and some exertional dyspnea. Autopsy reveals rupture of an unsuspected ascending aortic aneurysm. Heart examination shows a septal thickness of 1.2 cm (normal: <1.1), a posterior wall thickness of 1.2 cm (normal: <1.1), and an internal left ventricular diameter of 6.8 cm (normal: 3.5-5.9). No focal myocardial scarring is seen. Which of the following is the most likely cause of the cardiac findings seen in this individual?

A. Aortic regurgitation

B. Aortic stenosis

C. Hypertrophic cardiomyopathy

D. Obstructive coronary artery disease

E. Restrictive cardiomyopathy

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A 64-year-old man dies suddenly while playing tennis. In the preceding months, the patient experienced fatigue and some exertional dyspnea. Autopsy reveals rupture of an unsuspected ascending aortic aneurysm. Heart examination shows a septal thickness of 1.2 cm (normal: <1.1), a posterior wall thickness of 1.2 cm (normal: <1.1), and an internal left ventricular diameter of 6.8 cm (normal: 3.5-5.9). No focal myocardial scarring is seen. Which of the following is the most likely cause of the cardiac findings seen in this individual?

Omitted

Correct answer
A

57%

Answered correctly

2 Seconds

Time Spent

01/31/2019

Last Updated

Explanation

Concentric & eccentric cardiac hypertrophy

Normal

Block Time Remaining: 00:01:11

TUTOR

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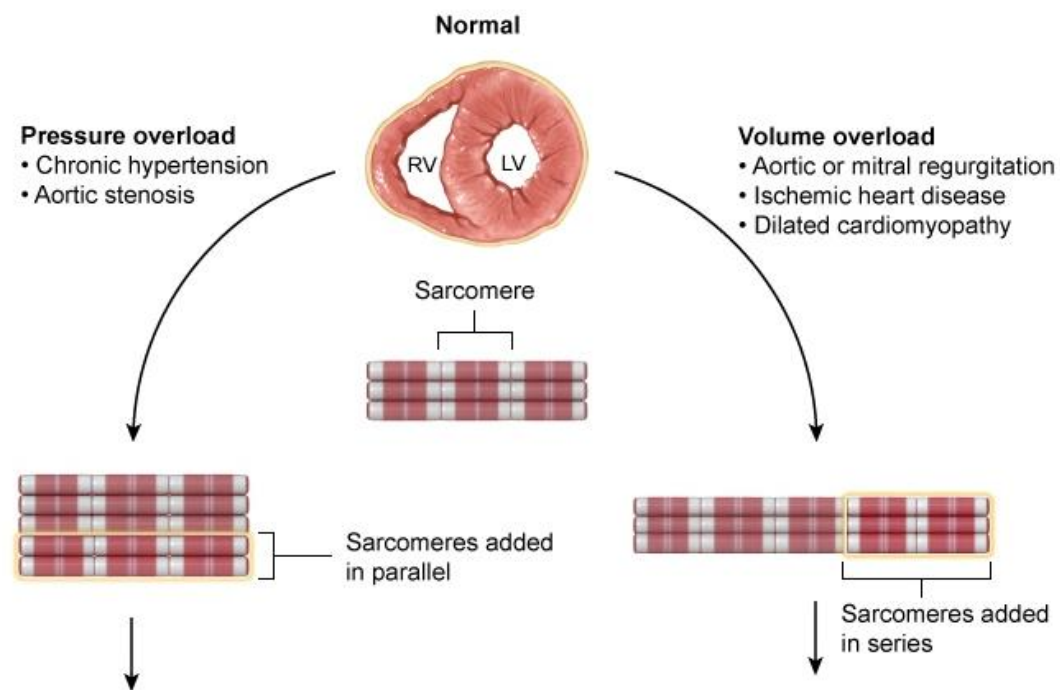
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2/7/2019



Explanation

Concentric & eccentric cardiac hypertrophy



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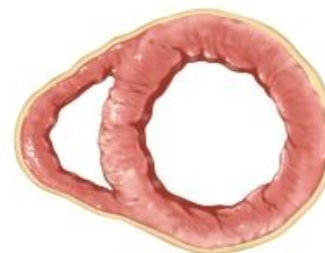
Sarcomeres added
in parallel



**Concentric left
ventricular hypertrophy**



Sarcomeres added
in series



**Eccentric left
ventricular hypertrophy**

LV = left ventricle; RV = right ventricle.
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This individual's autopsy reveals a **dilated left ventricular (LV) cavity** with relatively thin ventricular walls, a finding consistent with **eccentric hypertrophy**. Eccentric hypertrophy results from the addition of myocardial contractile fibers in series in response to chronic **volume overload**. The adaptation allows the left ventricle to increase stroke volume and maintain cardiac output; however, LV **wall stress increases**, resulting in eventual decompensation and the development of heart failure. Common causes of eccentric hypertrophy include cardiomyopathy, ischemic heart disease, and chronic aortic or mitral valve regurgitation.

In developed countries, **aortic root dilation** is one of the most common causes of **chronic aortic regurgitation**. This individual with an

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TUTOR





This individual's autopsy reveals a **dilated left ventricular (LV) cavity** with relatively thin ventricular walls, a finding consistent with **eccentric hypertrophy**. Eccentric hypertrophy results from the addition of myocardial contractile fibers in series in response to chronic **volume overload**. The adaptation allows the left ventricle to increase stroke volume and maintain cardiac output; however, LV **wall stress increases**, resulting in eventual decompensation and the development of heart failure. Common causes of eccentric hypertrophy include cardiomyopathy, ischemic heart disease, and chronic aortic or mitral valve regurgitation.

In developed countries, **aortic root dilation** is one of the most common causes of **chronic aortic regurgitation**. This individual with an ascending aortic aneurysm likely had aortic root dilation with consequent chronic aortic regurgitation, leading to LV volume overload and eccentric hypertrophy.

(Choice B) Aortic stenosis leads to pressure overload of the left ventricle and the development of concentric hypertrophy (thickening of the LV walls via the addition of myocardial contractile fibers in parallel). However, the ventricular cavity size is typically normal or reduced (not increased).

(Choice C) Hypertrophic cardiomyopathy is an inherited cardiomyopathy that involves localized thickening of the LV septal wall and resulting LV outflow tract obstruction. Although sudden cardiac death can occur (mostly in young athletes), the uniform thickness of this patient's ventricular walls and enlarged chamber size is more consistent with eccentric hypertrophy due to aortic regurgitation.

(Choice D) Obstructive coronary artery disease can lead to eccentric hypertrophy via myocardial contractile dysfunction and consequent LV volume overload (ie, ischemic heart disease). However, chronic aortic regurgitation is more likely in this individual with ascending aortic aneurysm and no evidence of myocardial scarring (scarring would suggest previous myocardial infarction).

(Choice E) Restrictive cardiomyopathy involves a decrease, rather than an increase, in the LV cavity size.

Educational objective:

Eccentric ventricular hypertrophy results in a dilated cavity with relatively thin ventricular walls due to the addition of myocardial contractile fibers in series in response to chronic volume overload. Chronic aortic regurgitation can result from aortic root dilation and is a common cause of eccentric hypertrophy.

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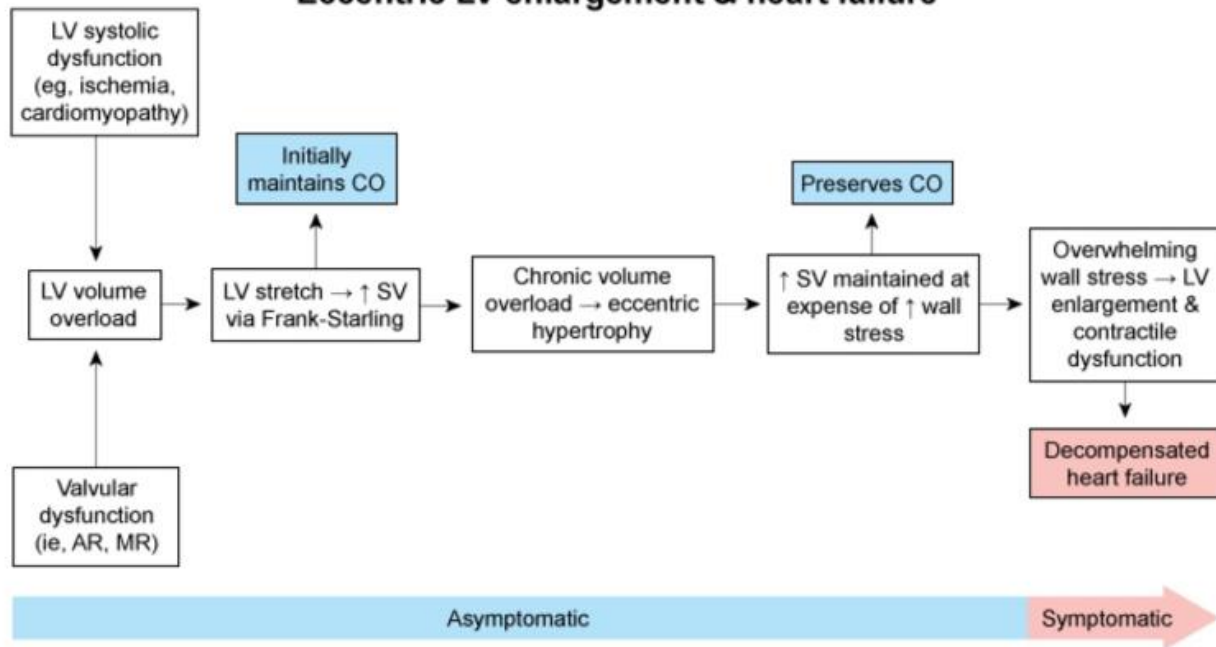




This individual's autopsy reveals a dilated left ventricular (LV) cavity with relatively thin ventricular walls, a finding consistent with eccentric

Exhibit Display

Eccentric LV enlargement & heart failure



AR = aortic regurgitation; CO = cardiac output; LV = left ventricular; MR = mitral regurgitation; SV = stroke volume.

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Text Zoom

A 6-year-old boy is evaluated in the office for difficulty hearing. The patient has no ear pain, discharge, or upper respiratory symptoms. Initial testing suggests that he has bilateral sensorineural hearing loss. He has no motor deficits or cerebellar signs. His paternal uncle died suddenly at age 12. Examination of the ears, nose, and throat is normal. ECG shows normal sinus rhythm with a prolonged QT interval (520 msec). Echocardiogram shows normal left and right ventricular function with no significant valvular disease. A genetic defect affecting which of the following is most likely present in this patient?

A. Calcium channels

B. Membrane anchoring protein

C. Potassium channels

D. Sodium channels

E. Sodium-potassium ATPase

Submit

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TUTOR

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Text Zoom

A 6-year-old boy is evaluated in the office for difficulty hearing. The patient has no ear pain, discharge, or upper respiratory symptoms. Initial testing suggests that he has bilateral sensorineural hearing loss. He has no motor deficits or cerebellar signs. His paternal uncle died suddenly at age 12. Examination of the ears, nose, and throat is normal. ECG shows normal sinus rhythm with a prolonged QT interval (520 msec). Echocardiogram shows normal left and right ventricular function with no significant valvular disease. A genetic defect affecting which of the following is most likely present in this patient?

☐

A. Calcium channels [8%]

☐

B. Membrane anchoring protein [5%]

☒

C. Potassium channels [70%]

☐

D. Sodium channels [8%]

☐

E. Sodium-potassium ATPase [6%]

Omitted

Correct answer
C

70%

Answered correctly

3 Seconds

Time Spent

01/13/2019

Last Updated

Explanation

Ventricular action potential

Slowed

Block Time Remaining: 00:01:14

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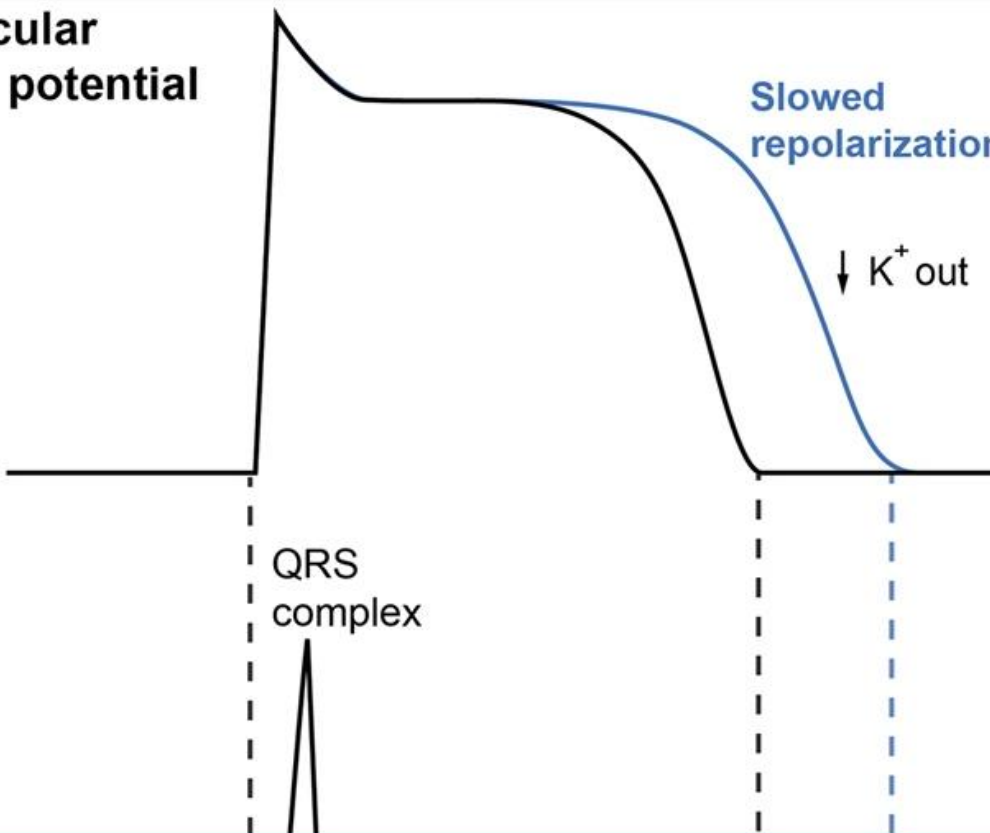
Calculator

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Text Zoom

Explanation

Ventricular action potential



ECG

QRS complex

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Calculator

Reverse Color

Text Zoom

ECG

P wave

QRS complex

T wave

Normal QT interval

Prolonged QT interval

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Block Time Remaining: 00:01:14

TUTOR

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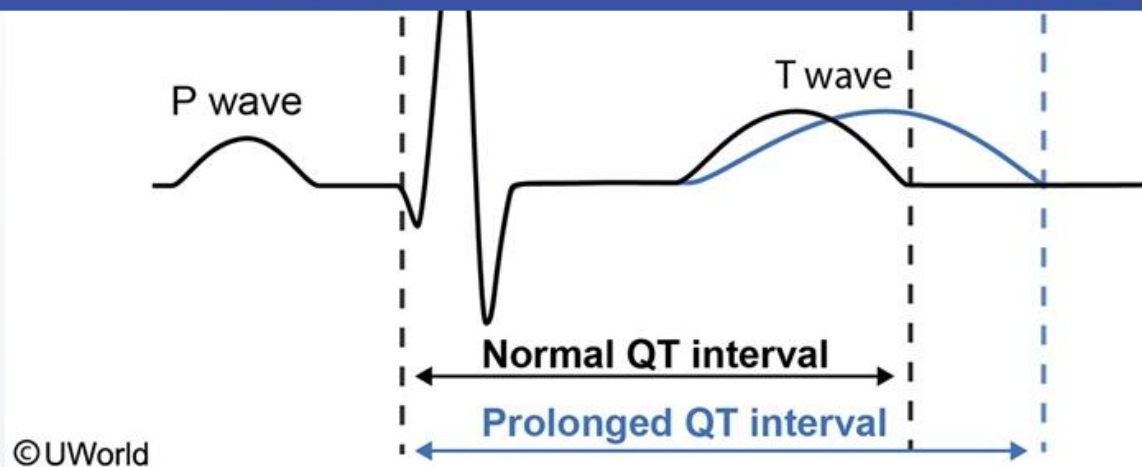
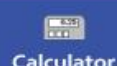
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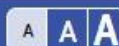


This patient's clinical presentation is suggestive of **Jervell and Lange-Nielsen syndrome**, an autosomal recessive disorder characterized by profound bilateral **sensorineural hearing loss** and **congenital long QT syndrome**, which predisposes individuals to syncope and sudden cardiac death.

This syndrome occurs secondary to **mutations** in genes (eg, KCNQ1, KCNE1) that encode the alpha and beta subunits of **voltage-gated potassium channels**. These subunits contribute to the slow-acting component of the outward-rectifying potassium current, which is responsible for ventricular repolarization during phase 3 of the **cardiac action potential**. Mutations in the potassium channel lead to a decrease in potassium current with prolongation of action potential duration and the QT interval. QT interval prolongation predisposes to the development of life-threatening **ventricular arrhythmias**, such as torsades de pointes and ventricular fibrillation.

(Choices A and D) Brugada syndrome is an autosomal dominant condition that can be associated with mutations in cardiac sodium or L-type calcium channels, leading to characteristic ECG changes (eg, pseudo right bundle branch block, ST-segment elevation in leads V1-V3) and an increased risk of ventricular tachyarrhythmias and sudden cardiac death.





potassium channels. These subunits contribute to the slow-acting component of the outward-rectifying potassium current, which is responsible for ventricular repolarization during phase 3 of the **cardiac action potential**. Mutations in the potassium channel lead to a decrease in potassium current with prolongation of action potential duration and the QT interval. QT interval prolongation predisposes to the development of life-threatening **ventricular arrhythmias**, such as torsades de pointes and ventricular fibrillation.

(Choices A and D) Brugada syndrome is an autosomal dominant condition that can be associated with mutations in cardiac sodium or L-type calcium channels, leading to characteristic ECG changes (eg, pseudo right bundle branch block, ST-segment elevation in leads V1-V3) and an increased risk of ventricular tachyarrhythmias and sudden cardiac death.

(Choice B) Duchenne muscular dystrophy is an X-linked disorder caused by mutations in dystrophin, a structural membrane protein that stabilizes the plasma membrane of myocytes. Patients typically present with progressive proximal muscle degeneration and weakness. Additional features may include cardiomyopathy and conduction abnormalities.

(Choice E) Genetic defects in the sodium-potassium ATPase pump typically are not associated with long QT syndrome. Cardiac glycosides (eg, digoxin) inhibit the sodium-potassium ATPase in myocardial cells, resulting in a mild increase in intracellular sodium and subsequent inhibition of the sodium-calcium exchanger. This leads to increased intracellular calcium and cardiac contractility, which can help reduce symptoms in patients with advanced systolic heart failure.

Educational objective:

Jervell and Lange-Nielsen syndrome is an autosomal recessive disorder characterized by profound bilateral sensorineural hearing loss and congenital long QT syndrome, which predisposes to ventricular arrhythmias and sudden cardiac death. This condition occurs secondary to mutations in genes that encode voltage-gated potassium channels.

References

- [The long QT syndromes: genetic basis and clinical implications.](#)
- [A spectrum of functional effects for disease causing mutations in the Jervell and Lange-Nielsen syndrome.](#)



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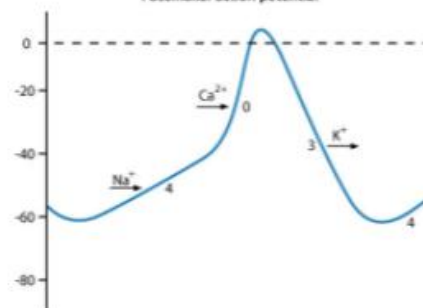
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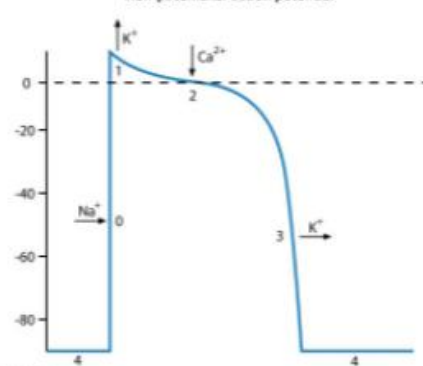
Exhibit Display

Cardiac action potentials

Pacemaker action potential



Non-pacemaker action potential



Zoom In

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Additional features may include cardiomyopathy and conduction abnormalities

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A 62-year-old man is brought to the emergency department by a friend due to severe shortness of breath. He is agitated and gasping for air and is unable to provide history. His friend says the patient has a history of alcohol abuse and attends Alcoholics Anonymous meetings. He is unaware of any other medical history. Temperature is 36.7 C (98 F), blood pressure is 110/70 mm Hg, pulse is 104/min, and respirations are 32/min. Due to worsening respiratory distress, immediate endotracheal intubation is performed and mechanical ventilation is begun. The patient is admitted to the intensive care unit and, despite appropriate therapy, dies several hours later. Autopsy examination is performed, and histopathology of the lungs reveals engorged pulmonary capillaries and intraalveolar, acellular, pink material that is more prominent at the bases. This patient most likely had which of the following conditions?

- ☐ A. Aspiration pneumonia
- ☐ B. Centriacinar emphysema
- ☐ C. Hypersensitivity pneumonitis
- ☐ D. Pulmonary arterial hypertension
- ☐ E. Pulmonary edema
- ☐ F. Pulmonary infarction

Submit

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TUTOR





Item 19 of 40

Question Id: 7616



A 62-year-old man is brought to the emergency department by a friend due to severe shortness of breath. He is agitated and gasping for air and is unable to provide history. His friend says the patient has a history of alcohol abuse and attends Alcoholics Anonymous meetings. He is unaware of any other medical history. Temperature is 36.7 C (98 F), blood pressure is 110/70 mm Hg, pulse is 104/min, and respirations are 32/min. Due to worsening respiratory distress, immediate endotracheal intubation is performed and mechanical ventilation is begun. The patient is admitted to the intensive care unit and, despite appropriate therapy, dies several hours later. Autopsy examination is performed, and histopathology of the lungs reveals engorged pulmonary capillaries and intraalveolar, acellular, pink material that is more prominent at the bases. This patient most likely had which of the following conditions?

- ☐ A. Aspiration pneumonia [20%]
- ☐ B. Centriacinar emphysema [2%]
- ☐ C. Hypersensitivity pneumonitis [5%]
- ☐ D. Pulmonary arterial hypertension [15%]
- ☒ E. Pulmonary edema [49%]
- ☐ F. Pulmonary infarction [5%]

Omitted

Correct answer
E49%
Answered correctly3 Seconds
Time Spent02/01/2019
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Explanation

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Explanation

Engorged alveolar capillaries reflect increased pulmonary venous pressure, and pink, acellular material within the alveoli results from transudation of fluid plasma across the alveolar-capillary membrane. These histopathologic findings are most consistent with **acute pulmonary edema** caused by **increased alveolar capillary hydrostatic pressure** from **left-sided heart failure**. This patient likely had **dilated cardiomyopathy** due to **chronic alcohol abuse** and had an acute exacerbation of heart failure, resulting in cardiogenic shock with pulmonary edema and acute respiratory failure.

(Choice A) Aspiration pneumonia may be chemical (eg, aspiration of gastric contents) or bacterial (eg, aspiration of oral flora). Histopathology shows neutrophil-rich, inflammatory exudates within the bronchioles and alveolar spaces.

(Choice B) Centriacinar emphysema most commonly results from heavy smoking. **Enlarged airspaces** with macrophage-predominant infiltrates are expected.

(Choice C) Hypersensitivity pneumonitis results from exposure to an environmental irritant (eg, mold). Histopathology shows lymphocyte-predominant, interstitial inflammation, sometimes accompanied by necrotizing granulomas and loose fibroelastic tissue within the small airways.

(Choice D) Histopathology in pulmonary arterial hypertension typically shows hypertrophy of the pulmonary vascular **smooth muscle**.

(Choice F) Pulmonary infarction yields hemorrhagic, ischemic necrosis of the lung parenchyma. Histopathology shows a well-demarcated area of alveolar and vascular wall necrosis with cells that lack nuclei, and the alveolar spaces may contain red blood cells.

Educational objective:

Acute cardiogenic pulmonary edema results from increased pulmonary venous pressure. The alveolar capillaries become engorged with blood and there is a transudation of fluid plasma across the alveolar-capillary membrane, appearing as pink, acellular material within the alveoli.

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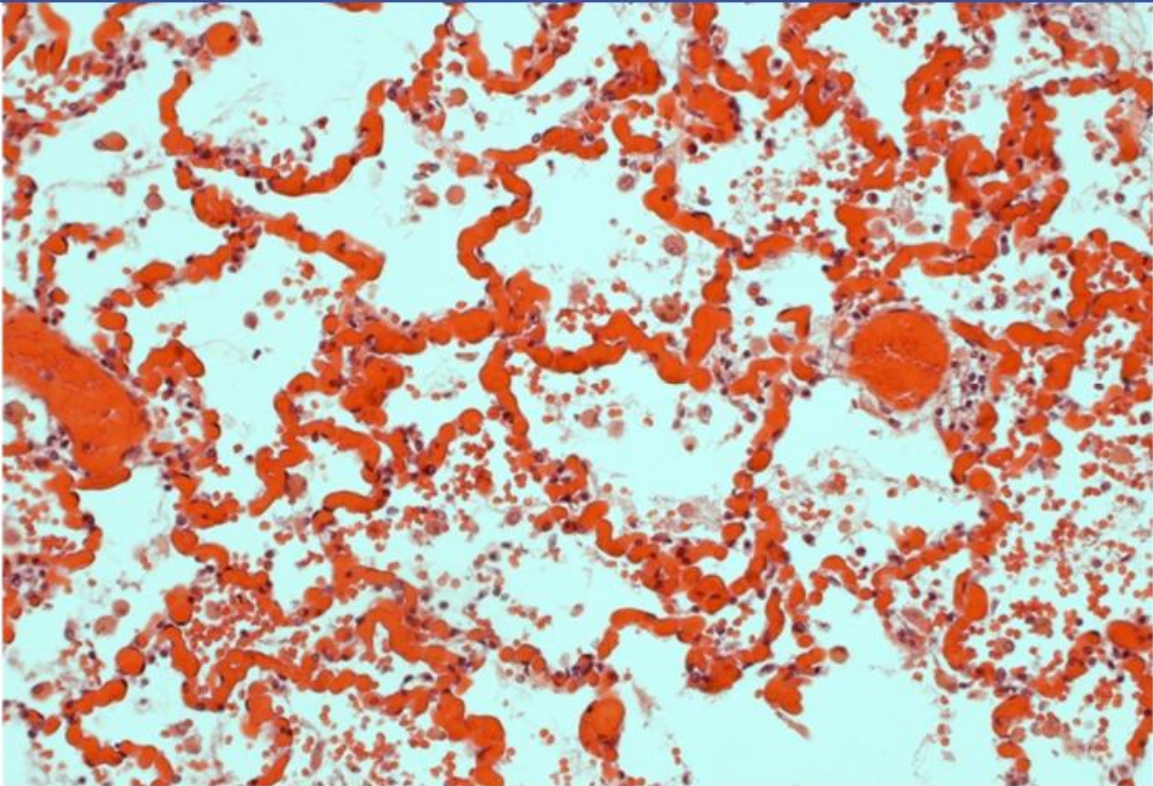
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Text Zoom

Exhibit Display

A microscopic image of tissue, likely a histological section, showing a complex network of red-stained structures (possibly blood vessels or connective tissue) and blue-stained areas (likely nuclei or other cellular components). The image is displayed within a window titled "Exhibit Display".

Zoom In

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TUTOR

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2/7/2019

A 22-year-old Caucasian male presents to the emergency room complaining of severe headaches and vomiting. Soon after, he slips into a coma and dies. Autopsy shows a ruptured cerebral aneurysm with extensive intracranial hemorrhage. This patient's condition is most likely associated with:

- ☐ A. Primum-type atrial septal defect
- ☐ B. Secundum-type atrial septal defect
- ☐ C. Ventricular septal defect
- ☐ D. Patent ductus arteriosus
- ☐ E. Coarctation of the aorta
- ☐ F. Tetralogy of Fallot

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Lab Values

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A 22-year-old Caucasian male presents to the emergency room complaining of severe headaches and vomiting. Soon after, he slips into a coma and dies. Autopsy shows a ruptured cerebral aneurysm with extensive intracranial hemorrhage. This patient's condition is most likely associated with:

☐

A. Primum-type atrial septal defect [7%]

☐

B. Secundum-type atrial septal defect [11%]

☐

C. Ventricular septal defect [12%]

☐

D. Patent ductus arteriosus [6%]

☒

E. Coarctation of the aorta [58%]

☐

F. Tetralogy of Fallot [4%]

Omitted

Correct answer
E

58%

Answered correctly

3 Seconds

Time Spent

10/04/2018

Last Updated

Explanation

Since there is no history of head trauma, this patient most likely suffered a spontaneous intracranial hemorrhage (SICH). The most common causes of SICH in young adults are arteriovenous malformations, ruptured cerebral aneurysms, or abuse of sympathomimetic drugs such as cocaine.

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Correct answer

Answered correctly

Time Spent

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E

Explanation

Since there is no history of head trauma, this patient most likely suffered a spontaneous intracranial hemorrhage (SICH). The most common causes of SICH in young adults are arteriovenous malformations, ruptured cerebral aneurysms, or abuse of sympathomimetic drugs such as cocaine.

We are told that this patient had a ruptured cerebral aneurysm, and are asked to identify an associated congenital cardiac anomaly. Although coarctation of the aorta may occur as a solitary defect, it may also be associated with other congenital cardiac anomalies or with berry aneurysms of the Circle of Willis. Berry aneurysms are particularly prone to rupture when associated with coarctation, because of hypertension in branches of the aortic arch proximal to the coarct. Subarachnoid hemorrhage is the result.

(Choices A, B, and C) Isolated atrial or ventricular septal defects are not commonly associated with spontaneous rupture of intracranial aneurysms in young adults. The incidence of intracerebral aneurysms is not increased in patients with isolated septal defects, nor are septal defects associated with severe systemic hypertension (though they may cause severe pulmonary hypertension via left-to-right intracardiac shunt flow). However, the incidence of *thromboembolic stroke* as the result of paradoxical embolism might be increased in patients with an atrial or ventricular septal defect and late-onset right-to-left shunting.

Educational Objective:

Patients with adult-type coarctation of the aorta commonly die of hypertension-associated complications, including left ventricular failure, ruptured dissecting aortic aneurysm, and intracranial hemorrhage. These patients are at increased risk for ruptured intracranial aneurysms because of the increased incidence of congenital berry aneurysms of the Circle of Willis as well as aortic arch hypertension.

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Text Zoom

A 24-year-old woman comes to the physician with a 3-week history of progressive fatigue. Physical examination shows lesions involving the nail beds, as seen in the photograph below.

Further evaluation would most likely reveal additional abnormalities during which part of the physical examination?

☐ A. Neck palpation

☐ B. Lung auscultation

☐ C. Cardiac auscultation

☐ D. Abdominal palpation

☐ E. Rectal examination

Submit

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beds, as seen in the photograph below.



Further evaluation would most likely reveal additional abnormalities during which part of the physical examination?

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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Store, Mail, Calendar, Photos, and other background applications.

System tray showing date and time: 2:49 PM 2/9/2019.

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Text Zoom

A 24-year-old woman comes to the physician with a 3-week history of progressive fatigue. Physical examination shows lesions involving the nail beds, as seen in the photograph below.

Further evaluation would most likely reveal additional abnormalities during which part of the physical examination?

☐ A. Neck palpation

☐ B. Lung auscultation

☐ C. Cardiac auscultation

☐ D. Abdominal palpation

☐ E. Rectal examination

Submit

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System Tray

Further evaluation would most likely reveal additional abnormalities during which part of the physical examination?

- Omitted
 Correct answer
 C

 89%
 Answered correctly

 2 Seconds
 Time Spent

 10/14/2018
 Last Updated

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Omitted

Correct answer
C

89%

Answered correctly

2 Seconds

Time Spent

10/14/2018

Last Updated

Explanation

These lesions are subungual splinter hemorrhages, splinter- or flame-shaped hemorrhagic streaks in the nail bed that appear as a consequence of microemboli. Janeway lesions (small, macular, erythematous or hemorrhagic, nontender lesions on the palms and soles) are also a sign of microembolism. Infectious endocarditis (IE) is the usual cause of such microemboli, and progressive fatigue may be the presenting symptom of subacute bacterial endocarditis. Chronically destructive, infected vegetations involving the cardiac valves are the source of the microemboli in bacterial endocarditis. Patients with suspected IE should therefore be evaluated for a regurgitant murmur during cardiac auscultation.

(Choices A, B, and D) Unless the primary source of the bacteremia initiating the IE is a focal neck, pulmonary, or abdominal infection, one would not expect to find any abnormalities in these systems.

(Choice E) Unless the primary source of the original bacteremia was a peri-rectal infection or necrotic, infected rectal neoplasm, one would not expect to find any abnormalities on rectal examination.

Educational objective:

Microemboli from the valvular vegetations of bacterial endocarditis are the most common cause of subungual splinter hemorrhages. The presence of these lesions necessitates careful cardiac auscultation to detect a possible new-onset regurgitant murmur.

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Text Zoom

A 54-year-old man comes to the emergency department due to severe shortness of breath and palpitations. During initial assessment, pulse oximetry shows 83%. Diffuse, bilateral crackles are heard on lung auscultation. Chest x-ray is consistent with pulmonary edema. ECG shows atrial fibrillation with rapid ventricular response. The patient dies despite appropriate medical care. Autopsy is performed and gross examination of the heart is shown below.

Histologic examination of the myocardial tissue reveals thick myocardial fibers with large, hyperchromatic nuclei as well as mild interstitial fibrosis. Which of the following is the most likely diagnosis?

A. Cardiac amyloidosis

B. Hypertensive heart disease

C. Hypertrophic cardiomyopathy

D. Mitral stenosis

E. Pulmonary arterial hypertension

Submit

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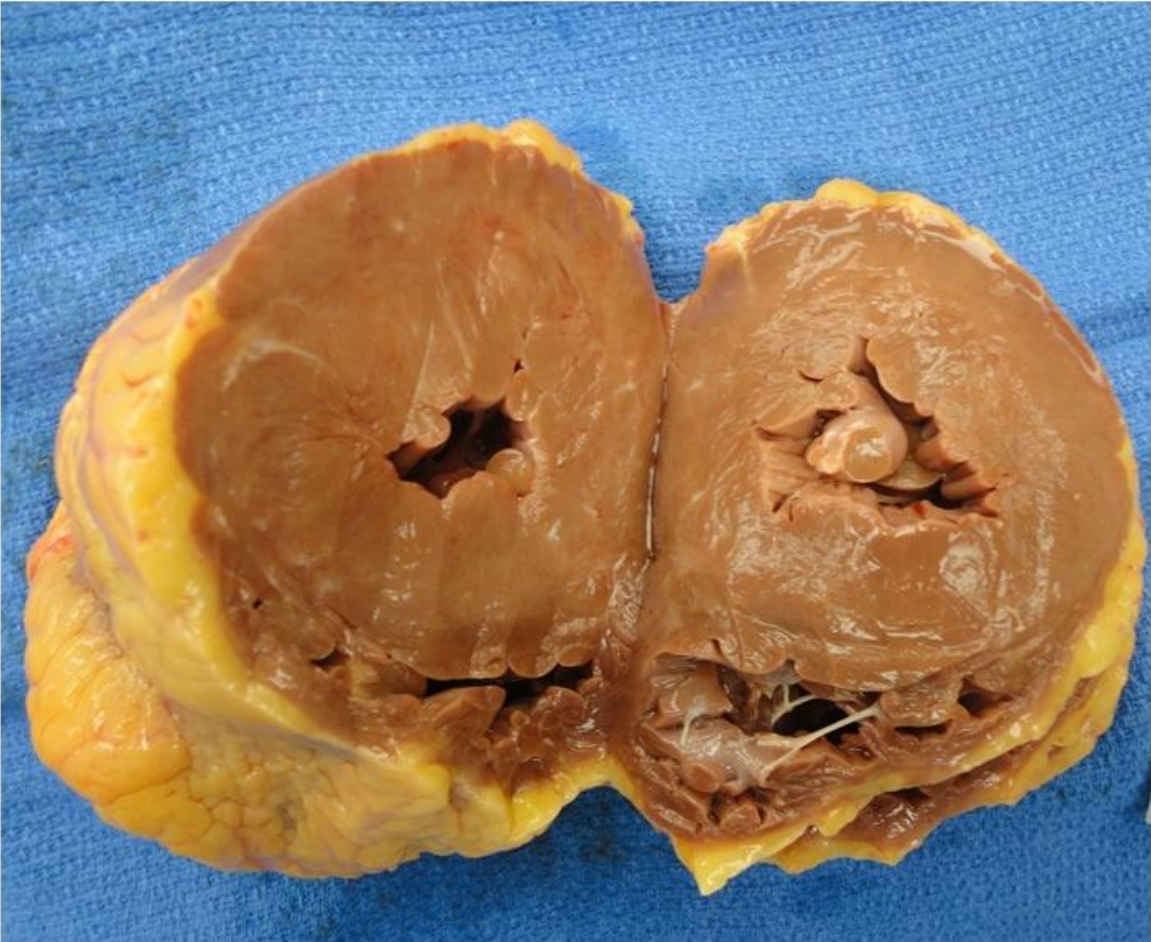
Lab Values

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A 54-year-old man comes to the emergency department due to severe shortness of breath and palpitations. During initial assessment, pulse oximetry shows 83%. Diffuse, bilateral crackles are heard on lung auscultation. Chest x-ray is consistent with pulmonary edema. ECG shows atrial fibrillation with rapid ventricular response. The patient dies despite appropriate medical care. Autopsy is performed and gross examination of the heart is shown below.

Histologic examination of the myocardial tissue reveals thick myocardial fibers with large, hyperchromatic nuclei as well as mild interstitial fibrosis. Which of the following is the most likely diagnosis?

- ☐ A. Cardiac amyloidosis
- ☐ B. Hypertensive heart disease
- ☐ C. Hypertrophic cardiomyopathy
- ☐ D. Mitral stenosis
- ☐ E. Pulmonary arterial hypertension

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A 54-year-old man comes to the emergency department due to severe shortness of breath and palpitations. During initial assessment, pulse oximetry shows 83%. Diffuse, bilateral crackles are heard on lung auscultation. Chest x-ray is consistent with pulmonary edema. ECG shows atrial fibrillation with rapid ventricular response. The patient dies despite appropriate medical care. Autopsy is performed and gross examination of the heart is shown below.

Histologic examination of the myocardial tissue reveals thick myocardial fibers with large, hyperchromatic nuclei as well as mild interstitial fibrosis. Which of the following is the most likely diagnosis?

☐ A. Cardiac amyloidosis [10%]

☒ B. Hypertensive heart disease [45%]

☐ C. Hypertrophic cardiomyopathy [35%]

☐ D. Mitral stenosis [4%]

☐ E. Pulmonary arterial hypertension [5%]

Omitted

Correct answer B

45% Answered correctly

2 Seconds Time Spent

02/05/2019 Last Updated

Explanation

This individual developed acute decompensated heart failure (eg, shortness of breath, pulmonary edema) and likely died due to subsequent

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This individual developed acute decompensated heart failure (eg, shortness of breath, pulmonary edema) and likely died due to subsequent cardiogenic shock. Autopsy findings reveal **uniformly thickened** left ventricular (LV) walls and a small LV cavity, which are consistent with concentric LV hypertrophy.

Concentric LV hypertrophy most commonly results from prolonged **systemic hypertension** as myocardial fibers are added in parallel to increase contractile strength and handle the increased pressure load. As concentric hypertrophy worsens, it can manifest clinically as **hypertensive heart disease**, consisting of **impaired diastolic filling** and heart failure with preserved ejection fraction. The elevated diastolic filling pressures are transmitted back to the left atrium, causing atrial dilation and altered conduction that predisposes to **atrial fibrillation**. Loss of atrial contraction can lead to a dramatic **reduction in left ventricular filling** in patients with diastolic heart failure, and can precipitate **acute decompensation** (as likely occurred in this patient).

On histopathology, hypertensive heart disease demonstrates transverse thickening of cardiomyocytes with prominent hyperchromatic nuclei. The increased myocardial mass may be inadequately supplied with oxygen, contributing to cellular necrosis and interstitial fibrosis.

(Choice A) Cardiac amyloidosis involves deposition of abnormal proteins in the myocardium and can lead to restrictive cardiomyopathy, which is characterized by impaired diastolic filling due to stiffening, rather than thickening, of the ventricular walls. Histopathology in **cardiac amyloidosis** reveals collections of pink amorphous material abutting normal cardiomyocytes.

(Choice C) Hypertrophic cardiomyopathy involves localized, rather than uniform, thickening of the LV walls, predominantly affecting the interventricular septum. As in hypertensive heart disease, histopathology can reveal cardiomyocyte thickening, hyperchromatic nuclei, and interstitial fibrosis; however, **cardiomyocyte disarray** (loss of organized cellular alignment) is also expected.

(Choice D) Mitral stenosis causes impaired diastolic filling and can lead to decompensated heart failure and atrial fibrillation; however, it does not increase the pressure load on the left ventricle and does not cause concentric hypertrophy.

(Choice E) Pulmonary arterial hypertension results from primary changes in the pulmonary arteries that increase pulmonary arterial resistance. The elevated pressure load on the right ventricle can lead to concentric right ventricular hypertrophy and right-sided heart failure. Pulmonary

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atrial contraction can lead to a dramatic **reduction in left ventricular filling** in patients with diastolic heart failure, and can precipitate **acute decompensation** (as likely occurred in this patient).

On histopathology, hypertensive heart disease demonstrates transverse thickening of cardiomyocytes with prominent hyperchromatic nuclei. The increased myocardial mass may be inadequately supplied with oxygen, contributing to cellular necrosis and interstitial fibrosis.

(Choice A) Cardiac amyloidosis involves deposition of abnormal proteins in the myocardium and can lead to restrictive cardiomyopathy, which is characterized by impaired diastolic filling due to stiffening, rather than thickening, of the ventricular walls. Histopathology in **cardiac amyloidosis** reveals collections of pink amorphous material abutting normal cardiomyocytes.

(Choice C) Hypertrophic cardiomyopathy involves localized, rather than uniform, thickening of the LV walls, predominantly affecting the interventricular septum. As in hypertensive heart disease, histopathology can reveal cardiomyocyte thickening, hyperchromatic nuclei, and interstitial fibrosis; however, **cardiomyocyte disarray** (loss of organized cellular alignment) is also expected.

(Choice D) Mitral stenosis causes impaired diastolic filling and can lead to decompensated heart failure and atrial fibrillation; however, it does not increase the pressure load on the left ventricle and does not cause concentric hypertrophy.

(Choice E) Pulmonary arterial hypertension results from primary changes in the pulmonary arteries that increase pulmonary arterial resistance. The elevated pressure load on the right ventricle can lead to concentric right ventricular hypertrophy and right-sided heart failure. Pulmonary edema is not present in right-sided heart failure.

Educational objective:

Concentric left ventricular (LV) hypertrophy involves uniform thickening of the LV walls with reduction in LV cavity size and most commonly results from prolonged systemic hypertension. It can progress to hypertensive heart disease with impaired diastolic filling and heart failure with preserved ejection fraction. Histopathology demonstrates transverse thickening of cardiomyocytes with prominent hyperchromatic nuclei and interstitial fibrosis.

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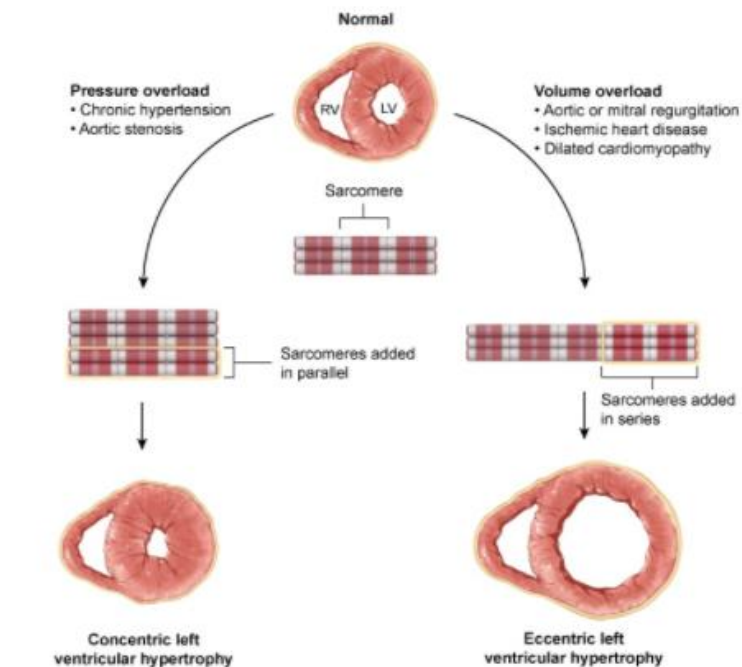
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02/05/2019

Exhibit Display

Concentric & eccentric cardiac hypertrophy

LV = left ventricle; RV = right ventricle.
(© 2010 MedNet)

Zoom In

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Tutorial

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A 37-year-old man is found unresponsive under a tree during a thunderstorm. He is not breathing when paramedics arrive on the scene. On examination, his pupils are fixed and dilated. Several cutaneous erythematous marks in a fern-leaf pattern are seen on his lower extremities. Second-degree burns are present on both arms. Cardiopulmonary resuscitation is started; however, the patient does not respond and is pronounced dead on arrival at the hospital. Which of the following is most likely to be the primary cause of his death?

A. Cardiac arrhythmia

B. Extensive deep-tissue burns

C. Intracranial hemorrhage

D. Myocardial contusion

E. Pulmonary barotrauma

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Text Zoom

Settings

A 37-year-old man is found unresponsive under a tree during a thunderstorm. He is not breathing when paramedics arrive on the scene. On examination, his pupils are fixed and dilated. Several cutaneous erythematous marks in a fern-leaf pattern are seen on his lower extremities. Second-degree burns are present on both arms. Cardiopulmonary resuscitation is started; however, the patient does not respond and is pronounced dead on arrival at the hospital. Which of the following is most likely to be the primary cause of his death?

✓

☒

A. Cardiac arrhythmia [83%]

☐

B. Extensive deep-tissue burns [5%]

☐

C. Intracranial hemorrhage [6%]

☐

D. Myocardial contusion [2%]

☐

E. Pulmonary barotrauma [1%]

Omitted

Correct answer
A

83%

Answered correctly

3 Seconds

Time Spent

09/05/2018

Last Updated

Explanation

The finding of an unconscious person on wet ground or under trees during a thunderstorm suggests a lightning injury. This patient's Lichtenberg figures (erythematous cutaneous marks in a fern-leaf pattern) and second-degree burns confirm the suspicion. Although lightning injuries are rare, they are associated with a 25% fatality rate, and more than 70% of survivors have long-lasting complications. Two-thirds of lightning-related

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Text Zoom

Explanation

The finding of an unconscious person on wet ground or under trees during a thunderstorm suggests a lightning injury. This patient's Lichtenberg figures (erythematous cutaneous marks in a fern-leaf pattern) and second-degree burns confirm the suspicion. Although lightning injuries are rare, they are associated with a 25% fatality rate, and more than 70% of survivors have long-lasting complications. Two-thirds of lightning-related deaths occur within the first hour following injury; the most common causes are fatal arrhythmias and respiratory failure. A comprehensive and thorough physical examination should always be performed on lightning strike victims as injury patterns are widely variable and skin findings can significantly underestimate the extent of internal injury.

(Choice B) Superficial burns are common in lightning injury. In contrast, deep-tissue burns are relatively rare due to the short duration of electrical contact and the "flashover effect," the tendency of lightning to travel over the skin surface and discharge to the ground. This effect is responsible for the formation of Lichtenberg figures.

(Choice C) Intracranial hemorrhage only rarely occurs following lightning injury. Lightning strikes can damage the autonomic system and cause fixed and dilated or asymmetric pupils. These signs may not accurately reflect the extent of neurologic injury and should not be a reason to stop resuscitation.

(Choices D and E) Lightning induces rapid heating of the surrounding air, generating shock waves that can travel through the body and cause mechanical trauma. As a result, patients should be thoroughly assessed for skeletal fractures. Myocardial contusion and pulmonary barotrauma occur less frequently and would be extremely rare causes of death.

Educational objective:

Although lightning injuries are rare, they are associated with a 25% fatality rate. Two-thirds of lightning-related deaths occur within the first hour after injury, with fatal arrhythmias and respiratory failure as the most common causes. Patients with minor cutaneous involvement may still have major internal injury after lightning strikes and high-voltage electrical contact.

References

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Text Zoom

A 59-year-old African American male presents to the emergency room with crushing chest pain, sweating, and lightheadedness. His blood pressure is 90/60 mm Hg and his heart rate is 48 beats per minute. Electrocardiogram (ECG) shows sinus bradycardia and ST segment elevation in leads II, III, and aVF. Occlusion of which of the following coronary arteries is most likely responsible for this patient's symptoms?

A. Left main coronary artery

B. Left anterior descending artery

C. Left circumflex artery

D. Right coronary artery

E. Intramural arteries

Submit

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System Tray



A 59-year-old African American male presents to the emergency room with crushing chest pain, sweating, and lightheadedness. His blood pressure is 90/60 mm Hg and his heart rate is 48 beats per minute. Electrocardiogram (ECG) shows sinus bradycardia and ST segment elevation in leads II, III, and aVF. Occlusion of which of the following coronary arteries is most likely responsible for this patient's symptoms?

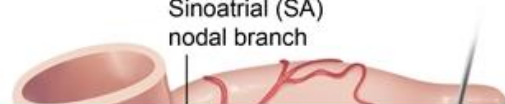
- ☐ A. Left main coronary artery [2%]
- ☐ B. Left anterior descending artery [35%]
- ☐ C. Left circumflex artery [5%]
- ☒ D. Right coronary artery [55%]
- ☐ E. Intramural arteries [1%]

Omitted

Correct answer
D55%
Answered correctly7 Seconds
Time Spent10/17/2018
Last Updated

Explanation

Coronary arteries: anterior view

Sinoatrial (SA)
nodal branch

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Coronary arteries: anterior view

Sinoatrial (SA) nodal branch

Atrial branch of right coronary artery

Right coronary artery

Right marginal branch of right coronary artery

Left coronary artery

Circumflex branch of left coronary artery

Anterior interventricular branch (anterior descending) of left coronary artery

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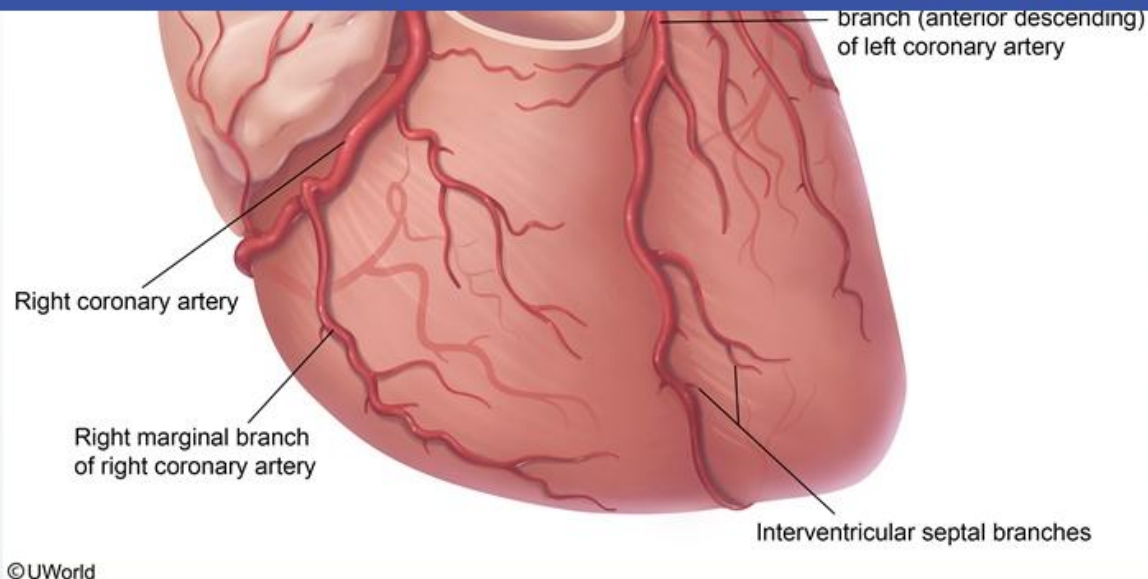
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2/7/2019



[Click here for posterior view.](#)

This patient presents with symptoms and ECG findings consistent with transmural ischemic injury to the inferior wall of the heart. In 90% of individuals, the posteroinferior wall of the left ventricle is supplied by the posterior descending branch of the right coronary artery (RCA). The patient's bradycardia and resultant hypotension suggests that there may have been ischemic injury to the sinus node as well. (The sinus node normally receives its arterial blood supply from the RCA.)

(Choices A, B, and C) The left main coronary artery gives rise to the left anterior descending (LAD) and the left circumflex (LCX) coronary arteries. The LAD supplies the interventricular septum and the anterior wall of the left ventricle. Transmural ischemia of the septum would produce ST elevation mainly in leads V1 and V2. Infranodal (Mobitz type II) second-degree or third-degree heart block is a possible result as well; but sinus bradycardia would not occur with this type of injury. Transmural ischemia of the anterior left ventricular wall would produce ST elevations

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This patient presents with symptoms and ECG findings consistent with transmural ischemic injury to the inferior wall of the heart. In 90% of individuals, the posteroinferior wall of the left ventricle is supplied by the posterior descending branch of the right coronary artery (RCA). The patient's bradycardia and resultant hypotension suggests that there may have been ischemic injury to the sinus node as well. (The sinus node normally receives its arterial blood supply from the RCA.)

(Choices A, B, and C) The left main coronary artery gives rise to the left anterior descending (LAD) and the left circumflex (LCX) coronary arteries. The LAD supplies the interventricular septum and the anterior wall of the left ventricle. Transmural ischemia of the septum would produce ST elevation mainly in leads V1 and V2. Infranodal (Mobitz type II) second-degree or third-degree heart block is a possible result as well; but sinus bradycardia would not occur with this type of injury. Transmural ischemia of the anterior left ventricular wall would produce ST elevations mainly in leads V3 and V4. An occlusion of the proximal LAD would cause anteroseptal transmural ischemia, with ST elevations in leads V1–V4.

The LCX supplies the lateral wall of the left ventricle. Transmural ischemia secondary to LCX occlusion would produce ST elevations mainly in leads V5 and V6, and possibly also in leads I and aVL. An occlusion of the left main coronary artery would therefore be expected to produce widespread transmural ischemia of the interventricular septum and left ventricle, with ST elevations in all of the chest (V) leads, and possibly also in leads I and aVL.

(Choice E) Intramural arteries in the substance of the myocardium are unlikely sites of occlusion because, unlike the epicardial coronary arteries, they are rarely affected by atherosclerosis. Moreover, if occlusion of intramural arteries did occur, subendocardial ischemia (with associated ST depression) would be more likely than transmural ischemia (with ST elevation).

Educational Objective:

In 90% of individuals, occlusion of the right coronary artery can result in transmural ischemia of the inferior wall of the left ventricle, producing ST elevation in leads II, III, and aVF as well as possible sinus node dysfunction. Occlusion of the proximal LAD would be expected to result in anteroseptal transmural ischemia, with ST elevations in leads V1–V4. Occlusion of the LCX would produce transmural ischemia of the lateral wall of the left ventricle, with ST elevations mainly in V5 and V6, and possibly also in I and aVL.

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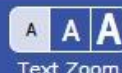
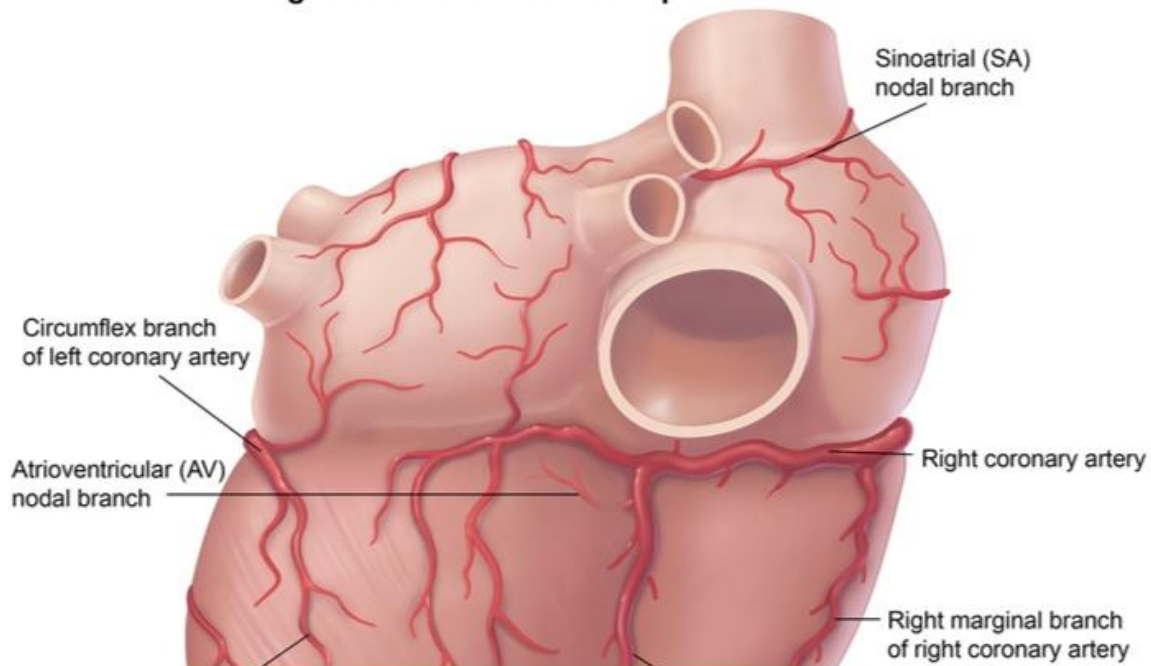


Exhibit Display

Right dominant circulation: posterior view



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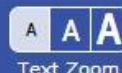


Exhibit Display

Circumflex branch
of left coronary arteryAtrioventricular (AV)
nodal branchPosterior left
ventricular branch

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Right coronary artery

Right marginal branch
of right coronary arteryPosterior interventricular branch
(posterior descending) of right
coronary artery

Interventricular septal branches

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Text Zoom

A 38-year-old man dies suddenly at his workplace and cannot be resuscitated. He had experienced chest discomfort, shortness of breath, and fatigue in the week preceding the event. The man was overweight but otherwise had no significant medical history. He had no established primary care physician. He did not use alcohol, tobacco, or illicit drugs. Both parents are alive and healthy; a maternal uncle reportedly died of a heart attack at age 50. Autopsy was performed and myocardial findings are shown in the slide below.

Which of the following is the most likely diagnosis?

A. Acute myocarditis

B. Cardiac amyloidosis

C. Congenital long QT syndrome

D. Early-onset coronary artery disease

E. Hypertrophic cardiomyopathy

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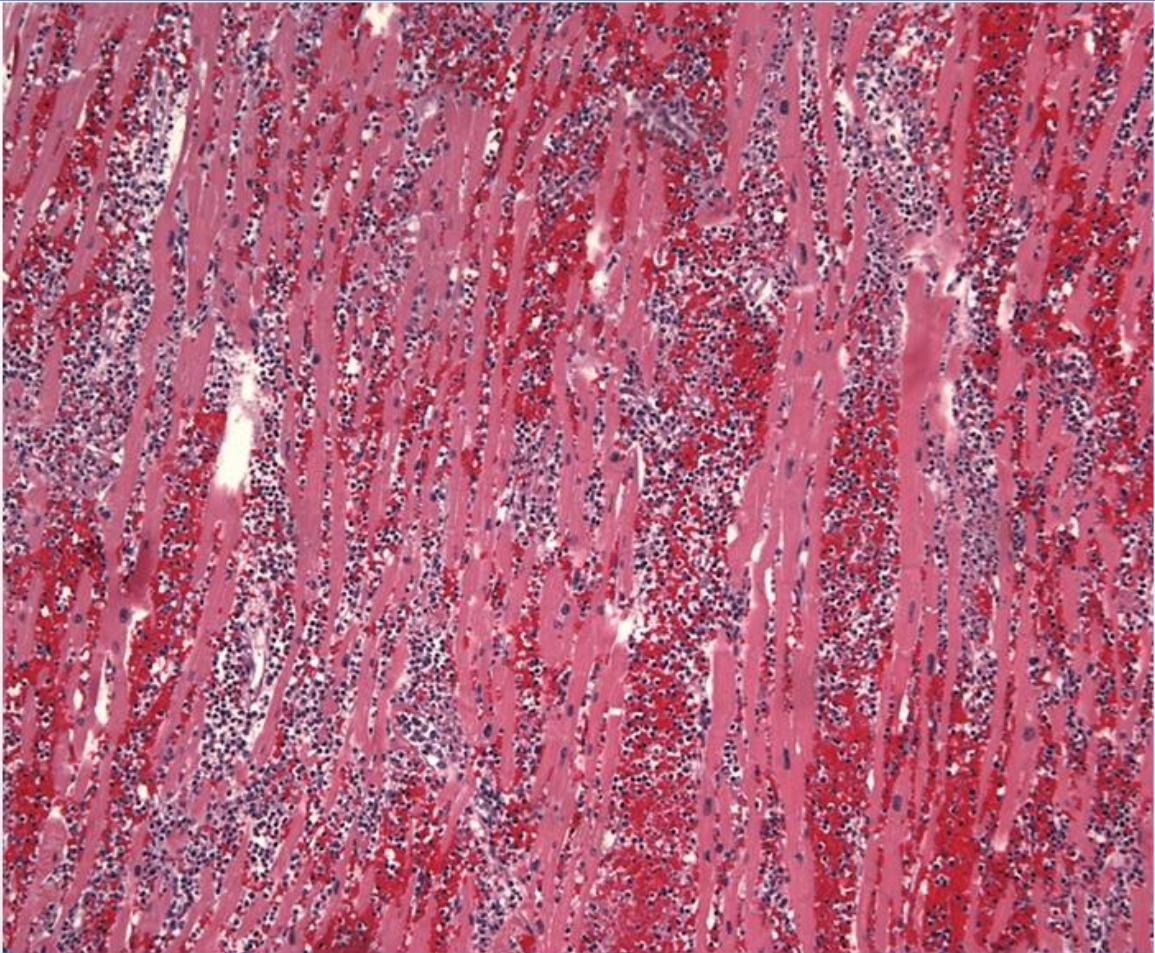
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Block Time Remaining: 00:03:05

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Windows taskbar with icons for File Explorer, Edge, Mail, and others.

System tray showing time 2:49 PM, date 2/9/2019, and network status.

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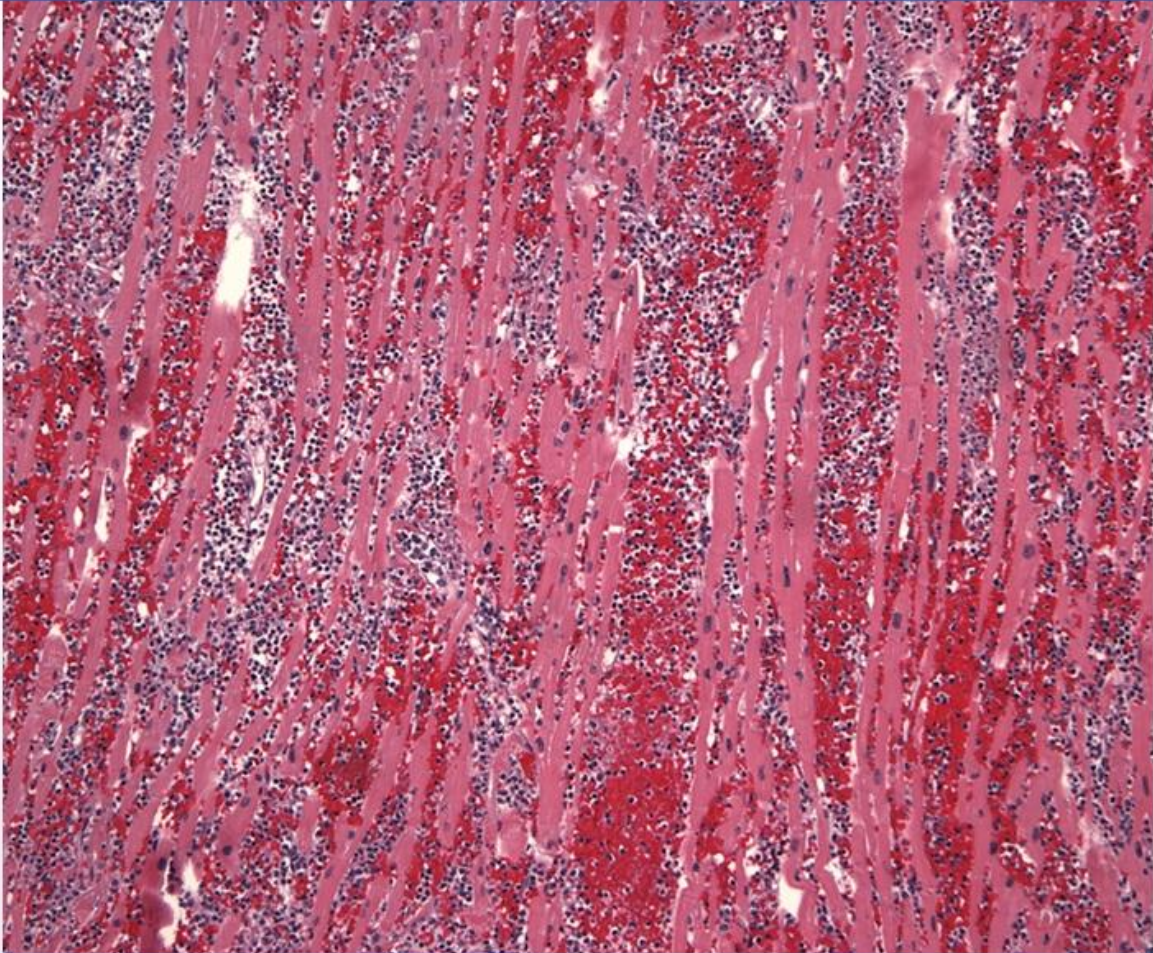
Lab Values

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End Block

Windows taskbar with icons for File Explorer, Edge, and other applications.

System tray showing time 2:49 PM, date 2/9/2019, and network status.

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A 38-year-old man dies suddenly at his workplace and cannot be resuscitated. He had experienced chest discomfort, shortness of breath, and fatigue in the week preceding the event. The man was overweight but otherwise had no significant medical history. He had no established primary care physician. He did not use alcohol, tobacco, or illicit drugs. Both parents are alive and healthy; a maternal uncle reportedly died of a heart attack at age 50. Autopsy was performed and myocardial findings are shown in the slide below.

Which of the following is the most likely diagnosis?

☐ A. Acute myocarditis

☐ B. Cardiac amyloidosis

☐ C. Congenital long QT syndrome

☐ D. Early-onset coronary artery disease

☐ E. Hypertrophic cardiomyopathy

Submit

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Windows Taskbar

System Tray



A 38-year-old man dies suddenly at his workplace and cannot be resuscitated. He had experienced chest discomfort, shortness of breath, and fatigue in the week preceding the event. The man was overweight but otherwise had no significant medical history. He had no established primary care physician. He did not use alcohol, tobacco, or illicit drugs. Both parents are alive and healthy; a maternal uncle reportedly died of a heart attack at age 50. Autopsy was performed and myocardial findings are shown in the slide below.

Which of the following is the most likely diagnosis?

- ☒ A. Acute myocarditis [60%]
☐ B. Cardiac amyloidosis [11%]
☐ C. Congenital long QT syndrome [3%]
☐ D. Early-onset coronary artery disease [9%]
☐ E. Hypertrophic cardiomyopathy [14%]

Omitted

Correct answer
A

60%
Answered correctly

2 Seconds
Time Spent

01/10/2019
Last Updated

Explanation

This patient likely died from **acute myocarditis**, a condition most often caused by **viral infection**. Numerous viruses, including coxsackievirus, adenovirus, and influenza, have been implicated. Bacterial (eg, tuberculosis) or parasitic (eg, Chagas disease) infection and rheumatologic

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Text Zoom

This patient likely died from **acute myocarditis**, a condition most often caused by **viral infection**. Numerous viruses, including coxsackievirus, adenovirus, and influenza, have been implicated. Bacterial (eg, tuberculosis) or parasitic (eg, Chagas disease) infection and rheumatologic disease (eg, systemic lupus erythematosus) are less common causes. Histopathology typically shows **myofibrillary necrosis** accompanied by inflammatory **mononuclear cell infiltrate** consisting of lymphocytes and macrophages.

The clinical presentation of acute myocarditis can vary significantly; many patients remain asymptomatic with subclinical infection, but some experience severe complications including decompensated **heart failure** (eg, dyspnea, fatigue, peripheral edema) due to **dilated cardiomyopathy**. Ventricular arrhythmias leading to **sudden cardiac death** (SCD) can also occur.

(Choice B) Cardiac amyloidosis involves deposition of abnormal proteins in myocardial tissue and typically manifests as restrictive cardiomyopathy initially. SCD can occur, but histopathology demonstrates pink amorphous material (amyloid) surrounding normal-appearing cardiomyocytes.

(Choice C) Congenital long QT syndrome can trigger ventricular arrhythmias and lead to SCD; however, histopathology is generally unremarkable and would not demonstrate myocardial necrosis with inflammatory infiltrate.

(Choice D) Coronary artery disease can lead to SCD due to **acute myocardial infarction** or ventricular arrhythmia triggered by chronic **ischemic heart disease**. However, this patient's relatively young age and 1 week of preceding exertional symptoms make acute myocarditis a more likely diagnosis.

(Choice E) Hypertrophic cardiomyopathy is characterized by cardiac myofibrillary disarray and interstitial fibrosis that can predispose to ventricular arrhythmia and SCD. An inflammatory infiltrate is not expected.

Educational objective:

Acute myocarditis is most commonly caused by a viral infection (eg, coxsackievirus, adenovirus, influenza). It often resolves without noticeable symptoms, but patients can develop serious complications, including decompensated heart failure due to dilated cardiomyopathy or sudden cardiac death due to ventricular arrhythmia. Histopathology typically demonstrates myofibrillary necrosis with inflammatory mononuclear infiltrate.

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A 60-year-old man comes to the emergency department with acute substernal chest pain, nausea, and diaphoresis. The patient has a history of liver cirrhosis, hypertension, and hyperlipidemia. He quit smoking 3 months ago but previously smoked a pack of cigarettes daily for 25 years. ECG shows ST elevation in leads II, III, and aVF. He is diagnosed with a myocardial infarction and, after careful discussion of risks and benefits, reperfusion therapy is not performed due to underlying cirrhosis and history of variceal bleeding. The patient is eventually discharged from the hospital with conservative management. Twelve days after his myocardial infarction, he dies suddenly. Light microscopy would most likely show which of the following changes in the myocardium of the inferior wall?

☐

A. Dense collagen scar

☐

B. Dense interstitial neutrophil infiltrate

☐

C. Edema and wavy myocyte fibers

☐

D. Granulation tissue with neovascularization

☐

E. Hypereosinophilic myocytes

☐

F. Macrophage infiltration with foam cell formation

☐

G. Normal myocardium

Submit

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2/7/2019



A 60-year-old man comes to the emergency department with acute substernal chest pain, nausea, and diaphoresis. The patient has a history of liver cirrhosis, hypertension, and hyperlipidemia. He quit smoking 3 months ago but previously smoked a pack of cigarettes daily for 25 years. ECG shows ST elevation in leads II, III, and aVF. He is diagnosed with a myocardial infarction and, after careful discussion of risks and benefits, reperfusion therapy is not performed due to underlying cirrhosis and history of variceal bleeding. The patient is eventually discharged from the hospital with conservative management. Twelve days after his myocardial infarction, he dies suddenly. Light microscopy would most likely show which of the following changes in the myocardium of the inferior wall?

- ☐ A. Dense collagen scar [8%]
- ☐ B. Dense interstitial neutrophil infiltrate [5%]
- ☐ C. Edema and wavy myocyte fibers [3%]
- ☒ D. Granulation tissue with neovascularization [63%]
- ☐ E. Hypereosinophilic myocytes [1%]
- ☐ F. Macrophage infiltration with foam cell formation [16%]
- ☐ G. Normal myocardium [0%]

Omitted

Correct answer
D63%
Answered correctly3 Seconds
Time Spent01/08/2019
Last Updated

Explanation

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Sudden cardiac death **12 days** after myocardial infarction (MI) is most likely due to **ventricular arrhythmias** originating from the infarcted myocardium. The microscopic diagnosis of MI depends on the presence of necrotic myocardium, with areas of acute inflammation and necrosis separated from viable myocardium. After the initial event, several characteristic microscopic changes occur in the infarcted zone in a specific temporal sequence with some overlap in different stages. **During the second week** after MI, the damaged tissue is replaced by **granulation tissue** and **neovascularization** is found in the infarct zone.

(Choice A) Increased collagen deposition and decreased cellularity in the zone of infarcted myocardium generally become evident beginning at 2 weeks postinfarction. Fibrosis continues during weeks 2-8, resulting in a dense collagenous scar at about 2 months postinfarction.

(Choice B) Prominent interstitial neutrophil infiltration is found at the border zone of infarcted myocardium 1-3 days after MI onset. This acute inflammatory infiltrate becomes increasingly prominent over the next few days before receding around days 5-7.

(Choice C) Edema and wavy myocyte fibers with normal-appearing nuclei can be detected in infarcted myocardium by light microscopy approximately 4-12 hours following MI.

(Choice E) Cytoplasmic hypereosinophilia is a characteristic finding of early MI and occurs 12-24 hours after onset.

(Choice F) Macrophage phagocytosis of infarcted myocardium begins at about 3 days postinfarction. The macrophages become laden with degenerating myoglobin pigment and hemosiderin (**not foam cells**, which are indicative of extensive lipid engulfment). By day 10, the accumulated macrophages begin to be replaced by granulation tissue.

(Choice G) Infarcted myocardium usually appears normal under light microscopy up to 4 hours after the onset of myocardial ischemia.

Educational objective:
Prominent granulation tissue and neovascularization are found in the infarct zone during the second week after myocardial infarction.

References

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A 22-year-old woman comes to the office due to worsening dyspnea. The patient has also experienced low-grade fevers, a 6.8-kg (15-lb) weight loss, and syncopal episodes over the last 3 months. Her shortness of breath worsens when sitting and improves when lying down. Medical history is insignificant. The patient does not use tobacco, alcohol, or illicit drugs. Temperature is 37.4 C (99.3 F), blood pressure is 115/72 mm Hg, pulse is 75/min and regular, and respirations are 12/min. The lungs are clear. Cardiac auscultation reveals a low-pitched, mid-diastolic rumble at the cardiac apex. The remainder of the physical examination is normal. ECG reveals left atrial enlargement, and echocardiography shows a large, pedunculated mass in the left atrium. Histologic analysis of this mass will most likely reveal which of the following?

A. Coarse, filamented, branching septate hyphae

B. Gram-positive cocci in clusters

C. Malignant myeloid cells

D. Scattered cells within a mucopolysaccharide stroma

E. Solid mass of platelets and fibrin

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Question Id: 8296



A 22-year-old woman comes to the office due to worsening dyspnea. The patient has also experienced low-grade fevers, a 6.8-kg (15-lb) weight loss, and syncopal episodes over the last 3 months. Her shortness of breath worsens when sitting and improves when lying down. Medical history is insignificant. The patient does not use tobacco, alcohol, or illicit drugs. Temperature is 37.4 C (99.3 F), blood pressure is 115/72 mm Hg, pulse is 75/min and regular, and respirations are 12/min. The lungs are clear. Cardiac auscultation reveals a low-pitched, mid-diastolic rumble at the cardiac apex. The remainder of the physical examination is normal. ECG reveals left atrial enlargement, and echocardiography shows a large, pedunculated mass in the left atrium. Histologic analysis of this mass will most likely reveal which of the following?

- ☐ A. Coarse, filamented, branching septate hyphae [1%]
- ☐ B. Gram-positive cocci in clusters [5%]
- ☐ C. Malignant myeloid cells [10%]
- ☒ D. Scattered cells within a mucopolysaccharide stroma [71%]
- ☐ E. Solid mass of platelets and fibrin [11%]

Omitted

Correct answer
D71%
Answered correctly3 Seconds
Time Spent11/28/2018
Last Updated

Explanation

This patient with a large, pedunculated mass in the left atrium most likely has an **atrial myxoma**. Myxomas are the most common primary cardiac

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This patient with a large, pedunculated mass in the left atrium most likely has an **atrial myxoma**. Myxomas are the most common primary cardiac neoplasm, and approximately 80% arise within the **left atrium**.

Left atrial myxomas frequently obstruct blood flow from the left atrium to the left ventricle, leading to a **murmur** mimicking that of mitral stenosis (ie, mid-diastolic rumble at the apex). The obstruction can also lead to a decrease in cardiac output that manifests as dyspnea, lightheadedness, or syncope. Because the mass is typically mobile, **obstructive symptoms** may be transient and **influenced by position** (ie, upright posture exacerbates mitral obstruction, whereas lying down alleviates it). In some patients, fragments of the mass may embolize into the systemic circulation (eg, resulting in stroke or acute limb ischemia). In addition, some myxomas can produce cytokines (eg, interleukin-6) that lead to **constitutional symptoms** including fever and weight loss.

Histologically, these tumors demonstrate scattered cells within a **mucopolysaccharide stroma** (see image above) and abnormal blood vessels. Myxomas produce a large amount of vascular endothelial growth factor, which contributes to the **angiogenesis** (white arrows), hemorrhaging (seen as brown hemosiderin deposits), and friability characterizing these tumors.

(Choices A and B) Coarse, filamented, branching septate hyphae are found in fungal endocarditis, whereas gram-positive cocci in clusters are consistent with bacterial endocarditis. Both are unlikely in this young woman with no history of valvular disease or intravenous drug use. In addition, endocarditis typically causes a regurgitant murmur, with valvular vegetations seen on echocardiography.

(Choice C) Acute myeloid leukemia (AML) rarely may involve the heart. Signs and symptoms related to anemia (eg, weakness, fatigue), neutropenia (eg, infections), and thrombocytopenia (eg, ecchymoses, epistaxis), are typically present with AML.

(Choice E) Nonbacterial thrombotic endocarditis involves the valvular deposition of sterile platelets and fibrin and most commonly occurs in the setting of advanced malignancy or systemic lupus erythematosus. The disease usually presents with systemic embolization, and heart murmurs are often absent; if a murmur is present, it is typically regurgitant rather than stenotic.

Educational objective:

Myxomas are the most common primary cardiac neoplasm and usually arise within the left atrium. The tumors typically cause position-dependent obstruction of the mitral valve, leading to a mid-diastolic murmur and symptoms of decreased cardiac output (eg, dyspnea, syncope).

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constitutional symptoms including fever and weight loss.

Histologically, these tumors demonstrate scattered cells within a **mucopolysaccharide stroma** (see image above) and abnormal blood vessels. Myxomas produce a large amount of vascular endothelial growth factor, which contributes to the **angiogenesis** (white arrows), hemorrhaging (seen as brown hemosiderin deposits), and friability characterizing these tumors.

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Educational objective:

Myxomas are the most common primary cardiac neoplasm and usually arise within the left atrium. The tumors typically cause position-dependent obstruction of the mitral valve, leading to a mid-diastolic murmur and symptoms of decreased cardiac output (eg, dyspnea, syncope). Constitutional symptoms (eg, fever, weight loss) may also be present. Histologically, the tumors demonstrate scattered cells within a mucopolysaccharide stroma and abnormal blood vessels with hemorrhaging.

References

- Atrial myxoma: case report and a review of the literature.

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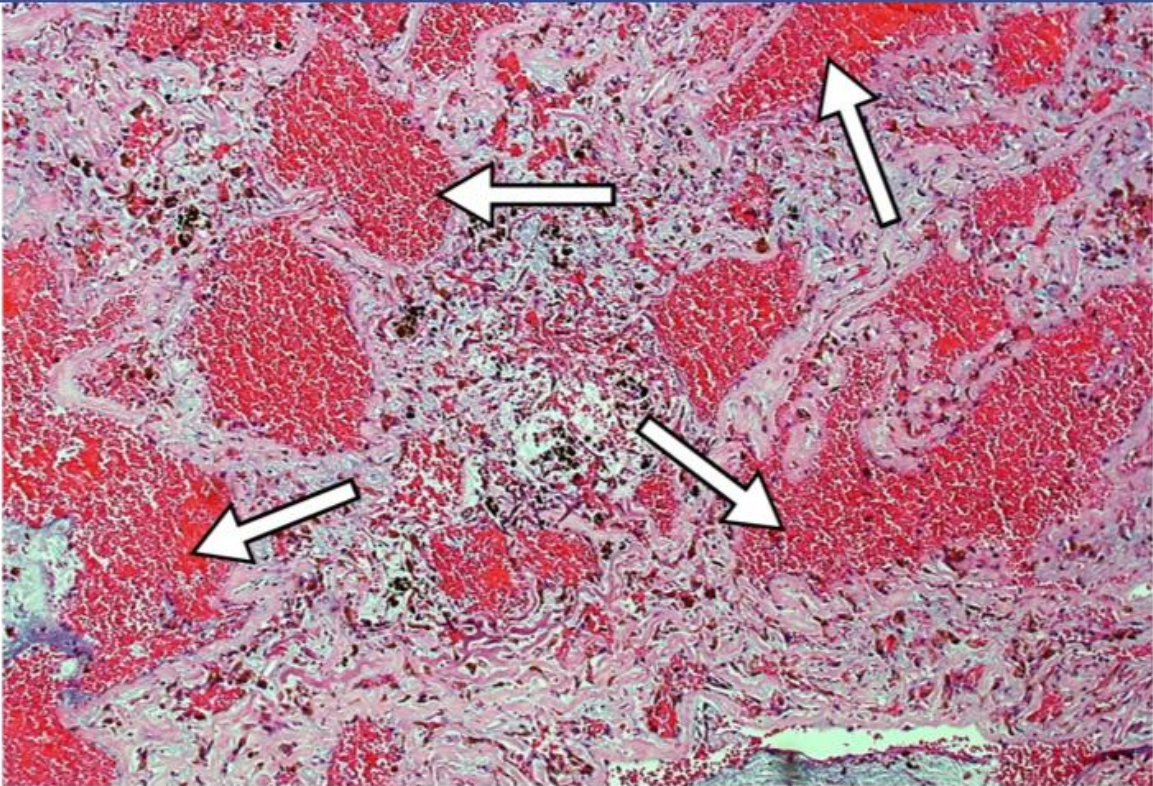
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2/7/2019



A 62-year-old woman comes to the office due to a vague feeling of heaviness in her legs, especially when standing for long periods. She has no history of trauma to the legs. The patient's other medical problems include osteoarthritis and gastroesophageal reflux disease. She has worked as a cashier at a department store for the last 20 years. Vital signs are normal. Jugular venous pressure is normal. The heart has regular rate and rhythm with no murmurs or extra sounds. Lung sounds are clear with normal air movement. The abdomen is soft and without masses. Examination of the lower extremities shows dilated, tortuous veins at the calves and ankles bilaterally, with no erythema or edema. Upper extremity examination is unremarkable. This patient's current condition increases the risk for which of the following?

- ☐ A. Intermittent claudication
- ☐ B. Ischemic stroke
- ☐ C. Myocardial infarction
- ☐ D. Phlegmasia alba dolens
- ☐ E. Pulmonary embolism
- ☐ F. Skin ulcerations

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A 62-year-old woman comes to the office due to a vague feeling of heaviness in her legs, especially when standing for long periods. She has no history of trauma to the legs. The patient's other medical problems include osteoarthritis and gastroesophageal reflux disease. She has worked as a cashier at a department store for the last 20 years. Vital signs are normal. Jugular venous pressure is normal. The heart has regular rate and rhythm with no murmurs or extra sounds. Lung sounds are clear with normal air movement. The abdomen is soft and without masses. Examination of the lower extremities shows dilated, tortuous veins at the calves and ankles bilaterally, with no erythema or edema. Upper extremity examination is unremarkable. This patient's current condition increases the risk for which of the following?

☐

A. Intermittent claudication [13%]

☐

B. Ischemic stroke [0%]

☐

C. Myocardial infarction [0%]

☐

D. Phlegmasia alba dolens [5%]

☐

E. Pulmonary embolism [44%]

☒

F. Skin ulcerations [35%]

Omitted

Correct answer

F

35%

Answered correctly

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Explanation

This patient has **varicose veins** - dilated, tortuous veins typically involving the superficial venous drainage of the leg. Increased **intraluminal pressure** or loss of vessel wall tensile strength can lead to venous dilation and failure of the venous valves. The resulting backflow of blood exacerbates the venous hypertension, precipitating a vicious cycle that leads to further valvular incompetence and venous congestion.

Varicose veins are most common in patients age >50, and the risk is increased by conditions that cause elevated venous pressure (eg, prolonged standing, pregnancy, obesity). There is also often a familial association, suggesting possible genetic factors involving the venous wall or valves. Common complications include chronic edema, stasis dermatitis, **skin ulcerations**, poor wound healing, and infection.

(Choices A, B, and C) Intermittent claudication is achy or crampy pain in the legs caused by inadequate arterial blood flow, usually due to atherosclerosis in the branches of the femoral artery. In contrast to venous disease, in which the pain is worsened by prolonged standing or sitting with the legs dependent, claudication due to arterial disease is worse with exertion and relieved with rest. Because peripheral artery atherosclerosis is often associated with atherosclerotic disease elsewhere, patients with intermittent claudication have an increased risk for ischemic stroke and myocardial infarction.

(Choices D and E) Deep venous thrombosis of the lower extremity can lead to pulmonary embolism. In addition, massive iliofemoral thrombosis can cause an acute rise in tissue pressure that impairs arterial inflow, leading to phlegmasia alba dolens (painful white "milk leg"). Although varicose veins can sometimes be complicated by thrombophlebitis, it primarily involves the superficial rather than deep venous system and does not increase the risk for pulmonary embolism or phlegmasia alba dolens.

Educational objective:

Varicose veins are dilated, tortuous veins most commonly found in the superficial veins of the leg. They are caused by chronically increased intraluminal pressure and/or loss of tensile strength in the vessel wall, leading to incompetence of the venous valves. Common complications include edema, stasis dermatitis, skin ulcerations, poor wound healing, and infections.

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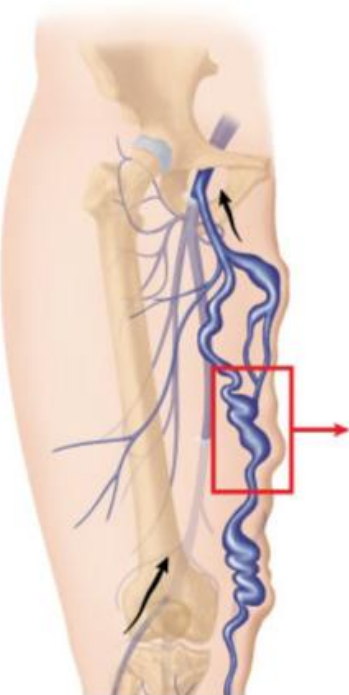
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Pathogenesis of varicose veins



Dysfunctional valve

Retrograde blood flow

Dilatated, tortuous vein

Varicose vein

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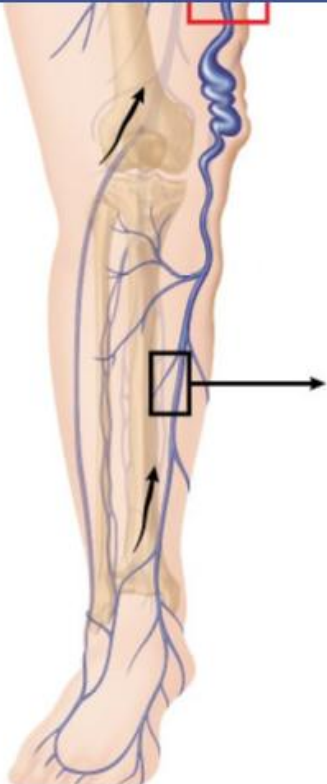
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Dilatated, tourtuuous vein

Varicose vein

Normal blood flow

Competent venous valve

Normal vein

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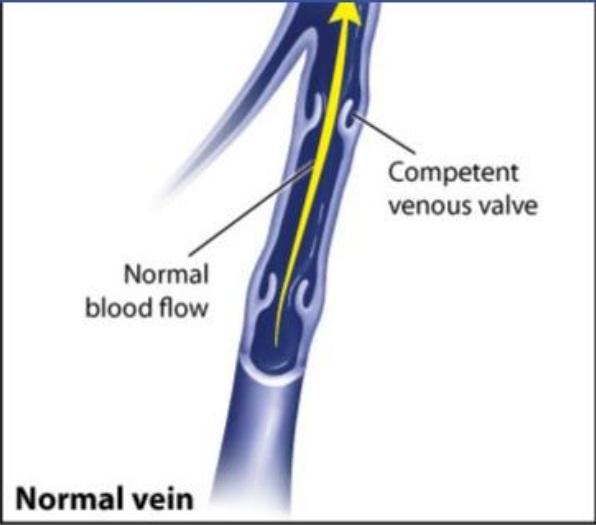
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Normal blood flow

Competent venous valve

Normal vein

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An 18-year-old man suddenly collapses during a high school soccer game. There was no preceding collision or trauma. Immediate resuscitation is initiated but he dies before the emergency medical team arrives. The patient had recently experienced occasional exertion-related chest discomfort and dyspnea. Otherwise, he had no significant medical history. He took no medications and did not use tobacco, alcohol, or illicit drugs. Which of the following is most likely to be seen on autopsy?

☐

A. Atherosclerotic narrowing of the left anterior descending artery

☐

B. Cardiac hypertrophy with patchy interstitial fibrosis

☐

C. Dilation of all cardiac chambers and flabby ventricular walls

☐

D. Endocardial thickening and noncompliant ventricular walls

☐

E. Thrombus at the bifurcation of the main pulmonary artery

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Text Zoom

An 18-year-old man suddenly collapses during a high school soccer game. There was no preceding collision or trauma. Immediate resuscitation is initiated but he dies before the emergency medical team arrives. The patient had recently experienced occasional exertion-related chest discomfort and dyspnea. Otherwise, he had no significant medical history. He took no medications and did not use tobacco, alcohol, or illicit drugs. Which of the following is most likely to be seen on autopsy?

A. Atherosclerotic narrowing of the left anterior descending artery [1%]

B. Cardiac hypertrophy with patchy interstitial fibrosis [53%]

C. Dilation of all cardiac chambers and flabby ventricular walls [6%]

D. Endocardial thickening and noncompliant ventricular walls [37%]

E. Thrombus at the bifurcation of the main pulmonary artery [1%]

Omitted

Correct answer
B

53%

Answered correctly

3 Seconds

Time Spent

01/07/2019

Last Updated

Explanation

Sudden cardiac death (SCD) is most often precipitated by cardiac arrhythmia (eg, ventricular tachycardia/fibrillation) that leads to markedly decreased cardiac output and impaired coronary artery perfusion. In older adults (eg, age ≥ 35), myocardial ischemia due to atherosclerotic coronary artery disease (**Choice A**) is the most common cause of SCD. However, young adults who experience SCD usually have underlying structural heart disease: **hypertrophic cardiomyopathy** (HCM) is one of the most common underlying diseases present. Most patients with HCM

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Sudden cardiac death (SCD) is most often precipitated by cardiac arrhythmia (eg, ventricular tachycardia/fibrillation) that leads to markedly decreased cardiac output and impaired coronary artery perfusion. In older adults (eg, age ≥ 35), myocardial ischemia due to atherosclerotic coronary artery disease (**Choice A**) is the most common cause of SCD. However, young adults who experience SCD usually have underlying structural heart disease; **hypertrophic cardiomyopathy** (HCM) is one of the most common underlying diseases present. Most patients with HCM are asymptomatic, but some may experience exertional dyspnea, fatigue, chest pain, or syncope.

HCM is an autosomal dominant genetic disorder caused by mutation of one of several genes coding for cardiac sarcomere proteins. It is characterized by **cardiomyocyte hypertrophy** (predominantly affecting the septum) and **myofiber disarray** consisting of an irregular arrangement of **abnormally shaped cardiomyocytes** with bizarrely-shaped nuclei and increased **interstitial fibrosis**. This structural disarray leads to conduction abnormalities that predispose to ventricular arrhythmia and SCD.

(Choice C) Dilated cardiac chambers and flabby ventricular walls are consistent with dilated cardiomyopathy, a less common cause of SCD than HCM in young adults. A patient with DCM would likely have symptoms of heart failure (eg, lower extremity swelling, dyspnea, orthopnea) and would not be capable of participating in strenuous activities such as soccer.

(Choice D) Endocardial thickening and noncompliant ventricular walls are suggestive of restrictive cardiomyopathy, which may occur due to infiltrative disease (eg, amyloidosis, sarcoidosis). Although restrictive cardiomyopathy can cause SCD, there is usually a preceding course of progressive and symptomatic heart failure.

(Choice E) Thrombus at the bifurcation of the main pulmonary artery (ie, saddle pulmonary embolism) is a potential cause of SCD; however, a preceding history of exertion-related dyspnea and chest discomfort would not be expected.

Educational objective:

Hypertrophic cardiomyopathy is a common cause of sudden cardiac death (SCD) in young adults. Histologic features include cardiomyocyte hypertrophy and myofiber disarray with increased interstitial fibrosis. The structural disarray creates a substrate for ventricular arrhythmia (eg, ventricular tachycardia, ventricular fibrillation) that can lead to SCD.

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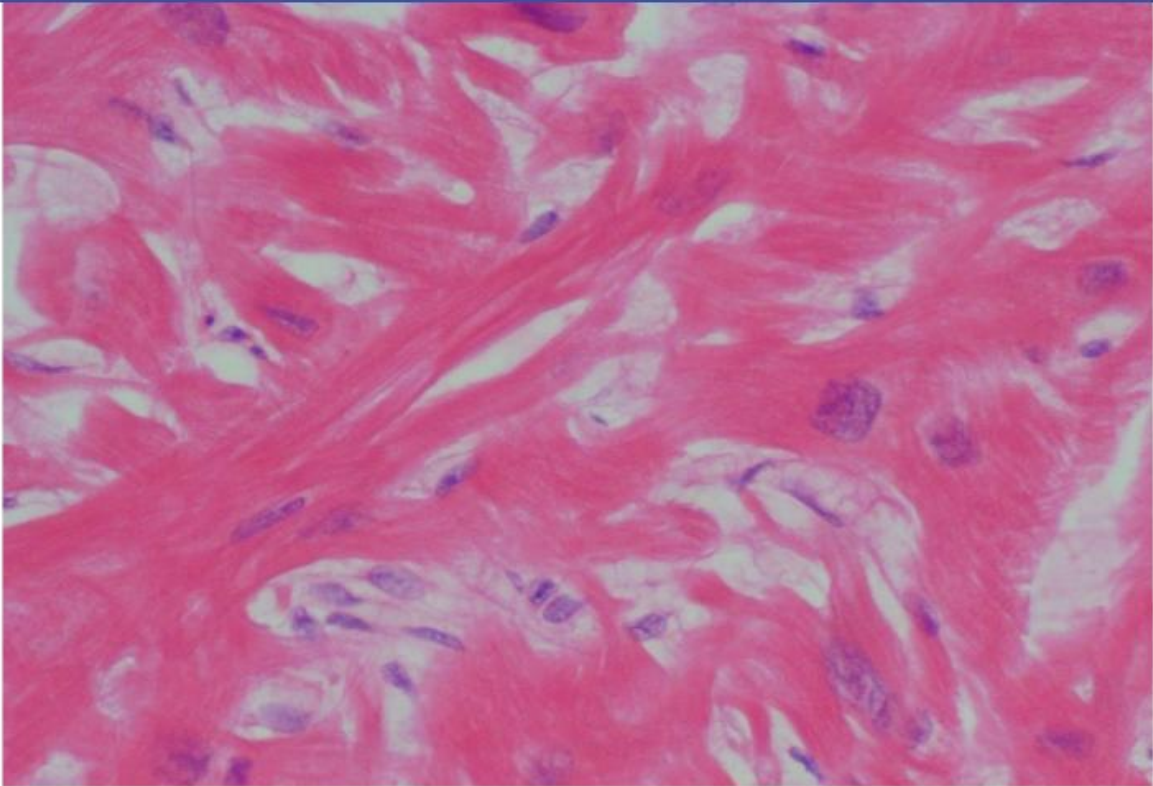
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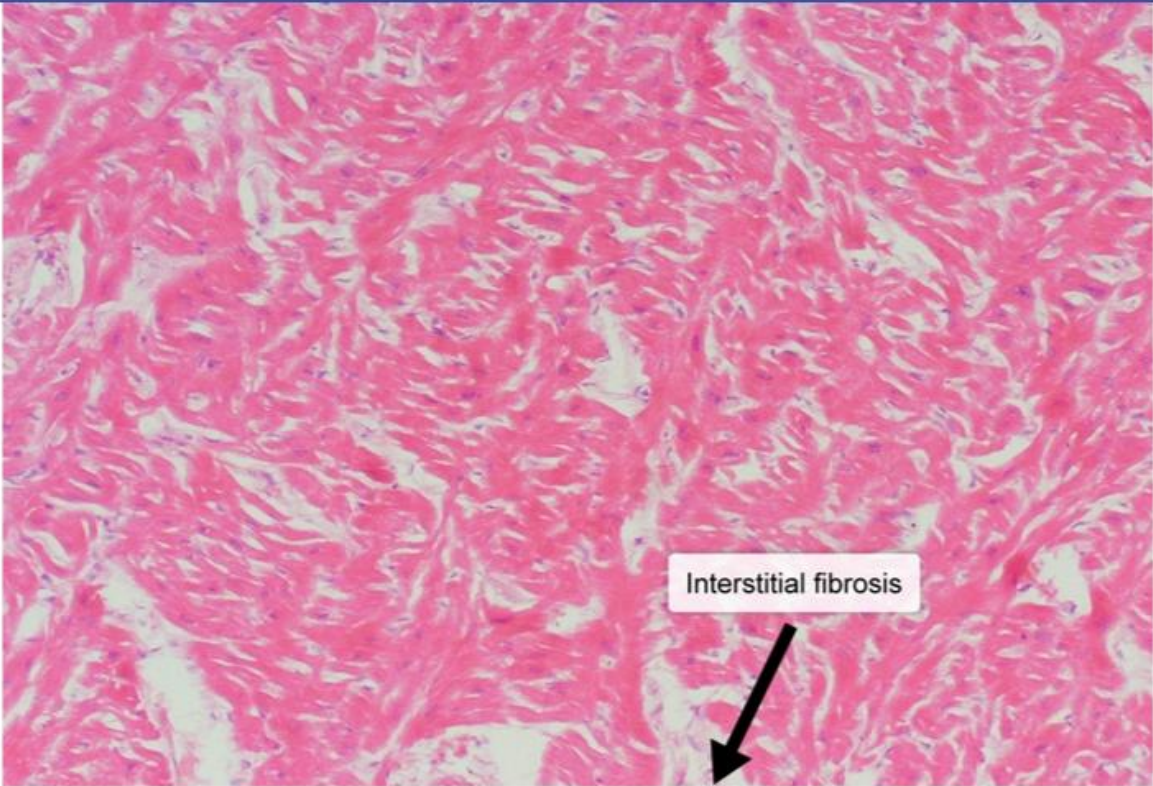
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Interstitial fibrosis

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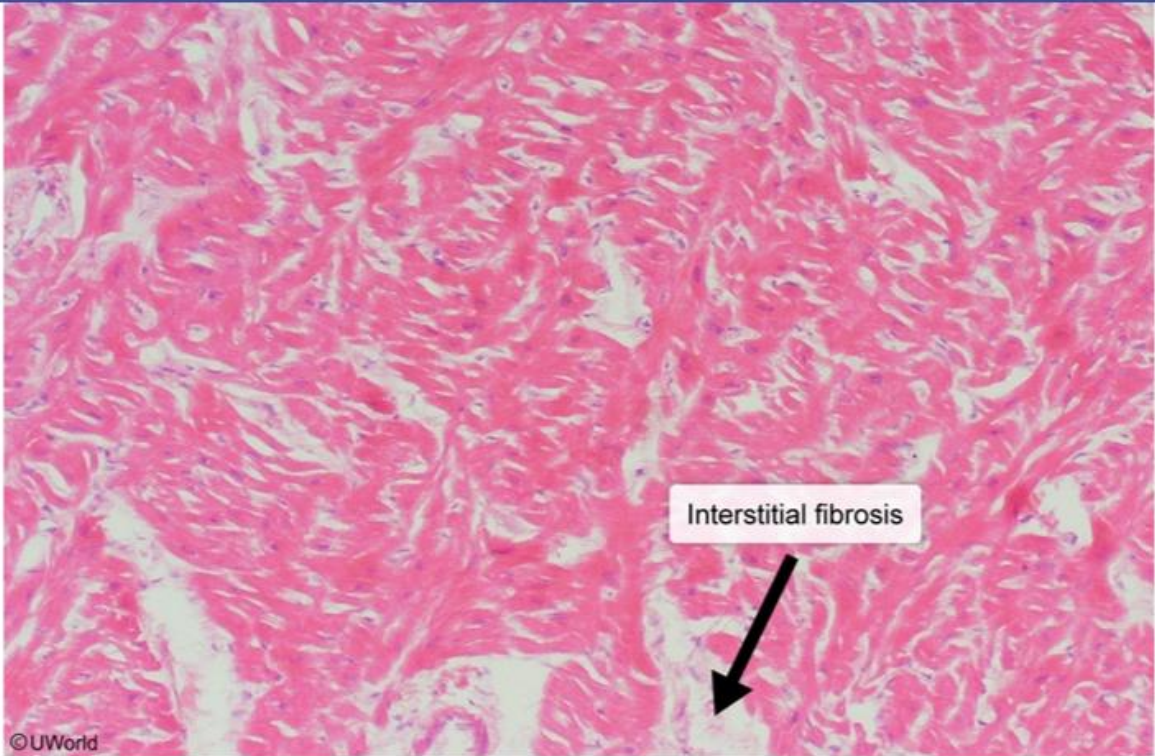
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Interstitial fibrosis

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A 69-year-old man comes to the clinic due to a squeezing pressure in his midline chest and neck that occurs when walking. He has a history of hypertension and type 2 diabetes mellitus. The patient is an ex-smoker with a 20-pack-year history. Coronary angiography shows extensive atherosclerosis and near-total occlusion of the left anterior descending artery. Further testing shows normal resting left ventricular ejection fraction with no regional wall motion abnormalities. The absence of myocardial necrosis and scarring despite vessel occlusion in this patient can be best explained by which of the following features of the occluding plaque?

☐ A. Active peripheral inflammation

☐ B. High calcium content

☐ C. Lipid-rich core

☐ D. Ostial location

☐ E. Slow growth rate

☐ F. Thin fibrous cap

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A 69-year-old man comes to the clinic due to a squeezing pressure in his midline chest and neck that occurs when walking. He has a history of hypertension and type 2 diabetes mellitus. The patient is an ex-smoker with a 20-pack-year history. Coronary angiography shows extensive atherosclerosis and near-total occlusion of the left anterior descending artery. Further testing shows normal resting left ventricular ejection fraction with no regional wall motion abnormalities. The absence of myocardial necrosis and scarring despite vessel occlusion in this patient can be best explained by which of the following features of the occluding plaque?

A. Active peripheral inflammation [2%]

B. High calcium content [4%]

C. Lipid-rich core [6%]

D. Ostial location [7%]

E. Slow growth rate [62%]

F. Thin fibrous cap [16%]

Omitted

Correct answer
E

62%

Answered correctly

6 Seconds

Time Spent

10/29/2018

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Explanation

In response to progressive reductions in blood flow caused by atherosclerotic coronary artery disease, **ischemic myocardium** may release

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In response to progressive reductions in blood flow caused by atherosclerotic coronary artery disease, **ischemic myocardium** may release growth factors (eg, vascular endothelial growth factor) to stimulate the formation and maturation of **collateral vessels**. In this patient, the **slow development** of a **stable atherosclerotic plaque** in the left anterior descending artery (LAD) likely allowed collateral vessels from the right coronary artery to supply the ischemic myocardium located distal to the LAD occlusion. Because there is low hydrostatic pressure distal to the LAD occlusion, a pressure gradient is created that facilitates blood flow through the small collateral vessels.

An **unstable atherosclerotic plaque** is less likely to allow the development of viable collateral vessels, as it is more likely to rupture before it becomes large enough to cause significant chronic ischemia; rupture leads to overlying thrombus and **abrupt arterial occlusion** with acute myocardial ischemia/**infarction** and cellular necrosis. The rapid development of ischemia allows insufficient time for collateral vessels to develop and mature.

(Choices A, C, and F) Atherosclerotic plaques with active inflammation (containing activated macrophages that secrete metalloproteinases and degrade collagen), high core concentration of lipids, and/or a thin fibrous cap are unstable and more likely to rupture. All of these properties are less likely in an atherosclerotic lesion that has grown to create near-total occlusion without rupturing.

(Choice B) The total coronary artery calcium content correlates with the total atherosclerotic plaque burden, and calcium scoring by cardiac CT scan is sometimes used to estimate the severity of coronary artery disease. However, the calcium content of an individual plaque does not correlate with its stability or likelihood of rupture.

(Choice D) Ostial atherosclerotic plaques (those located at a branch point of 2 arteries) are more likely to occlude blood flow to multiple areas of myocardium, thereby limiting the availability of nearby well-perfused arteries from which viable collateral vessels can develop.

Educational objective:

Gradually developing myocardial ischemia encourages the formation and maturation of collateral vessels and is most likely to occur in the setting of a slow-growing, stable atherosclerotic plaque. An unstable atherosclerotic plaque (eg, that with active inflammation, a lipid-rich core, and/or a thin fibrous cap) is more likely to rupture, resulting in the abrupt onset of ischemia/infarction that precludes the development of viable collateral vessels.

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A 57-year-old man dies 30 minutes after the onset of severe chest pain while driving to the emergency department. According to his wife, he had been complaining of intermittent short-lived episodes of chest pain recently. His past medical history was significant for hypertension, insulin-dependent type 2 diabetes mellitus, and hypercholesterolemia. The patient was not compliant with medications or follow-up. He had a strong family history of heart disease and type 2 diabetes mellitus. An autopsy is performed. Cross-section of the mid right coronary artery is shown in this [exhibit](#). Which of the following is the most likely cause of death in this patient?

☐ A. Atrial fibrillation

☐ B. Cardiac free wall rupture

☐ C. Embolic stroke

☐ D. Regional contractile dysfunction

☐ E. Ventricular fibrillation

☐ F. Ventricular septal rupture

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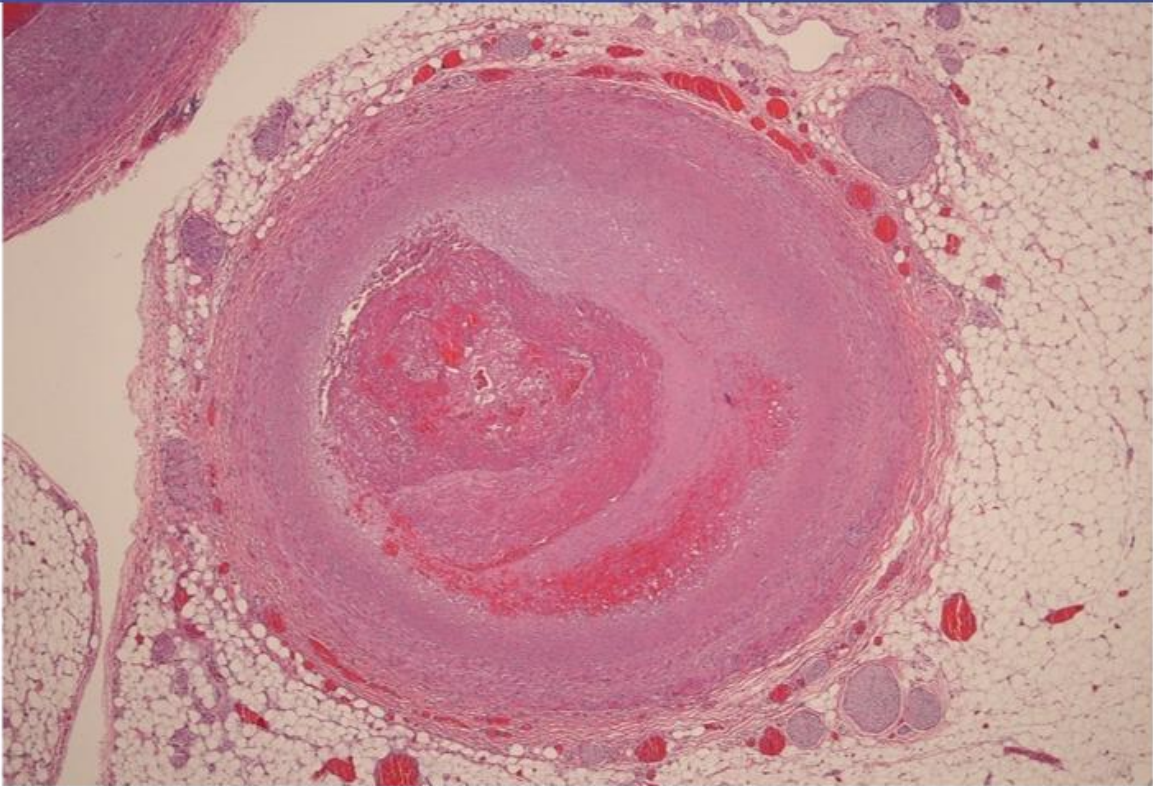
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A 57-year-old man dies 30 minutes after the onset of severe chest pain while driving to the emergency department. According to his wife, he had been complaining of intermittent short-lived episodes of chest pain recently. His past medical history was significant for hypertension, insulin-dependent type 2 diabetes mellitus, and hypercholesterolemia. The patient was not compliant with medications or follow-up. He had a strong family history of heart disease and type 2 diabetes mellitus. An autopsy is performed. Cross-section of the mid right coronary artery is shown in this [exhibit](#). Which of the following is the most likely cause of death in this patient?

- ☐ A. Atrial fibrillation [8%]
- ☐ B. Cardiac free wall rupture [4%]
- ☐ C. Embolic stroke [16%]
- ☐ D. Regional contractile dysfunction [18%]
- ☒ E. Ventricular fibrillation [49%]
- ☐ F. Ventricular septal rupture [1%]

Omitted

Correct answer
E49%
Answered correctly26 Seconds
Time Spent11/17/2018
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Explanation

This patient's clinical presentation is suggestive of **coronary artery disease** (chest pain and multiple risk factors) causing an **acute myocardial**

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This patient's clinical presentation is suggestive of **coronary artery disease** (chest pain and multiple risk factors) causing an **acute myocardial infarction** (MI) due to thrombotic occlusion of the right coronary artery (RCA). The cross-sectional image shows a large atheromatous plaque causing significant stenosis with superimposed thrombosis (arrow) leading to complete lumen occlusion of the RCA.

Sudden cardiac death (SCD) refers to abrupt cessation of organized cardiac activity with hemodynamic collapse, causing an inability to maintain adequate tissue perfusion. SCD typically occurs due to malignant ventricular arrhythmias (sustained ventricular tachycardia/fibrillation), and it is related to coronary heart disease in approximately 70% of the patients. **Ventricular fibrillation (VF)** is the most frequent mechanism of SCD in the first 48 hours after acute MI and is related to electrical instability due to lack of perfusion in the ischemic myocardium.

(Choice A) Atrial fibrillation (AF) can occur during acute MI; however, AF does not lead to SCD in such patients.

(Choices B and F) Ventricular septal rupture (most common with left anterior descending artery occlusion) and cardiac free wall rupture can occur as mechanical complications of acute MI and lead to cardiogenic shock and/or sudden death. These complications typically occur 3-7 days after the initial MI episode. Autopsy in free wall rupture shows hemopericardium and a perforating wound in the wall of the left ventricle.

(Choice C) Systemic thromboembolism or embolic stroke can arise from left ventricular (LV) mural thrombus from an area of infarcted and akinetic myocardium. However, LV thrombus typically takes several days to form and does not lead to SCD.

(Choice D) Severe LV contractile dysfunction can cause profound cardiogenic shock with multiorgan systemic failure and death. This is typically seen in large anterior MI due to abrupt occlusion of the proximal left anterior descending artery involving >50% of the myocardium (but VF is still the leading cause of SCD in these patients). However, a mid right coronary artery obstruction is less likely to result in severe LV dysfunction.

Educational objective:

Ventricular fibrillation is the most frequent mechanism of sudden cardiac death in the first 48 hours after acute myocardial infarction and is related to electrical instability in the ischemic myocardium.

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A 35-year-old previously healthy man is brought to the emergency department after being involved in a motor vehicle accident. He has significant blunt chest and head trauma. Shortly after he arrives, his blood pressure drops suddenly and he begins experiencing respiratory distress. On physical examination, the patient is tachycardic and tachypneic. His lungs are clear to auscultation with vesicular breath sounds heard bilaterally. He has jugular venous distention, and his systolic blood pressure falls 15 mm Hg with inspiration. Which of the following is the most likely cause of this patient's deterioration?

☐ A. Tension pneumothorax

☐ B. Epidural hematoma

☐ C. Hemothorax

☐ D. Aortic rupture

☐ E. Cardiac tamponade

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A 35-year-old previously healthy man is brought to the emergency department after being involved in a motor vehicle accident. He has significant blunt chest and head trauma. Shortly after he arrives, his blood pressure drops suddenly and he begins experiencing respiratory distress. On physical examination, the patient is tachycardic and tachypneic. His lungs are clear to auscultation with vesicular breath sounds heard bilaterally. He has jugular venous distention, and his systolic blood pressure falls 15 mm Hg with inspiration. Which of the following is the most likely cause of this patient's deterioration?

☐

A. Tension pneumothorax [4%]

☐

B. Epidural hematoma [0%]

☐

C. Hemothorax [2%]

☐

D. Aortic rupture [3%]

☒

E. Cardiac tamponade [88%]

Omitted

Correct answer
E

88%

Answered correctly

4 Seconds

Time Spent

02/06/2019

Last Updated

Explanation

This patient has pulsus paradoxus (a fall in systolic blood pressure ≥ 10 mm Hg on inspiration), jugular venous distention, and tachycardia. Based on his history and physical examination findings, cardiac tamponade is the most likely diagnosis. Cardiac tamponade should be suspected in any patient who has the characteristic triad of muffled heart sounds, jugular venous distention, and hypotension.

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Explanation

This patient has pulsus paradoxus (a fall in systolic blood pressure ≥ 10 mm Hg on inspiration), jugular venous distention, and tachycardia. Based on his history and physical examination findings, cardiac tamponade is the most likely diagnosis. Cardiac tamponade should be suspected in any patient who has the characteristic triad of muffled heart sounds, jugular venous distention, and hypotension.

During inspiration, the pressure in the pleural space and lung interstitium decreases, increasing pulmonary vascular capacitance. This causes a fall in venous inflow to the left heart, resulting in decreased left ventricular stroke volume and a drop in systolic blood pressure (normally < 10 mm Hg). The inspiratory drop in systolic blood pressure is exacerbated in cardiac tamponade due to extrinsic compression of the ventricles. This external compression acts to equalize left and right ventricular diastolic pressures, allowing the intraventricular septum to bulge into the left ventricle as a result of the inspiratory increase in right ventricular filling. This further reduces left ventricular stroke volume, leading to a drop in systolic blood pressure > 10 mm Hg.

Pulsus paradoxus can also occur in other conditions, including constrictive pericarditis, chronic obstructive pulmonary disease, asthma, and pulmonary embolism.

(Choice A) Tension pneumothorax can cause hypotension, tachycardia, tachypnea, and jugular venous distention. It can result from blunt or penetrating chest trauma that injures the visceral pleura or tracheobronchial tree. However, tension pneumothorax would cause absent breath sounds and hyperresonance to percussion on the affected side.

(Choice B) An epidural hematoma may initially cause hypertension and bradycardia due to a Cushing's response to increased intracranial pressure. If an epidural hematoma progresses to cause herniation, hypotension may follow. An epidural hematoma would not cause elevated jugular venous distention or pulsus paradoxus.

(Choice C) Blunt or penetrating chest trauma could cause a massive hemothorax with hemorrhagic hypotension, tachycardia, and tachypnea. However, absent breath sounds and dullness to percussion would be found on the affected side.

(Choice D) An aortic rupture would cause hypovolemic shock, not elevated jugular venous distention.

Educational objective:

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fall in venous inflow to the left heart, resulting in decreased left ventricular stroke volume and a drop in systolic blood pressure (normally < 10 mm Hg). The inspiratory drop in systolic blood pressure is exacerbated in cardiac tamponade due to extrinsic compression of the ventricles. This external compression acts to equalize left and right ventricular diastolic pressures, allowing the intraventricular septum to bulge into the left ventricle as a result of the inspiratory increase in right ventricular filling. This further reduces left ventricular stroke volume, leading to a drop in systolic blood pressure > 10 mm Hg.

Pulsus paradoxus can also occur in other conditions, including constrictive pericarditis, chronic obstructive pulmonary disease, asthma, and pulmonary embolism.

(Choice A) Tension pneumothorax can cause hypotension, tachycardia, tachypnea, and jugular venous distension. It can result from blunt or penetrating chest trauma that injures the visceral pleura or tracheobronchial tree. However, tension pneumothorax would cause absent breath sounds and hyperresonance to percussion on the affected side.

(Choice B) An epidural hematoma may initially cause hypertension and bradycardia due to a Cushing's response to increased intracranial pressure. If an epidural hematoma progresses to cause herniation, hypotension may follow. An epidural hematoma would not cause elevated jugular venous distension or pulsus paradoxus.

(Choice C) Blunt or penetrating chest trauma could cause a massive hemothorax with hemorrhagic hypotension, tachycardia, and tachypnea. However, absent breath sounds and dullness to percussion would be found on the affected side.

(Choice D) An aortic rupture would cause hypovolemic shock, not elevated jugular venous distension.

Educational objective:

The combination of jugular venous distension, hypotension, and muffled heart sounds is highly suggestive of cardiac tamponade. Tachycardia and pulsus paradoxus are also frequently seen with tamponade. Lung examination is normal, which can help distinguish cardiac tamponade from tension pneumothorax.

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Text Zoom

A 36-year-old woman is brought to the emergency department with sudden-onset right side weakness and speech difficulty. During the last 3 weeks, the patient has experienced progressive fatigue, malaise, and low-grade fevers. Despite the symptoms, she did not seek medical attention and did not take any medications. She had a dental extraction 5 weeks ago, which was uncomplicated. The patient has never previously been significantly ill or hospitalized. She works as a receptionist at a legal firm and has never traveled outside the United States. She does not use tobacco, alcohol, or illicit drugs. The patient is admitted to the hospital, but despite adequate resuscitative measures, she dies 2 hours later. Gross autopsy shows large, friable irregular masses attached to the atrial surface of a valve. Which of the following underlying conditions most likely predisposed this patient to her presenting disease?

A. Atrial septal defect

B. Coronary artery disease

C. Hypertrophic cardiomyopathy

D. Mitral annular calcification

E. Regurgitant mitral valve prolapse

F. Rheumatic heart disease

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2/7/2019

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Tutorial

Lab Values

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Calculator

Reverse Color

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Text Zoom

A 36-year-old woman is brought to the emergency department with sudden-onset right side weakness and speech difficulty. During the last 3 weeks, the patient has experienced progressive fatigue, malaise, and low-grade fevers. Despite the symptoms, she did not seek medical attention and did not take any medications. She had a dental extraction 5 weeks ago, which was uncomplicated. The patient has never previously been significantly ill or hospitalized. She works as a receptionist at a legal firm and has never traveled outside the United States. She does not use tobacco, alcohol, or illicit drugs. The patient is admitted to the hospital, but despite adequate resuscitative measures, she dies 2 hours later. Gross autopsy shows large, friable irregular masses attached to the atrial surface of a valve. Which of the following underlying conditions most likely predisposed this patient to her presenting disease?

A. Atrial septal defect [9%]

B. Coronary artery disease [0%]

C. Hypertrophic cardiomyopathy [0%]

D. Mitral annular calcification [11%]

E. Regurgitant mitral valve prolapse [33%]

F. Rheumatic heart disease [44%]

Omitted

Correct answer
E

33%

Answered correctly

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Explanation

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Explanation

This patient had **subacute infective endocarditis (IE)** complicated by an **embolic stroke**. The large, friable irregular masses seen on autopsy are most likely vegetations and are the most probable source of the embolus that lodged in one of the patient's intracranial arteries (presenting symptoms indicate the left middle cerebral artery).

The mitral valve is the most common valve affected by IE. **Mitral valve prolapse (MVP)** is the most common underlying valvular disease predisposing to the development of IE in **developed countries**, particularly when it is associated with coexistent **mitral regurgitation**. Microscopic deposits of platelets and fibrin occur spontaneously in individuals with valvular disease secondary to endocardial injury from turbulent blood flow. These deposits then become colonized by microorganisms (eg, *Streptococcus viridans*) during episodes of bacteremia (eg, following dental extraction).

(Choice A) Congenital heart lesions that predispose to increased risk of IE include bicuspid aortic valve, ventricular septal defects, patent ductus arteriosus, and unrepaired tetralogy of Fallot. Isolated atrial septal defects are not commonly associated with an increased risk of IE, likely due to the low-pressure differential between the atria and the absence of high-velocity intracardiac flow jets that can damage the endocardium.

(Choice B) Coronary heart disease can cause ischemic papillary muscle dysfunction with resultant mitral regurgitation. However, the clinical presentation and autopsy findings in this patient are most consistent with MVP and IE.

(Choice C) IE is a rare complication of hypertrophic cardiomyopathy and is almost always seen in patients with outflow obstruction. **Hypertrophic cardiomyopathy** is characterized by asymmetric ventricular hypertrophy leading to reduced left ventricular cavity size.

(Choice D) Mitral annular calcification refers to degeneration and progressive calcium deposition involving the fibrous annulus of the mitral valve. Mitral annular calcification is usually seen in older adults (age >60), and the annulus is not a common location for IE.

(Choice F) Rheumatic heart disease (RHD) is a late (10-20 years) complication of acute rheumatic fever (eg, migratory arthritis, carditis/valvulitis, Sydenham chorea, erythema marginatum, subcutaneous nodules). RHD was historically considered a frequent predisposing factor for IE; however, MVP, valvular sclerosis, and mechanical valves are now more common causes in developed nations due to the recognition and treatment of group A streptococcal pharyngitis. Alternatively, RHD is the most important form of acquired heart disease in children and young

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microscopic deposits of platelets and fibrin occur spontaneously in individuals with valvular disease secondary to endocardial injury from turbulent blood flow. These deposits then become colonized by microorganisms (eg, *Streptococcus viridans*) during episodes of bacteremia (eg, following dental extraction).

(Choice A) Congenital heart lesions that predispose to increased risk of IE include bicuspid aortic valve, ventricular septal defects, patent ductus arteriosus, and unrepaired tetralogy of Fallot. Isolated atrial septal defects are not commonly associated with an increased risk of IE, likely due to the low-pressure differential between the atria and the absence of high-velocity intracardiac flow jets that can damage the endocardium.

(Choice B) Coronary heart disease can cause ischemic papillary muscle dysfunction with resultant mitral regurgitation. However, the clinical presentation and autopsy findings in this patient are most consistent with MVP and IE.

(Choice C) IE is a rare complication of hypertrophic cardiomyopathy and is almost always seen in patients with outflow obstruction. **Hypertrophic cardiomyopathy** is characterized by asymmetric ventricular hypertrophy leading to reduced left ventricular cavity size.

(Choice D) Mitral annular calcification refers to degeneration and progressive calcium deposition involving the fibrous annulus of the mitral valve. Mitral annular calcification is usually seen in older adults (age >60), and the annulus is not a common location for IE.

(Choice F) Rheumatic heart disease (RHD) is a late (10-20 years) complication of acute rheumatic fever (eg, migratory arthritis, carditis/valvulitis, Sydenham chorea, erythema marginatum, subcutaneous nodules). RHD was historically considered a frequent predisposing factor for IE; however, MVP, valvular sclerosis, and mechanical valves are now more common causes in developed nations due to the recognition and treatment of group A streptococcal pharyngitis. Alternatively, RHD is the most important form of acquired heart disease in children and young adults in developing countries.

Educational objective:

Mitral valve prolapse with regurgitation is the most common predisposing condition for native valve infective endocarditis (IE) in developed nations. Rheumatic heart disease remains a frequent cause of IE in developing nations.

References

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A 10-year-old immigrant from Eastern Europe is brought to the office due to exertional dyspnea and fatigability. The boy tires easily when walking and cannot keep up with his peers at the playground. According to his parents, he was diagnosed with a congenital heart disease in infancy, for which they refused treatment. They cannot recall the details of his diagnosis. The patient also has had occasional respiratory infections throughout childhood that have not required hospitalization. He takes a daily multivitamin and no medications. He has received only a few childhood vaccinations based on parental preference. The patient has no family history of heart disease. Physical examination shows toe cyanosis and clubbing but no finger abnormalities. All extremity pulses are full and equal. Which of the following is the most likely diagnosis?

☐

A. Coarctation of the aorta

☐

B. Patent ductus arteriosus

☐

C. Primum-type atrial septal defect

☐

D. Secundum-type atrial septal defect

☐

E. Tetralogy of Fallot

☐

F. Ventricular septal defect

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Text Zoom

A 10-year-old immigrant from Eastern Europe is brought to the office due to exertional dyspnea and fatigability. The boy tires easily when walking and cannot keep up with his peers at the playground. According to his parents, he was diagnosed with a congenital heart disease in infancy, for which they refused treatment. They cannot recall the details of his diagnosis. The patient also has had occasional respiratory infections throughout childhood that have not required hospitalization. He takes a daily multivitamin and no medications. He has received only a few childhood vaccinations based on parental preference. The patient has no family history of heart disease. Physical examination shows toe cyanosis and clubbing but no finger abnormalities. All extremity pulses are full and equal. Which of the following is the most likely diagnosis?

☐

A. Coarctation of the aorta [17%]

☒

B. Patent ductus arteriosus [31%]

☐

C. Primum-type atrial septal defect [2%]

☐

D. Secundum-type atrial septal defect [5%]

☐

E. Tetralogy of Fallot [20%]

☐

F. Ventricular septal defect [21%]

Omitted

Correct answer
B

31%

Answered correctly

24 Seconds

Time Spent

01/07/2019

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Explanation

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Patent ductus arteriosus (PDA) is a vascular connection between the main pulmonary artery and the aorta that normally obliterates after birth. Clinical features vary depending on the size. Small PDAs are characterized by a **continuous, machinelike murmur** (left-to-right shunting) with no significant symptoms. Large PDAs can present anytime during childhood with progressive pulmonary hypertension and reversal of the shunt to right-to-left. The characteristic continuous murmur decreases as the pulmonary pressure rises and ultimately disappears. Typical consequences include **heart failure** (eg, shortness of breath, fatigability), and cyanosis (**Eisenmenger syndrome**). The cyanosis and clubbing are most pronounced in the lower extremities (**differential cyanosis and clubbing**) because the PDA delivers unoxygenated blood distal to the left subclavian artery.

(Choice A) **Coarctation of the aorta** is most commonly located in the juxtaductal region just distal to the left subclavian artery. It typically presents in children and adults as a blood pressure discrepancy and pulse delay between the upper and lower extremities. In neonates, severe coarctation can present with heart failure, especially once the ductal tissue collapses. If the ductus arteriosus remains patent, deoxygenated blood can bypass the coarctation and cause differential cyanosis in infancy. However, infants with severe coarctation are unlikely to survive without surgical correction.

(Choices C, D, and F) Atrial and ventricular septal defects initially are accompanied by left-to-right intracardiac shunting and are therefore acyanotic at birth. Large septal defects are at risk for shunt reversal and **Eisenmenger syndrome**. However, the arterial oxygen desaturation and cyanosis affect the upper and lower body equally.

(Choice E) **Tetralogy of Fallot** is generally characterized by whole-body cyanosis at birth as the result of right-to-left shunting across the ventricular septal defect. The right-to-left shunt is intracardiac, and therefore cyanosis typically involves the whole body.

Educational objective:

Differential clubbing and cyanosis without blood pressure or pulse discrepancy are pathognomonic for a large patent ductus arteriosus complicated by Eisenmenger syndrome (reversal of shunt flow from left-to-right to right-to-left). Severe coarctation of the aorta can cause lower extremity cyanosis. Right-to-left shunting in patients with large septal defects and tetralogy of Fallot results in whole-body cyanosis.

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(Choice A) Coarctation of the aorta is most commonly located in the juxtaductal region just distal to the left subclavian artery. It typically presents in children and adults as a blood pressure discrepancy and pulse delay between the upper and lower extremities. In neonates, severe coarctation can present with heart failure, especially once the ductal tissue collapses. If the ductus arteriosus remains patent, deoxygenated blood can bypass the coarctation and cause differential cyanosis in infancy. However, infants with severe coarctation are unlikely to survive without surgical correction.

(Choice E) Tetralogy of Fallot is generally characterized by whole-body cyanosis at birth as the result of right-to-left shunting across the ventricular septal defect. The right-to-left shunt is intracardiac, and therefore cyanosis typically involves the whole body.

Differential clubbing and cyanosis without blood pressure or pulse discrepancy are pathognomonic for a large patent ductus arteriosus complicated by Eisenmenger syndrome (reversal of shunt flow from left-to-right to right-to-left). Severe coarctation of the aorta can cause lower extremity cyanosis. Right-to-left shunting in patients with large septal defects and tetralogy of Fallot results in whole-body cyanosis.

- Differential clubbing and cyanosis: classic signs of patent ductus arteriosus with Eisenmenger syndrome.
- Differential clubbing and cyanosis: a pathognomonic finding in cardiology.



A 64-year-old man has had difficulty walking for 6 months, experiencing muscle cramps in his right thigh after walking a block on level ground. The cramps subside quickly with rest. He has also had decreased sexual performance. Medical history is significant for hypercholesterolemia and myocardial infarction. The patient smokes a pack of cigarettes daily but does not use alcohol or illicit drugs. He is afebrile. Blood pressure is 128/78 mm Hg, pulse is 76/min, and respirations are 16/min. Cardiac auscultation reveals normal S1 and S2, and equal vesicular breath sounds are heard over the bilateral lungs. The abdomen is soft and without masses. There is no peripheral edema, muscle tenderness, or muscle weakness. Which of the following is the most likely cause of this patient's symptoms?

- ☐ A. Granulomatous inflammation of the arterial media
- ☐ B. Homogenous acellular thickening of the arteriolar walls
- ☐ C. Lipid-filled arterial intimal plaques
- ☐ D. Medial band-like arterial calcification
- ☐ E. Onion-like concentric thickening of the arteriolar walls

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A 64-year-old man has had difficulty walking for 6 months, experiencing muscle cramps in his right thigh after walking a block on level ground. The cramps subside quickly with rest. He has also had decreased sexual performance. Medical history is significant for hypercholesterolemia and myocardial infarction. The patient smokes a pack of cigarettes daily but does not use alcohol or illicit drugs. He is afebrile. Blood pressure is 128/78 mm Hg, pulse is 76/min, and respirations are 16/min. Cardiac auscultation reveals normal S1 and S2, and equal vesicular breath sounds are heard over the bilateral lungs. The abdomen is soft and without masses. There is no peripheral edema, muscle tenderness, or muscle weakness. Which of the following is the most likely cause of this patient's symptoms?

- ☐ A. Granulomatous inflammation of the arterial media [5%]
- ☐ B. Homogenous acellular thickening of the arteriolar walls [5%]
- ☒ C. Lipid-filled arterial intimal plaques [74%]
- ☐ D. Medial band-like arterial calcification [6%]
- ☐ E. Onion-like concentric thickening of the arteriolar walls [7%]

Omitted

Correct answer
C74%
Answered correctly2 Seconds
Time Spent01/29/2019
Last Updated

Explanation

This patient's muscle pain, which occurs with exercise and remits with rest, is consistent with **intermittent claudication**; smoking is an important risk factor. Claudication is usually due to atherosclerosis of the large arteries, specifically resulting from fixed stenoses caused by lipid-filled

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Text Zoom

Explanation

This patient's muscle pain, which occurs with exercise and remits with rest, is consistent with **intermittent claudication**; smoking is an important risk factor. Claudication is usually due to atherosclerosis of the large arteries, specifically resulting from fixed stenoses caused by lipid-filled intimal plaques that bulge into the arterial lumen (**atheromas**). These stenoses prevent a sufficient increase in blood flow to muscles during exercise, leading to ischemic muscle pain. The pain is rapidly **relieved by rest**, as residual blood flow is adequate to meet the metabolic demands of resting, but not exercising, muscle.

Thigh claudication is suggestive of occlusive disease of the ipsilateral **external iliac** artery or its more distal branches (ie, common femoral, superficial femoral, profunda femoris arteries). Accompanying **impotence** and/or **gluteal claudication** suggests more proximal **aortoiliac occlusion** (so-called **Leriche syndrome**), which, in addition to affecting the external iliac artery, also diminishes blood flow to the internal pudendal and gluteal branches of the internal iliac artery.

(Choice A) **Granulomatous inflammation** of the arterial media occurs in temporal (giant cell) arteritis, the most common form of systemic vasculitis in adults. This condition typically affects patients age >50 and causes headache, facial pain, jaw claudication, and visual loss.

(Choice B) Hyaline arteriosclerosis involves homogeneous deposition of hyaline material in the intima and media of small arteries and arterioles, typically in patients with hypertension or diabetes mellitus (both absent in this patient). The underlying structure of the vessel wall is maintained, but the intima is thickened and the arterial lumen narrowed.

(Choice D) Medial band-like calcifications occur in **medial calcific sclerosis** (Mönckeberg arteriosclerosis). The condition is characterized by calcified deposits in muscular arteries and typically affects individuals age >50. Although visible radiologically and often palpable on physical examination, these calcifications are clinically asymptomatic because they do not narrow the vessel lumen.

(Choice E) **Onion-like concentric thickening** of arteriolar walls due to layers of smooth muscle cells and reduplicated basement membrane is seen in hyperplastic arteriosclerosis, which can occur in severe chronic hypertension (eg, blood pressure >180/120 mm Hg). Although arterioles in all tissues may be affected, those in the kidneys, retinas, and intestines are most common; skeletal muscle is less likely to be symptomatically

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Thigh claudication is suggestive of occlusive disease of the ipsilateral **external iliac** artery or its more distal branches (ie, common femoral, superficial femoral, profunda femoris arteries). Accompanying **impotence** and/or **gluteal claudication** suggests more proximal **aortoiliac occlusion** (so-called **Leriche syndrome**), which, in addition to affecting the external iliac artery, also diminishes blood flow to the internal pudendal and gluteal branches of the internal iliac artery.

(Choice A) **Granulomatous inflammation** of the arterial media occurs in temporal (giant cell) arteritis, the most common form of systemic vasculitis in adults. This condition typically affects patients age >50 and causes headache, facial pain, jaw claudication, and visual loss.

(Choice B) Hyaline arteriolosclerosis involves homogeneous deposition of hyaline material in the intima and media of small arteries and arterioles, typically in patients with hypertension or diabetes mellitus (both absent in this patient). The underlying structure of the vessel wall is maintained, but the intima is thickened and the arterial lumen narrowed.

(Choice D) Medial band-like calcifications occur in **medial calcific sclerosis** (Mönckeberg arteriosclerosis). The condition is characterized by calcified deposits in muscular arteries and typically affects individuals age >50. Although visible radiologically and often palpable on physical examination, these calcifications are clinically asymptomatic because they do not narrow the vessel lumen.

(Choice E) **Onion-like concentric thickening** of arteriolar walls due to layers of smooth muscle cells and reduplicated basement membrane is seen in hyperplastic arteriolosclerosis, which can occur in severe chronic hypertension (eg, blood pressure >180/120 mm Hg). Although arterioles in all tissues may be affected, those in the kidneys, retinas, and intestines are most common; skeletal muscle is less likely to be symptomatically involved.

Educational objective:

Intermittent claudication describes muscle pain that is reproducibly caused by exercise and relieved by rest; it occurs due to atherosclerotic stenoses (lipid-filled intimal plaques) in the large arteries that prevent sufficient blood flow to exercising muscle. The lower extremities are most commonly affected; however, proximal lesions (ie, aortoiliac occlusion) can cause gluteal claudication and/or impotence.

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Correct answer

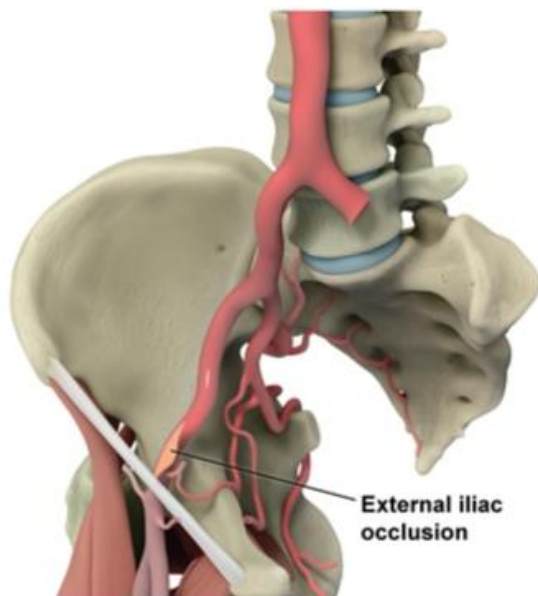
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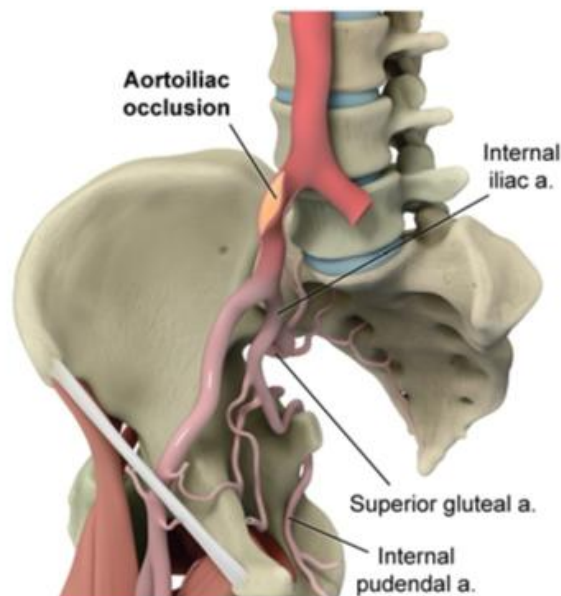
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Exhibit Display

Thigh claudication



External iliac occlusion

Thigh claudication +
gluteal claudication & impotence
(Leriche syndrome)

Aortoiliac occlusion

Internal iliac a.

Superior gluteal a.

Internal pudendal a.

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Add To Flash Card

(Choice E) Onion-like concentric thickening of arteriolar walls due to layers of smooth muscle cells and reduplicated basement membrane is

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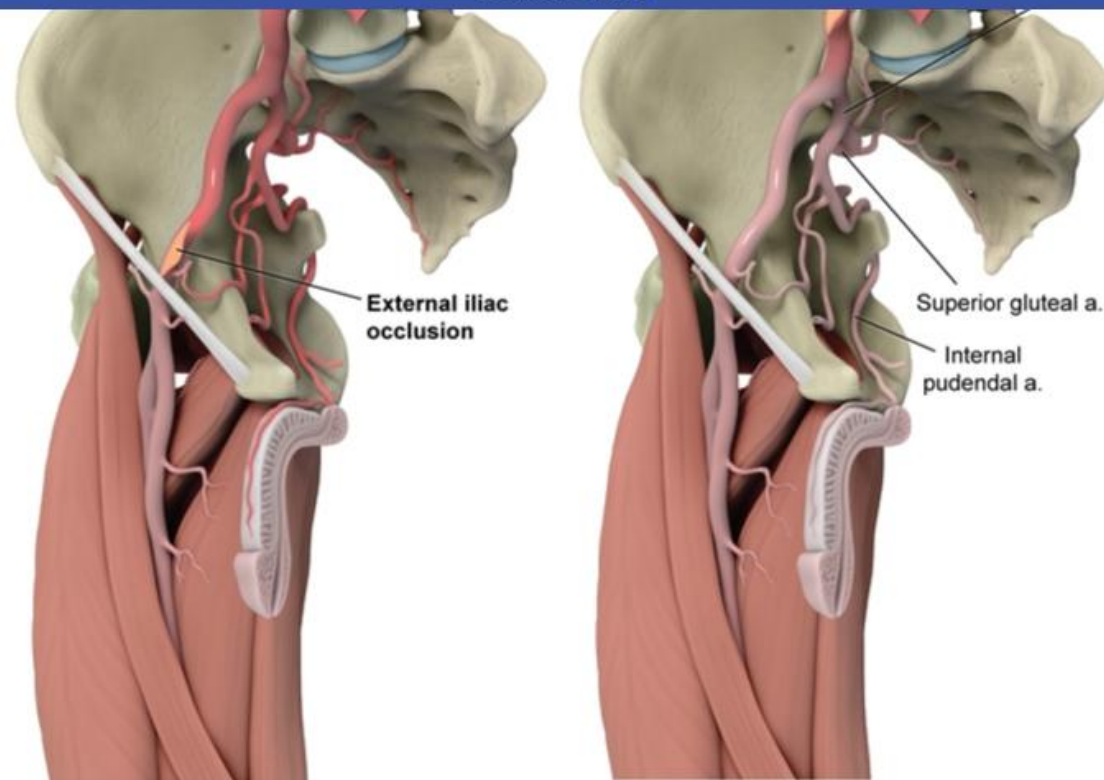
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(Choice E) **Onion-like concentric thickening** of arteriolar walls due to layers of smooth muscle cells and reduplicated basement membrane is

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Text Zoom

A 32-year-old Caucasian male experiences fever, weight loss, myalgias, and abdominal pain. His blood pressure is 150/90 mm Hg, and his heart rate is 90/min. If diagnosed with polyarteritis nodosa (PAN), which of the following arteries will most likely be spared by the disease?

☐ A. Renal

☐ B. Coronary

☐ C. Pulmonary

☐ D. Mesenteric

☐ E. Cutaneous

☐ F. Hepatic

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A 32-year-old Caucasian male experiences fever, weight loss, myalgias, and abdominal pain. His blood pressure is 150/90 mm Hg, and his heart rate is 90/min. If diagnosed with polyarteritis nodosa (PAN), which of the following arteries will most likely be spared by the disease?

☐ A. Renal [5%]

☐ B. Coronary [11%]

☒ C. Pulmonary [57%]

☐ D. Mesenteric [4%]

☐ E. Cutaneous [16%]

☐ F. Hepatic [4%]

Omitted

Correct answer C

57% Answered correctly

2 Seconds Time Spent

02/06/2019 Last Updated

Explanation

PAN is a segmental, transmural, necrotizing inflammation of medium to small sized arteries in any organ. The kidneys, heart, liver, and gastrointestinal tract are the most commonly involved organs. As for the lungs, PAN typically spares pulmonary arteries and rarely involves bronchial arteries. The inflammation of arteries can result in ischemia, infarction, or hemorrhage. There is also a propensity for bead-like aneurysm formation, especially in the mesenteric circulation. Up to one third of patients experience cutaneous manifestations, including livedo

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☐

D. Mesenteric [4%]

☐

E. Cutaneous [16%]

☐

F. Hepatic [4%]

Omitted

Correct answer
C

57%

Answered correctly

2 Seconds

Time Spent

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Last Updated

Explanation

PAN is a segmental, transmural, necrotizing inflammation of medium to small sized arteries in any organ. The kidneys, heart, liver, and gastrointestinal tract are the most commonly involved organs. As for the lungs, PAN typically spares pulmonary arteries and rarely involves bronchial arteries. The inflammation of arteries can result in ischemia, infarction, or hemorrhage. There is also a propensity for bead-like aneurysm formation, especially in the mesenteric circulation. Up to one-third of patients experience cutaneous manifestations, including livedo reticularis (a purplish network-patterned discoloration) and palpable purpura (small 1 to 1.5 cm infarcts, sometimes with ulceration).

Educational Objective:

PAN is segmental, transmural, necrotizing inflammation of medium- to small-sized arteries. Renal artery involvement is often prominent. Vessels of the kidneys, heart, liver, and gastrointestinal tract are most commonly involved in resulting ischemia, infarction, or hemorrhage. Cutaneous manifestations occur in up to one-third of patients, and include palpable purpura. The lung is very rarely involved.

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A 32-year-old man is brought to the emergency department with a several-day history of high-grade fever, dyspnea, and fatigue. The patient's temperature is 39.4 C (102.9 F), blood pressure is 122/70 mm Hg, and pulse is 102/min and regular. Physical examination reveals a new holosystolic murmur with a blowing quality that is best heard over the cardiac apex. Skin examination shows nontender lesions on the sole of the feet, as shown in the image below.

Which of the following do these lesions most likely represent?

☐ A. Areas of local vascular growth

☐ B. Foci of granulomatous inflammation

☐ C. Immune complex-mediated vascular lesions

☐ D. Microemboli to skin vessels

☐ E. Primary infection foci

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feet, as shown in the image below.



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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Store, Mail, and various background applications.

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☐ D. Microemboli to skin vessels

☐ E. Primary infection foci

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Which of the following do these lesions most likely represent?

- Omitted
Correct answer
D

74%
Answered correctly

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Explanation

This patient's presentation (fever, dyspnea, and a new blowing holosystolic murmur best heard at the cardiac apex) is suggestive of mitral regurgitation likely due to **infective endocarditis** (IE). Patients with IE can develop several peripheral cutaneous or mucocutaneous stigmata of endocarditis including petechiae, **splinter hemorrhages**, Roth spots, Janeway lesions (see above Image), and **Osler nodes**.

Janeway lesions are nontender, macular, and erythematous lesions typically located on the palms and soles. They are the result of septic embolization from valvular vegetations and are composed of bacteria, neutrophils (microabscesses), necrotic material, and subcutaneous hemorrhage.

(Choice A) A neoplastic proliferation of cells of vascular origin (eg, Kaposi sarcoma) cannot be ruled out based on the appearance of this patient's skin lesions alone. However, the overall clinical presentation is highly suggestive of bacterial endocarditis as the unifying diagnosis.

(Choice B) Granulomatous inflammation can produce cutaneous lesions in miliary tuberculosis, sarcoidosis, and leprosy. However, the lesions do not localize preferentially to the soles of the feet.

(Choice C) Osler nodes are tender, violaceous nodules typically located in the pulp of fingers and toes. The pathogenesis of Osler nodes is immune-complex deposition in the skin.

(Choice E) The lesions on the sole of the foot could represent primary foci of infection as might occur following focal skin abrasions/puncture or from sporotrichosis after walking barefoot. However, this patient's symptoms are most suggestive of mitral valve IE.

Educational objective:

Janeway lesions are nontender, macular, and erythematous lesions typically located on the palms and soles of patients with acute infective endocarditis and are the result of septic embolization from valvular vegetations.

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A 54-year-old Caucasian male comes to the emergency room with retrosternal chest pain of 30 minutes duration. The patient also complains of sweating and mild dyspnea. A single tablet of nitroglycerin is delivered sublingually, and the patient's pain decreases significantly. The patient has experienced several similar episodes of pain over the last 12 hours, all of which resolved spontaneously. Which of the following ultrastructural changes would most likely indicate irreversible myocardial cell injury in this patient?

- ☐ A. Myofibril relaxation
- ☐ B. Disaggregation of polysomes
- ☐ C. Mitochondrial vacuolization
- ☐ D. Disaggregation of nuclear granules
- ☐ E. Triglyceride droplet accumulation
- ☐ F. Glycogen loss

Submit

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- ☐ A. Myofibril relaxation [3%]
- ☐ B. Disaggregation of polysomes [3%]
- ☒ C. Mitochondrial vacuolization [59%]
- ☐ D. Disaggregation of nuclear granules [28%]
- ☐ E. Triglyceride droplet accumulation [2%]
- ☐ F. Glycogen loss [2%]

Correct answer
C

 59%
Answered correctly

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Explanation

The appearance of vacuoles and phospholipid-containing amorphous densities within mitochondria generally signifies irreversible injury, and

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The appearance of vacuoles and phospholipid-containing amorphous densities within mitochondria generally signifies irreversible injury, and implies a permanent inability to generate further ATP via oxidative phosphorylation. When the mitochondria are injured irreversibly, the cell cannot recover. Simple mitochondrial swelling may be associated with reversible cellular injury, however.

(Choice A) Myofibril relaxation is an early sign of *reversible* injury in cardiac myocytes, which occurs within the first 30 minutes of severe ischemia. Myofibril relaxation corresponds with intracellular ATP depletion and lactate accumulation due to anaerobic glycolysis during this period.

(Choice B) Disaggregation of polysomes denotes the dissociation of rRNA from mRNA in *reversible* ischemic/hypoxic injury. Depletion of intracellular ATP is thought to promote the dissolution of polysomes into monosomes as well as the detachment of ribosomes from the rough endoplasmic reticulum. Disaggregation of polysomes results in impaired protein synthesis.

(Choice D) Disaggregation of granular and fibrillar elements of the nucleus is associated with *reversible* cell injury. Another common nuclear change associated with reversible cell injury is clumping of nuclear chromatin, perhaps secondary to a decrease in intracellular pH.

(Choice E) Triglyceride droplet accumulation is characteristic of *reversible* cell injury, especially in hepatocytes, and also in striated muscle cells and renal cells. This fatty change may result from the decreased synthesis of intracellular proteins that occurs with cell injury. In the hepatocyte, decreased production of lipid acceptor proteins prevents the normal incorporation of triglycerides into lipoproteins. Since triglycerides cannot be rapidly exported from the cell in the form of lipoproteins, they accumulate intracellularly.

(Choice F) Glycogen loss is another early and *reversible* cellular response to injury. As a result of lowered mitochondrial ATP production, ATP must be supplied to the cell via anaerobic glycolysis of glucose derived from the cell's glycogen stores. Myocardial glycogen stores may be completely depleted within 30 minutes of the onset of severe ischemia.

Educational Objective:

Mitochondrial vacuolization is typically a sign of irreversible cell injury, signifying that the involved mitochondria are permanently unable to generate ATP.

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A 30-year-old man is brought to the emergency department due to left-sided weakness. His left arm was sluggish after awakening, and he had difficulty walking to the bathroom. The patient has no known medical conditions but has a 2-month history of positional dyspnea. Vital signs are within normal limits. On examination, strength of the left upper and left lower extremity is reduced, and touch/temperature sensation is similarly diminished. Cardiovascular examination reveals an intermittent murmur at the apex without carotid bruits. Echocardiography shows a mobile, 3-cm left atrial mass obstructing the mitral opening during diastole. Histologic examination of this mass would most likely show which of the following findings?

☐

A. Amorphous extracellular matrix

☐

B. Malignant epithelial cells with necrotic debris

☐

C. Malignant vascular spindle cells

☐

D. Organized thrombus

☐

E. Well-organized, mature adipocytes

Submit

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A 30-year-old man is brought to the emergency department due to left-sided weakness. His left arm was sluggish after awakening, and he had difficulty walking to the bathroom. The patient has no known medical conditions but has a 2-month history of positional dyspnea. Vital signs are within normal limits. On examination, strength of the left upper and left lower extremity is reduced, and touch/temperature sensation is similarly diminished. Cardiovascular examination reveals an intermittent murmur at the apex without carotid bruits. Echocardiography shows a mobile, 3-cm left atrial mass obstructing the mitral opening during diastole. Histologic examination of this mass would most likely show which of the following findings?

- ☒ A. Amorphous extracellular matrix [66%]
- ☐ B. Malignant epithelial cells with necrotic debris [7%]
- ☐ C. Malignant vascular spindle cells [8%]
- ☐ D. Organized thrombus [14%]
- ☐ E. Well-organized, mature adipocytes [2%]

Omitted

Correct answer
A66%
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Explanation

This patient's left atrial mass on echocardiography is consistent with an **atrial myxoma**; this benign tumor is the most common primary cardiac

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This patient's left atrial mass on echocardiography is consistent with an **atrial myxoma**; this benign tumor is the most common primary cardiac neoplasm. Approximately 80% originate in the **left atrium**, and they can present with **systemic embolization** (eg, stroke) from tumor fragments passing into the systemic circulation. The tumors may also lead to constitutional symptoms (eg, fatigue, weight loss, low-grade fever) resulting from cytokine release, and they may cause transient mitral valve obstruction, leading to symptoms that can **mimic mitral valve stenosis** (eg, dyspnea, orthopnea, hemoptysis).

Physical examination often reveals an intermittent or positional **mid-diastolic murmur** ("tumor plop") that results from the motion of the tumor mass obstructing the mitral valve orifice. Histopathologic examination of a myxoma reveals **amorphous extracellular matrix** with scattered stellate or globular myxoma cells within abundant mucopolysaccharide (myxoid) ground substance containing chondroitin sulfate and hyaluronic acid. Because of their high vascularity, these tumors often demonstrate areas of hemorrhage accompanied by brown, hemosiderin-laden macrophages.

(Choices B and C) Primary malignant cardiac tumors are quite rare; those that occur are usually either sarcomas or lymphomas. Malignant vascular spindle cells suggest primary cardiac angiosarcoma, which usually arises in the right atrium and is exceedingly rare. Malignant epithelial cells with necrotic debris suggest secondary involvement of the heart from a metastatic malignancy (eg, melanoma, lung cancer, breast cancer).

(Choice D) An organized thrombus can form in the left atrium or left atrial appendage in patients with atrial flutter or fibrillation and may cause stroke due to systemic embolization. However, thrombi are typically adherent to the cardiac endothelium; a mobile mass in the left atrium is more characteristic of a myxoma.

(Choice E) Lipomas are benign tumors composed of well-organized, mature adipocytes. They are the second most common type of primary cardiac neoplasm; however, unlike myxomas, lipomas rarely embolize.

Educational objective:

Myxomas are the most common primary cardiac neoplasm, and approximately 80% originate in the left atrium. Patients may have systemic embolization (eg, stroke) or symptomatic mitral valve obstruction that may be worse with certain body positions. Histopathologic examination reveals amorphous extracellular matrix with scattered stellate or globular myxoma cells within abundant mucopolysaccharide ground substance.

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Educational objective:

Myxomas are the most common primary cardiac neoplasm, and approximately 80% originate in the left atrium. Patients may have systemic embolization (eg, stroke) or symptomatic mitral valve obstruction that may be worse with certain body positions. Histopathologic examination reveals amorphous extracellular matrix with scattered stellate or globular myxoma cells within abundant mucopolysaccharide ground substance.

References

- Histopathologic and clinical characterization of cardiac myxoma: review of 53 cases from a single institution.
- Cardiac myxomas: 24 years of experience in 49 patients.
- Pathology of surgically excised primary cardiac tumors.

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A 68-year-old man is brought to the emergency department with chest pain. For the last year, the patient has had exertional chest pain that has progressively worsened. He was shoveling snow this morning when the pain became unbearable. He has smoked a pack of cigarettes daily for 40 years. Blood pressure is 140/100 mm Hg and pulse is 90/min. Examination showed mild diaphoresis. The lungs are clear to auscultation. ECG shows ST-and T-wave changes suggestive of ischemia. Cardiac enzymes are elevated. Emergent coronary angiography is performed, which demonstrates significant atherosclerotic involvement of the left anterior descending and circumflex arteries. Development of these vascular lesions most likely involved growth factors released from which of the following sources?

A. B lymphocytes

B. Eosinophils

C. Erythrocytes

D. Mast cells

E. Neutrophils

F. Platelets

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System Tray



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- ☐ A. B lymphocytes [2%]
- ☐ B. Eosinophils [0%]
- ☐ C. Erythrocytes [1%]
- ☐ D. Mast cells [3%]
- ☐ E. Neutrophils [23%]
- ☒ F. Platelets [68%]

Omitted

Correct answer
F68%
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Explanation

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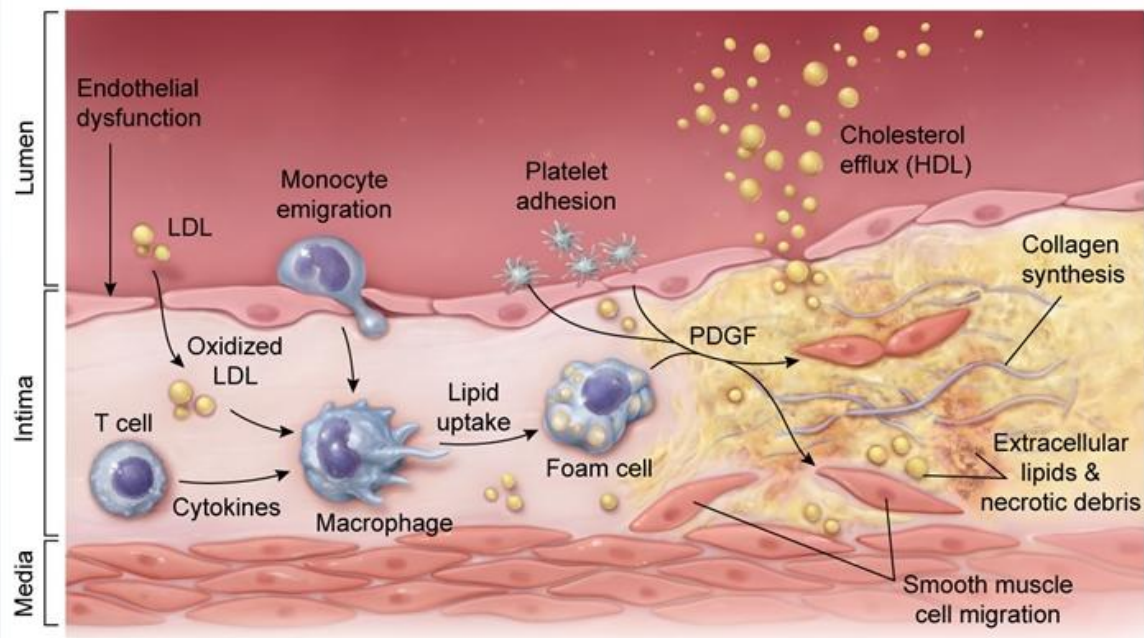
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Explanation

Formation of atheroma



PDGF = platelet-derived growth factor.

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The development of atherosclerotic plaque, or **atheroma**, involves a multistep process:

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PDGF = platelet-derived growth factor.

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The development of atherosclerotic plaque, or **atheroma**, involves a multistep process:

Initially, factors including hyperlipidemia, hypertension, hyperglycemia, and smoking trigger **endothelial injury and/or dysfunction**. This leads to increased vascular permeability, enhanced leukocyte adhesion, and a higher propensity for thrombosis.

Lipoproteins (ie, LDL and oxidized LDL) enter the arterial wall intima and begin to accumulate. Monocytes adhere to the endothelial wall and enter into the intima as well; these cells transform into macrophages and engulf lipid particles to become foam cells. **Platelets** adhere to the abnormal endothelium and become activated.

Growth factors, namely **platelet-derived growth factor (PDGF)**, are released from platelets, **activated macrophages**, and endothelial cells. This triggers **smooth muscle cell (SMC)** recruitment from the media and **proliferation** in the intima.

Over time, progressive SMC proliferation and accumulation of necrotic debris (due to macrophage/foam cell and SMC death) lead to growth of the atheroma. SMCs encourage plaque stability by synthesizing collagen, whereas activated inflammatory cells break down collagen and contribute to plaque instability. HDL likely extracts lipids from the intima back into the bloodstream and helps slow atheroma development.

(Choice A) B lymphocytes are not known to play a significant role in atheroma development. However, T lymphocytes in the arterial wall intima likely play a role in activating macrophages, endothelial cells, and SMCs by releasing inflammatory cytokines (eg, interferon-γ).

(Choices B and D) Eosinophils and mast cells are important in IgE-mediated (Type I) hypersensitivity reactions; mast cells release histamine in the early phase, and eosinophils release leukotrienes and major basic protein in the late phase of the reaction. However, these cells are not significantly involved in atheroma development.

(Choices C and E) Neither erythrocytes nor neutrophils release growth factors or play a significant role in atheroma development.

Educational objective:
During the development of atherosclerotic plaque (atheroma), activated macrophages, platelets, and endothelial cells release growth factors (eg, platelet-derived growth factor) that stimulate recruitment of smooth muscle cells from the arterial wall media and their subsequent proliferation in the intima.

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- Initially, factors including hyperlipidemia, hypertension, hyperglycemia, and smoking trigger **endothelial injury and/or dysfunction**. This leads to increased vascular permeability, enhanced leukocyte adhesion, and a higher propensity for thrombosis.
- Lipoproteins (ie, LDL and oxidized LDL) enter the arterial wall intima and begin to accumulate. Monocytes adhere to the endothelial wall and enter into the intima as well; these cells transform into macrophages and engulf lipid particles to become foam cells. **Platelets** adhere to the abnormal endothelium and become activated.
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