

1. What is the initial step in the pathogenesis of atherosclerosis?
 - a. Accumulation of the lipoproteins
 - b. Migration of monocytes into the intima
 - c. Chronic endothelial cell injury
 - d. Release of growth factors

2. Which molecules do dysfunctional injured endothelial cells express during atherosclerosis pathogenesis?
 - a. TNF- α
 - b. VCAM-1
 - c. IL-6
 - d. IL-10

3. What do monocytes transform into when they migrate into the intima during atherosclerosis?
 - a. T cells
 - b. Macrophages
 - c. Foam cells
 - d. B cells

4. What plays a significant role in stabilizing the atheroma and producing a fibrous cap in atherosclerosis?
 - a. Lipid accumulation
 - b. Collagen synthesis
 - c. Free radicals
 - d. Necrotic debris

5. Which vessels are most commonly involved in atherosclerosis?
 - a. Jugular veins
 - b. Femoral arteries
 - c. Coronary arteries
 - d. Pulmonary veins

6. What characterizes vulnerable plaques in atherosclerosis?
 - a. Thick fibrous caps
 - b. Dense collagen
 - c. Low of inflammatory cells
 - d. Minimal lipid accumulation

7. What type of complications can result from the rupture of the fibrous cap in atherosclerosis?
 - a. Stroke
 - b. Aneurysm
 - c. Thromboembolism
 - d. Gangrene

8. What leads to stable plaques in atherosclerosis?
 - a. Abundant extracellular lipid
 - b. Minimal inflammation
 - c. Large numbers of foam cells
 - d. Thin fibrous caps

9. What happens when the fibrous cap of a plaque in atherosclerosis ruptures?
 - a. Formation of thrombus
 - b. Vessel enlargement
 - c. Lipid synthesis
 - d. Aneurysm rupture

10. What is the consequence of the narrowing or complete occlusion of the arterial lumen in atherosclerosis progression?

- a. Decreased inflammation
- b. Increased artery elasticity
- c. Ischemic injury
- d. Thrombosis prevention

11. What can result from ulceration, fissuring, or erosion of a plaque's fibrous cap in atherosclerosis?

- a. Chronic ischemia
- b. Aneurysm formation
- c. Lipid accumulation
- d. Vessel dilation

12. Which condition can develop due to atheroma-induced pressure or ischemic atrophy of the arterial wall?

- a. Aortic dissection
- b. Aortic aneurysm
- c. Pulmonary embolism
- d. Deep vein thrombosis

13. What characteristic distinguishes an atheromatous aneurysm in atherosclerosis?

- a. Dense collagen
- b. Minimal lipid accumulation
- c. Thick fibrous cap
- d. Loss of elastic tissue

14. What role do activated T lymphocytes play in atherosclerosis?

- a. Formation of fibrous cap
- b. Production of free radicals
- c. Stimulation of growth factors
- d. Induction of foam cell formation

15. What factor leads to the recruitment of smooth muscle cells from the media during the pathogenesis of atherosclerosis?

- a. Lipid accumulation
- b. Growth factors
- c. Endothelial permeability
- d. Vascular occlusion

16. What results from the accumulation of oxidized LDL in macrophages and smooth muscle cells during atherosclerosis?

- a. Fatty streaks
- b. Vascular dilation
- c. Endothelial healing
- d. Reduced cholesterol levels

17. Which cells engulf oxidized LDL to become foam cells in atherosclerosis?

- a. T cells
- b. Macrophages
- c. Smooth muscle cells
- d. B cells

18. What process does the release of growth factors during atherosclerosis pathogenesis promote?

- a. Endothelial injury
- b. SMC proliferation
- c. Lipid accumulation
- d. Necrotic debris formation

19. What consequence does the thrombus formation have in atherosclerosis?

- a. Decreased vessel occlusion
- b. Increased arterial elasticity
- c. Systemic thromboembolism
- d. Plaque stabilization

20. How can atherosclerotic thrombi affect the arterial lumen?

- a. Cause aneurysm formation
- b. Lead to endothelial regeneration
- c. Result in partial or complete occlusion
- d. Induce smooth muscle cell death

21. What is the gold standard diagnostic test for atherosclerosis?

- a. MRI scan
- b. Blood test
- c. Invasive coronary angiography (ICA)
- d. Echocardiogram

22. Which of the following is a primary prevention strategy for atherosclerosis?

- a. Increasing LDL cholesterol levels
- b. Smoking cessation
- c. Sedentary lifestyle
- d. Poor diet

23. What cellular processes are involved in vascular remodeling in atherosclerosis?

- a. Cell division only
- b. Cell growth, cell death, cell migration, and extracellular matrix changes
- c. Cell migration and cell death
- d. Extracellular matrix changes only

24. Remodeling in atherosclerosis involves:

- a. Only expansive remodeling
- b. Both expansive and constrictive remodeling
- c. Constrictive remodeling only
- d. No remodeling at all

25. Which type of arteriosclerosis is seen in benign hypertension and DM?

- a. Hyaline arteriosclerosis
- b. Hyperplastic arteriosclerosis
- c. Monckeberg medial sclerosis
- d. Atherosclerotic Stenosis

26. What is the aim of secondary prevention programs for atherosclerosis?

- a. To delay plaque formation
- b. To prevent recurrence of IHD or stroke in symptomatic patients
- c. To initiate smoking habits
- d. To increase LDL cholesterol levels

27. What is atheroembolism in atherosclerotic disease?

- a. Formation of blood clots
- b. Production of microemboli composed of plaque contents
- c. Rupture of the aorta
- d. Enlargement of the vessel circumference

28. Why is arterial remodeling important in vascular pathology?

- a. It has no impact on vascular health
- b. It helps in maintaining the original vessel size
- c. It only occurs in younger individuals
- d. It is the primary cause of atherosclerosis

29. What contributes to thickening of the arterial intima in atherosclerosis?
- a. Migration and proliferation of vascular smooth muscle cells (VSMCs)
 - b. Removal of endothelial cells
 - c. Low cholesterol levels
 - d. Lack of physical activity
30. What outcomes depend on in atherosclerotic disease?
- a. Age of the patient
 - b. Size of the affected vessel, size, and stability of plaques
 - c. Daily diet
 - d. Medication history

- 1c - Chronic endothelial cell injury
- 2b - VCAM-1
- 3c - Foam cells
- 4b - Collagen synthesis
- 5c - Coronary arteries
- 6a - Thick fibrous caps
- 7c - Thromboembolism
- 8b - Minimal inflammation
- 9a - Formation of thrombus
- 10c - Ischemic injury
- 11a - Chronic ischemia
- 12b - Aortic aneurysm
- 13d - Loss of elastic tissue
- 14c - Stimulation of growth factors
- 15b - Growth factors
- 16a - Fatty streaks
- 17b - Macrophages
- 18b - SMC proliferation
- 19c - Systemic thromboembolism
- 20c - Result in partial or complete occlusion
- 21c. Invasive coronary angiography (ICA)
- 22b. Smoking cessation
- 23b. Cell growth, cell death, cell migration, and extracellular matrix changes
- 24b. Both expansive and constrictive remodeling
- 25a. Hyaline arteriosclerosis
- 26b. To prevent recurrence of IHD or stroke in symptomatic patients
- 27b. Production of microemboli composed of plaque contents
- 28b. It helps in maintaining the original vessel size
- 29a. Migration and proliferation of vascular smooth muscle cells (VSMCs)
- 30b. Size of the affected vessel, size, and stability of plaque