Pathology - Topic 26 - Cardiovascular system

Arteriosclerosis and Atherosclerosis

- **Arteriosclerosis** is a general term for thickening and loss of elasticity of arterial walls.
- Atherosclerosis is one important form, but the two words are not interchangeable.

Forms of Arteriosclerosis

1. Atherosclerosis

- Atherosclerosis affects the intima of large and medium-sized arteries.
- Its raised plaques contain a lipid-rich necrotic core covered by a fibrous cap.
    - The abdominal aorta, coronary arteries, carotid arteries and large arteries of the legs are common sites.

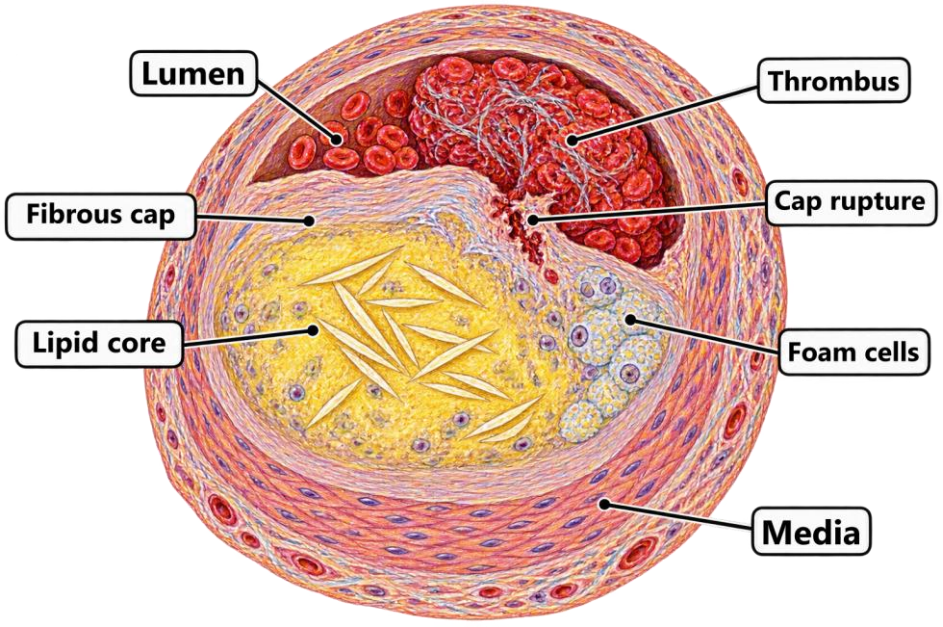
2. Arteriolosclerosis

- Arteriolosclerosis affects small arteries and arterioles.
- Hyaline and hyperplastic patterns are especially important in hypertension and kidney disease.
    - Hyaline thickening appears as smooth pink material in vessel walls.
    - Hyperplastic change produces concentric wall thickening with a narrowed lumen.

3. Medial Calcific Sclerosis

- Medial calcific sclerosis involves calcium deposition in the media of muscular arteries.
- It may increase arterial stiffness without producing the typical lipid-rich intimal plaque.

Development of an Atheromatous Plaque



A complicated atheromatous plaque within an arterial wall

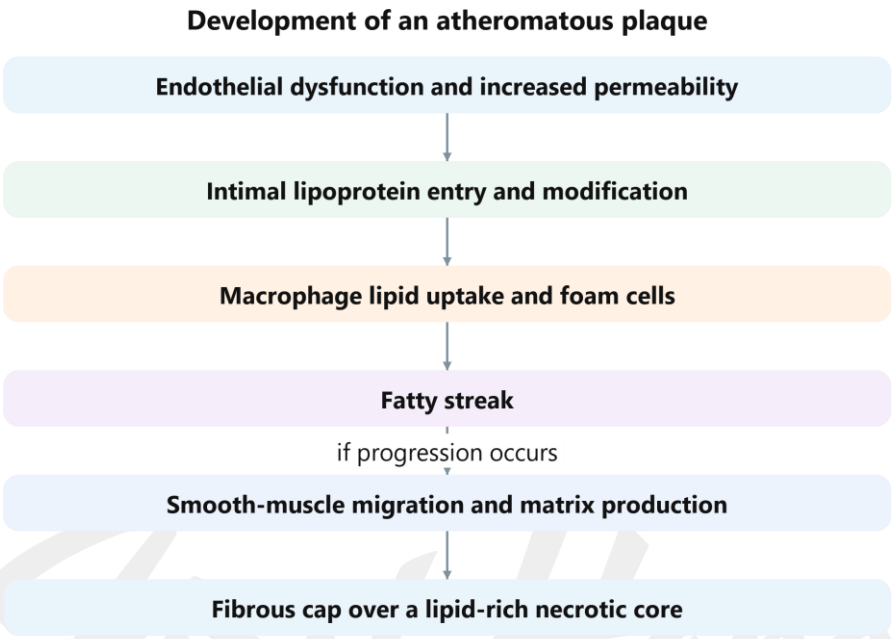
Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

Risk Factors and Endothelial Dysfunction

Risk Factors

- Important modifiable risks include high LDL cholesterol, hypertension, smoking and diabetes.
- Age and inherited susceptibility also influence risk.

Endothelial Dysfunction and Lipid Entry



- Disturbed flow and chemical injury alter endothelial function.
- The endothelium becomes more permeable and promotes the attachment of inflammatory cells.
- Lipoproteins enter the intima and become modified.
- Monocytes enter the wall and develop into macrophages that ingest lipid.
- Macrophage scavenger receptors take up modified lipoproteins, permitting lipid accumulation without the normal feedback limitation of ordinary LDL uptake.

Fatty Streak and Fibrofatty Plaque

Formation of the Fatty Streak

- Lipid-filled macrophages are called **foam cells**.
- Their accumulation forms a fatty streak, which may be present long before a complicated plaque develops.

Formation and Stability of the Fibrous Cap

- Smooth-muscle cells move into the intima, multiply and produce extracellular matrix.
- Cell death and retained lipid gradually form a necrotic core.
- The fibrous cap contains smooth-muscle cells and collagen.
- The shoulder region often contains inflammatory cells.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

- A thick cap with abundant collagen is relatively resistant to rupture. A thin inflamed cap over a large lipid core is more vulnerable, although plaque behaviour is not predicted by one feature alone.

Plaque Core

- Dissolved cholesterol crystals leave needle-like clefts among lipid, cellular debris and foam cells in routine sections.

Complicated Plaques

- Plaque rupture or surface erosion exposes thrombogenic material and can trigger an acute clot.
- The clot may partly or completely obstruct blood flow.
- Bleeding into a plaque can enlarge it suddenly.
- Calcification, ulceration and embolisation of plaque material are other complications.
    - Damage to the underlying media may weaken an artery and contribute to aneurysm formation.

Clinical Consequences

- Cholesterol embolisation from a plaque can leave similar clefts in small downstream arteries, producing ischaemic injury without a large thromboembolus.

Aneurysms and Aortic Dissection

- An aneurysm is an abnormal localised dilatation of a vessel or heart wall, whereas aortic dissection involves blood tracking between layers of the aortic wall.

Structural Types of Aneurysm

1. True Aneurysm

- A true aneurysm is a localised abnormal dilatation involving the arterial wall or a thinned ventricular wall.

Aneurysm Shape

- It may be fusiform or saccular in shape.
- A fusiform aneurysm expands much of the vessel circumference. A saccular aneurysm forms a more localised outpouching.

Abdominal and Thoracic Aortic Disease

- Abdominal aortic aneurysms commonly occur below the renal arteries and are associated with smoking, age and atherosclerotic disease.
- Inflammation and loss of elastic and collagen support weaken the wall. A mural thrombus commonly lines the dilated lumen.
- Thoracic aortic aneurysms may accompany hypertension, inherited connective-tissue disorders or bicuspid aortic valve-associated aortopathy.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

- Syphilitic aortitis is a classical but now uncommon cause.
- Syphilitic aortitis classically causes inflammation and scarring of the vasa vasorum, producing ischaemic injury and weakening of the ascending aortic media.

2. Pseudoaneurysm

- A pseudoaneurysm forms when blood escapes through a wall defect but remains contained by surrounding tissues.
- Its cavity communicates with the vessel lumen.
  - Trauma, procedures and breakdown of a vascular anastomosis are recognised causes.

Aortic Dissection

- Aortic dissection occurs when blood enters the aortic media and separates its layers.
- An intimal tear often provides the entry point.
  - Hypertension and inherited defects of connective tissue increase susceptibility.
  - Dissection can occur without a pre-existing aneurysm.
- Dissection may obstruct branch vessels or rupture into the pericardium, pleural cavity or other spaces.
- Proximal involvement can damage the aortic valve or coronary blood supply.

Complications and Mechanical Effects

Mechanical and Thromboembolic Complications

- Aneurysms can rupture, compress nearby structures or produce thromboemboli.
- Aortic root dilatation can cause aortic regurgitation.

Wall Tension and Expansion Risk

- Increasing radius raises wall tension for a given pressure.
- Wall weakness and expansion can therefore reinforce one another.
  - Risk depends on size, growth, location and cause; the same threshold should not be applied to every aneurysm.

Infected Aneurysms

- An infected aneurysm develops when infection weakens an arterial wall, sometimes after septic embolisation.
- The historical term mycotic aneurysm does not mean that the infection must be fungal.
  - Inflammatory destruction increases the risks of rapid expansion and rupture.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

Takayasu Arteritis

- **Takayasu arteritis** is a chronic granulomatous large-vessel vasculitis affecting the aorta and its major branches.

Vascular Distribution and Immune Mechanisms

- It commonly begins in young adults, especially women, but age and sex alone do not establish or exclude the diagnosis.
- The aortic arch branches, renal arteries and other aortic territories may be involved, and pulmonary arteries can also be affected.
- Immune-mediated inflammation damages the arterial wall and is followed by scarring, narrowing or occasionally aneurysmal dilatation.

Gross and Microscopic Morphology

1. Active Inflammatory Lesions

- Inflammation is prominent around the vasa vasorum and the junction of the media and adventitia.
- Lymphocytes, macrophages and sometimes multinucleated giant cells infiltrate the wall, with destruction of elastic fibres and smooth muscle.

2. Chronic and Healed Lesions

- Intimal thickening and fibrosis of the media and adventitia produce a thick, rigid artery with an irregularly narrowed or occluded lumen.
- Superimposed thrombosis may worsen obstruction, whereas severe medial destruction can cause an aneurysm.

Clinical Features and Consequences

- Early disease may cause fever, fatigue, weight loss and raised inflammatory markers.
- Stenosed arch vessels can cause arm claudication, unequal arm blood pressures, arterial bruits and weak or absent pulses, explaining the name **pulseless disease**.
- Carotid involvement can cause visual or neurological ischaemic symptoms, while renal-artery stenosis can cause hypertension.
- Aortic-root involvement may cause aortic regurgitation, and severe vascular disease can lead to stroke or heart failure.

Diagnosis and Treatment Principles

- Diagnosis combines clinical findings with vascular imaging, usually MR angiography or CT angiography, to demonstrate wall disease and its distribution.
- ESR and CRP support assessment but are nonspecific, and arterial biopsy is seldom available.
- Active disease is treated with glucocorticoids and an appropriate steroid-sparing immunosuppressive medicine under specialist care.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

FREE PREVIEW

Click here to visit our website

MBBS PREMIUM NOTES

Complete MBBS notes for every subject and year are available only in the FirstHope app.

Scan or click here

Click here to get the app >

Available on Google Play

Browse MBBS notes by year

firsthope.co.in/mbbs

Click here to explore MBBS >

Scan or click here

FirstHope Medical on Instagram

@firsthope.medical

Click here to follow

App and notes help

Find answers about access and downloads

Click here for help

YEAR 1 / YEAR 2 / YEAR 3 / YEAR 4