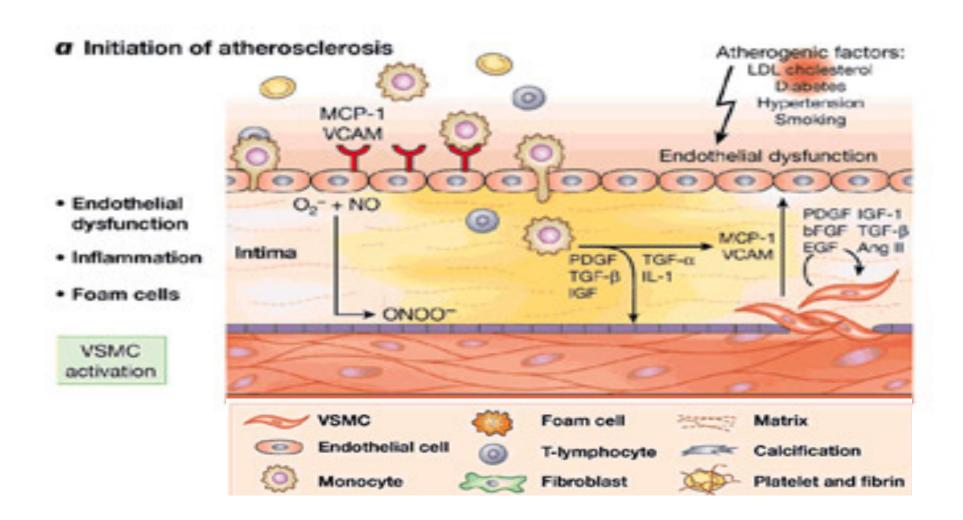
ATHEROSCLEROSIS-MORPHOLOGY & COMPLICATIONS

DR ANJALI GOYAL

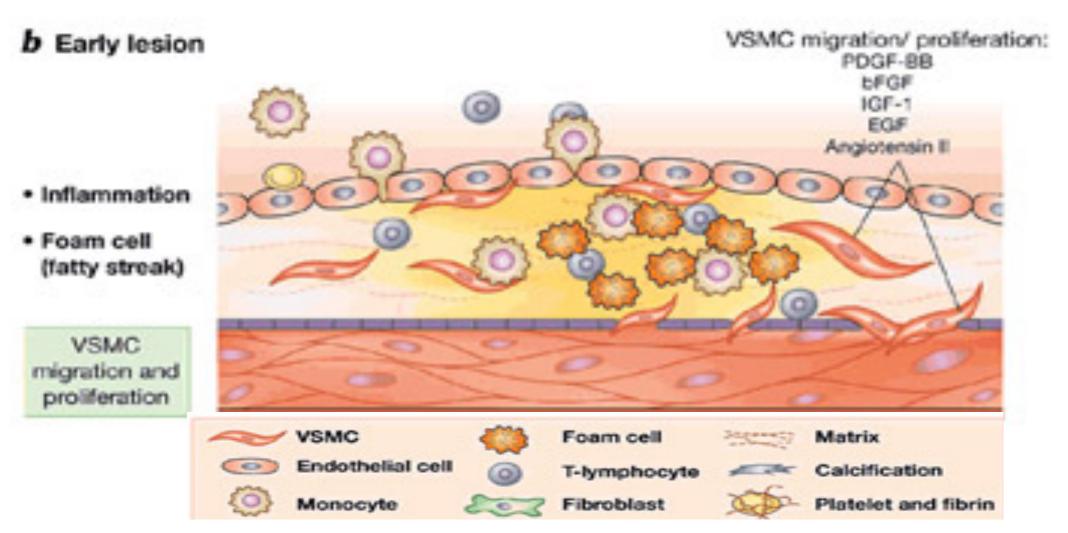
ATHEROSCLEROSIS- Morphology

- Fatty streak
- Simple/ Atheromatous plaque
- Complicated plaque

ATHEROSCLEROSIS- Morphology Initiation



ATHEROSCLEROSIS- Morphology Early Lesions



ATHEROSCLEROSIS- Morphology Early Lesions

Fatty Streaks/Gelatinous Lesions

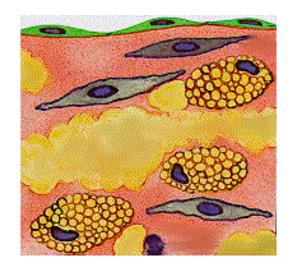
- Lipid Deposit in Intima
- Yellow, Slightly raised
- Can be seen in Aortas of infants < 1 year
- Possible precursors of Atheromas

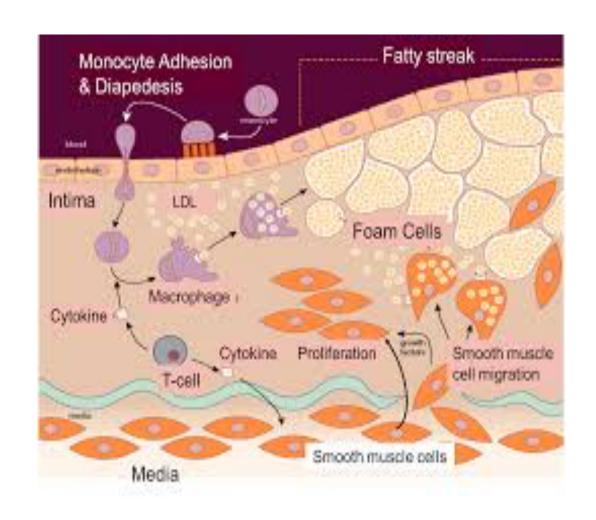




ATHEROSCLEROSIS- Early Lesions

- Early changes
 - proliferation of smooth muscle cells
 - accumulation of foam cells
 - extracellular lipid

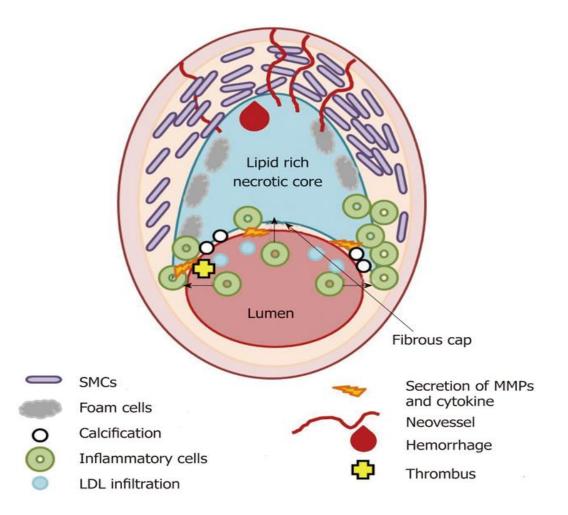




MORPHOLOGY

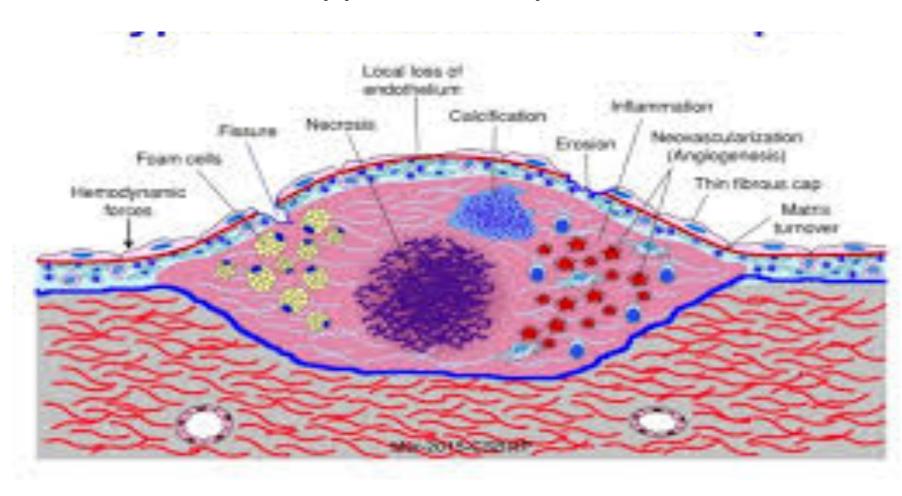
- Raised focal lesion in the intima which impinge on the lumen.
- Soft, yellow, grumous core of lipid (mainly cholesterol & cholesterol esters) covered by firm fibrous cap.
- Size; 0.3-1.5 cm- may coalese to form larger masses.
- Involvement mainly eccentric around the vessel wall.
- Initially focal/ sparse- may become more numerous & diffuse.

ATHEROMATOUS PLAQUES

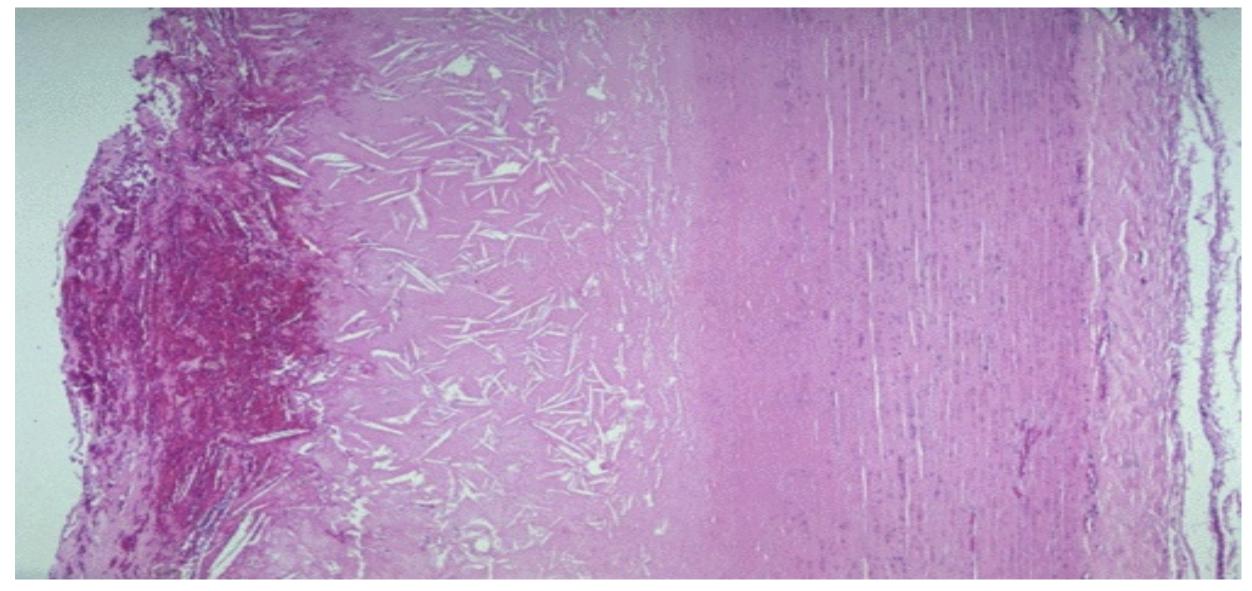


- Superficial fibrous cap Comprising of SMC & collagen. Beneath & to the side (shoulder) is a cellular area comprising of macrophages, smooth muscle cells & T- lymphocytes.
- Necrotic core- (Lying deep to the fibrous cap) comprising of lipids (Cholesterol & Cholesterol esters), clefts containing cholesterol, debris from dead cells, foam cells, fibrin, variably organized thrombus & plasma proteins.
- **Periphery** shows evidence of neovascularisation.
- Foam cells- These are large lipid laden cells derived from blood monocytes. SMC may also imbibe lipid to form foam cells.

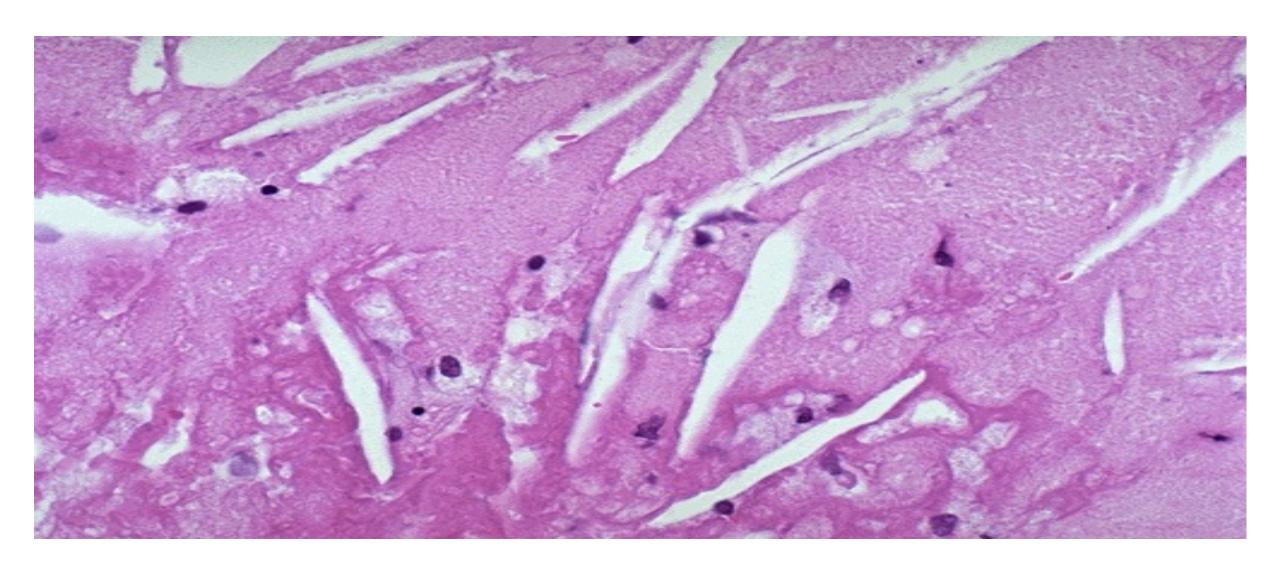
ATHEROSCLEROSIS- Morphology Typical Plaque



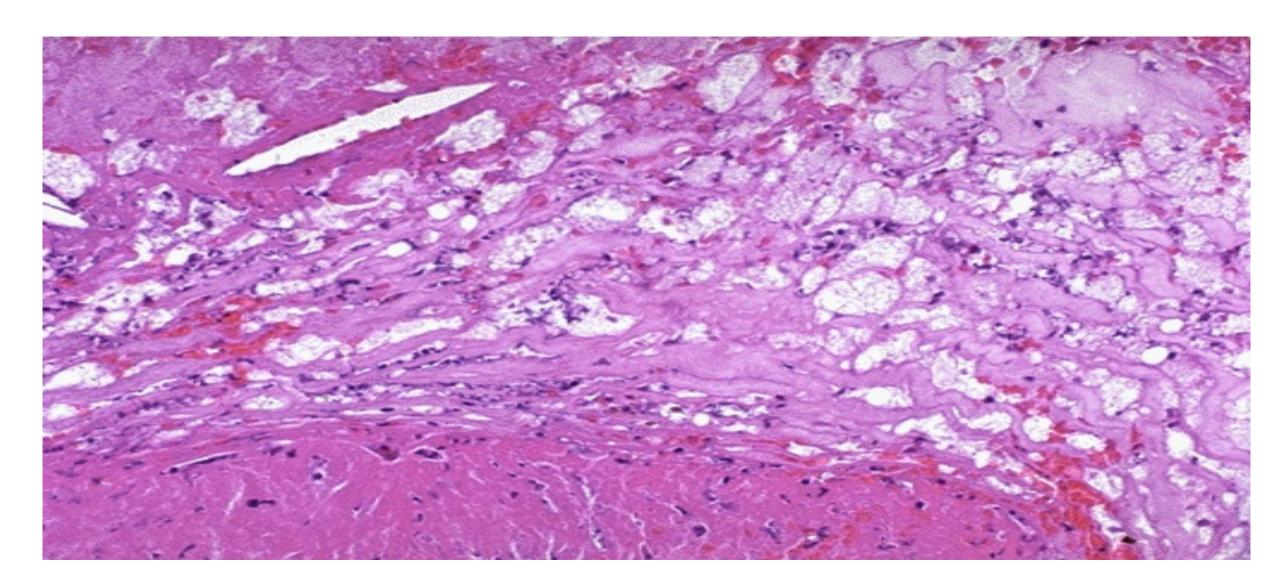
ATHEROMATOUS PLAQUES



ATHEROMATOUS PLAQUES-Cholesterol clefts



ATHEROMATOUS PLAQUES Foam cells



CLASSIFICATION OF HUMAN ATHEROSCLEROTIC LESIONS

- Type 1- (Initial lesion) Isolated macrophage foam cells
- Type 2- (Fatty streak) Mainly intracellular lipid deposition.
- Type 3 (Intermediate lesion) Type 2+ extracellular lipid pools
- Type 4 -(Atheroma) Type 2 + core of lipid
- Type 5-(Fibroatheroma) lipid core & fibrotic layers.
- Type 6 (Complicated lesion) Surface defect, hematoma, Haemorrhage, Thrombus.

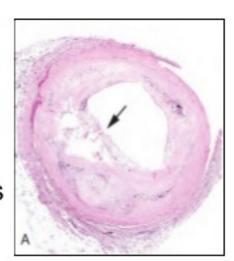
Atheroma - The Simple Plaque

- Raised yellow/white
- Irregular outline
- Widely distributed
- Enlarge and coalesce



ATHEROSCLEROSIS- Plaque Morphology

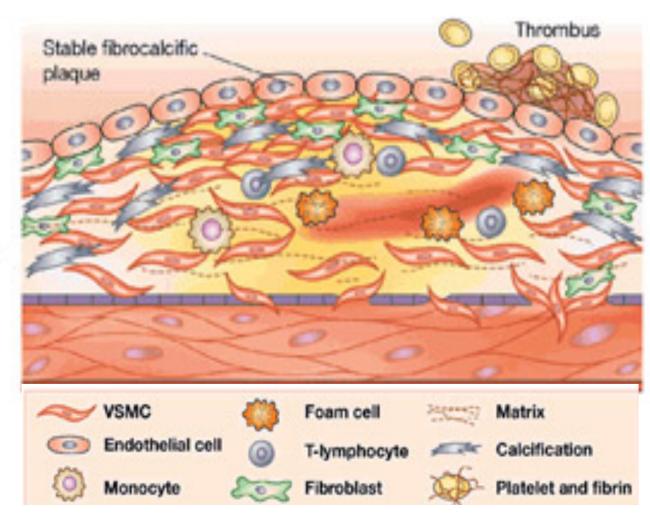
- Colour of plaque:
 - White-yellow patches
 - Red Brown: When ulcerated and superimposed by thrombus
- Involvement of the artery: Patchy
- Location: Eccentric (not circumferential)
- Size: Vary. May coalesce to form large masses
- Lesions at various stages often coexist
- Narrows the lumen of the artery



ATHEROSCLEROSIS- Morphology Advanced Lesion

VSMC abundance

- Fibroblasts and matrix
- Extracellular calcification

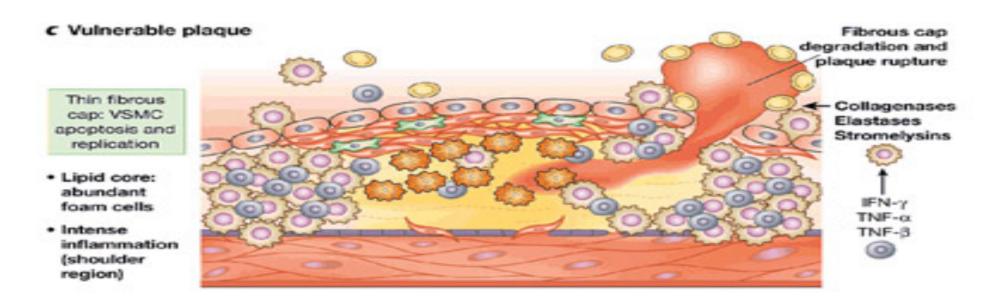


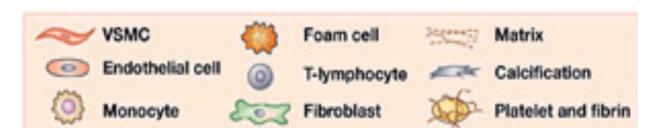
Atheroma - The Complicated Plaque

- Thrombosis
- Haemorrhage into plaque
- Calcification
- Aneurysm formation



Vulnerable Plaque - Morphology





ACUTE PLAQUE CHANGES

Acute coronary syndromes precipitated by abrupt changes in plaque followed by thrombosis.

Acute changes in plaque morphology include

- Fissuring
- H'ge into the plaque.
- Plaque rupture with embolisation of atheromatous debris into coronary vessels.
- Local plaque disruption -Increased risk of platelet aggregation & thrombosis.

DYNAMIC INSTABILITY OF PLAQUES

Distrupted plaques

- Eccentric (not uniform around the vessel circumference.)
- Large, soft core of necrotic debris and lipid.
- Thin, fibrous cap.
- Rich in macrophages & Tcells.

(Macrophages :-Secrete metalloproteinases which secrete collagen.

• (T-cells :- Activate macrophages)

DYNAMIC INSTABILITY OF PLAQUES

Hemodynamic Trauma

 Plaque tend to fissure at the function of fibrous cap and plaque free vessel wall; site where mechanical stresses induced by blood flow are maximal.

• Repeated "Silent" ruptures and thrombosis followed by organization plays an important role in the progression of atherosclerosis.

ATHEROSCLEROSIS- Morphology Complicated Lesions

- Rupture , Ulcerartion or Erosion of Luminal Surface
- Plaque superimposed with Thrombus
- Calcification
- Aneurysm



Atheroma - Common Sites

- Aorta especially abdominal
- Coronary arteries
- Carotid arteries
- Cerebral arteries
- Leg arteries

DISTRIBUTION OF LESIONS (order of frequency)

- Lower abdominal Aorta(around ostia of major branches)
- Coronary arteries
- Popliteal arteries
- Internal carotid arteries
- Vessels around the circle of willis
 - -Vessels of upper extremity are usually spared
 - -Renal/Mesentric vessels are involved at the ostia

ATHEROSCLEROSIS-Clinical significance

Small arteries

- Atheromas may occlude lumen & compromise blood flow leading to ischemic injury.
- Plaque disruption & thrombosis causes obstruction.

Large arteries

- Destructive plaque with encroachment of media causes weakening of wall with the development of aneurysms.
- Systemic embolization may result from friable atheromas.

ATHEROSCLEROSIS- Clinical Effects

Heart – Coronary Artery Disease

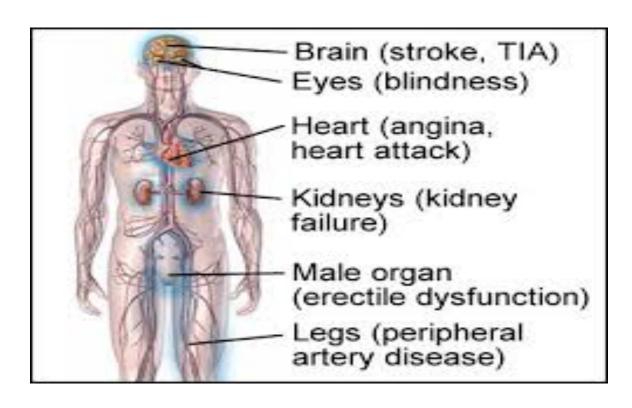
Brain – Stroke

Aorta- Aneurysm

Kidney- Failure

Intestine – Ischemia

Lower Extremities – Gangrene



ATHEROSCLEROSIS- Clinical Syndromes

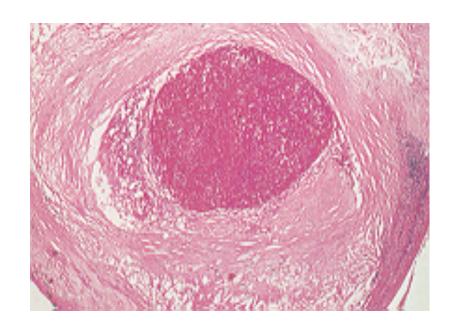
Heart- Angina or Myocardial Infarcts or Heart Attacks, CHID

Brain- Transient Cerebral Ischemia / Cerebral Infarcts/ Strokes

Peripheral Arteries – Peripheral arterial Disease, Mesenteric arterial occlusion

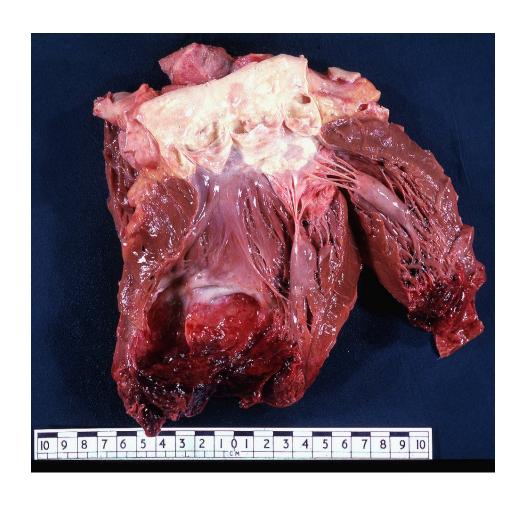
Atheroma - Coronary Artery





- •Ischaemic heart disease
 - sudden death
 - myocardial infarction
 - angina pectoris
 - arrhythmias
 - cardiac failure

Atheroma – myocardial infarction



Atheroma – myocardial infarction



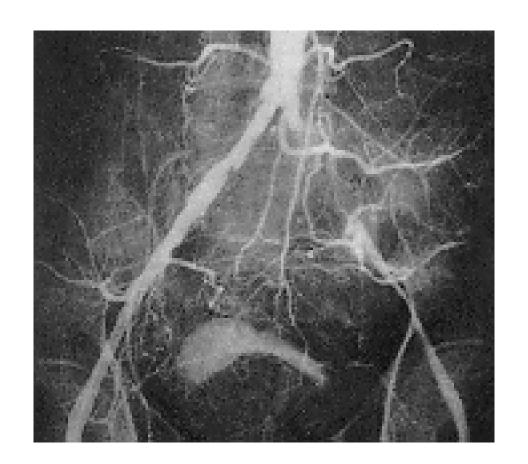
- Cerebral ischaemia
 - transient ischaemic attack
 - cerebral infarction (stroke)
 - multi-infarct dementia



- Mesenteric ischaemia
 - ischaemic colitis
 - malabsorption
 - intestinal infarction



- Peripheral vascular disease
 - intermittent claudication
 - Leriche syndrome
 - ischaemic rest pain
 - gangrene



Atheroma – Abdominal Aortic Aneurysm



PREVENTION

Primary Prevention

- Delaying Atheroma formation
- Regression of established lesions
 - Cessation of smoking
 - Control of Hypertension
 - Weight reduction/ Increased exercise
 - Diet Modification

Secondary Prevention

- Programs intended to prevent recurrence
 - Use of lipid lowering drugs
 - Use of antiplatelet drugs

THANK YOU