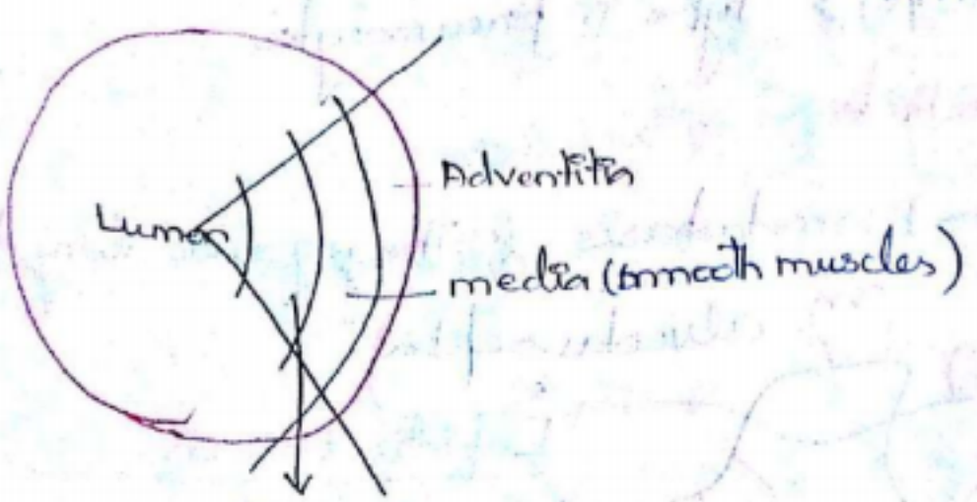
CVS



Intima
One/2 layers of endothelial cells

Neo-intima endothelial cells + New smooth muscle cells during injury, inflammation, grafting.

Atherosclerosis - Intimal based plaques.

Risk factors

Non-modifiable
→ old age
→ male
→ stress (Type-A)
Hyperhomocystinuria

Modifiable
- Obesity
- DM
- HTN

Infections-
CMV + Herpes + chlamydia.

Widely accepted Hypothesis - Endothelial injury

Intimal

Cytokines

Recruit Smooth muscles

Inflammation (macrophages

Oxidized LDL + macrophages / Smooth muscles → Foam cells

↓

Fibrous plaque

↓

Fatty streak (earliest feature visible)

m/c site - Abdominal aorta

Least/c site - Circle of willis

Aneurysm - Dilatation of Blood wall / Heart wall

a) True aneurysm: All 3 layers of Blood vessel wall

b) Pseudo Aneurysm - only one wall (Intimal) Tear

extravascular Hematoma

m/c Cause - Post myocardial cardiac wall ruptures

c) Mycotic aneurysm - m/c a/w Staphylococcus

m/c site - Femoral Artery

4) Luetic aneurysm - Syphilis (Tertiary stage)

Intimal wainkling - Tree Bark appearance

m/c site - Ascending aorta (Root)

AR → enlarged Heart chamber (Cor bovinum)

Ascending A
Hypertension

Atherosclerosis.

Aortic dissection - 40-60 years.

Dissection into T. media.

m/c Cause aortic dissection → HTN

m/c Histology - cystic medial degeneration:
[marfans, OI, EDS]

Vasculitis

Large	Medial	Small
1) Giant cell arteritis	1) PAN	Wegener's
2) Takayasu.	No ANCA	microscopic polyangiitis
	all stages of inflammation in all vessels	Churg-strauss

Large

Giant cell arteritis

Segmental Biopsy

granulomatous

fragmentation of

internal elastic lamina

Medium

Kawasaki

anti

endothelial

Ab

Small

Churg strauss

eosinophilic

granuloma

P-ANCA.

M/c Cause of vasculitis:

a) Pediatric – Henoch Schonlein purpura.

b) Adult – Giant cell arteritis.

c) m/c COD in ped – Kawasaki

II Takayasu arteritis - m/c site - Subclavian arteritis.

m/c Cause of mononeuritis multiplex

Diabetes mellitus (Ind/world)

→ amongst Vasculitis - PAN.

Hairy cell leukemia – PAN.

Anti endothelial antibodies - Kawasaki.

Coronary Artery disease

Significant Obstruction >75%

Risk factors -

1) hs-CRP.
2) Total cholesterol / HDL
3) ApoB / ApoA.
4) LDL
5) HDL.

m/c site

Left anterior descending artery.
↓
mI (LV)

Gross - upto 12 Hrs no finding / occasional microscopy

TTC Assay - Triphenyl Tetrazolium chloride assay.

TTC.

Normal → LDH + TTC. → Darkred Colour → normal.

m.I → all LDH Released into Serum. → (no LDH to combine) → Pale colour → M.I

Biochemical markers -

earliest — myoglobin.

m. sensitive/specific - Troponin I > T.

↑ 2-4hrs → 7-10days.

Re-infarction - CK-MB.

Flipping of LDH - Normal LDH 2 > 1

Flip pattern - LDH 1 > 2.

Gross	Microscopy	EM
12hrs Occasional dark mottling	4Hrs - waviness of fibres.	1st ½ hour - Mitochondrial Swelling
12-24Hrs - established dark mottling	12-24hrs - Coagulative necrosis. early neutrophilic infiltrate	> ½ hour - Large flocculent amorphous densities.
1-3days - yellow-tan-infarct	[1-3days max Neutrophilic infilt]	
3-7days - Hyperaemic Borders	7-10days - early granulation tissue	
2 months - Scarring complete.	10-14days - max. granulation tissue.	

Rheumatic Heart Disease.

GABH: 1, 3, 5, 6, 18

Pharyngitis → 3% → RHD

Valve m/c - mitral

4/c - pulmonary

Aschoff Bodies:

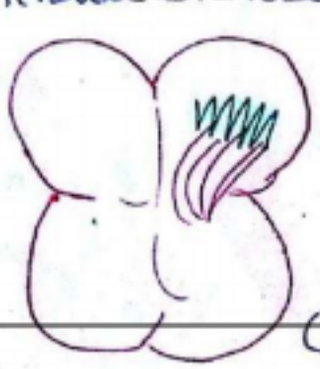
Swollen Collagen
Plasma cells
Histiocytes - Caterpillar like nuclear chromatin } Diagnostic of RHD
↓
Anitschow cells

→ No epithelioid cells
No langhans giant cells

Aschoff Bodies - myocardium (m/c) - Perivascular areas.
Pericardium
endocardium

Acute RHD:
m/c - mitral Regurgitation.
overall - mitral stenosis.

Chronic
mitral stenosis



(L) Atrium posterior wall
Subendocardial patches } MTac
Aschoff Bodies } Callum plaques

Commonly seen in Chronic RHD

Most common cause of Death - endocarditis

Chronic RHD - myocarditis.

Overall - myocarditis

RHD	NBTE (↑Coagulabe, malignancy, ↑estrogen)	LSE	I.E)
→ Small	Small NBTE > RHD more prone for embolisation.	medium vegetations.	Large
Sterile	Sterile.	flat Sterile	Non-sterile.
along line of closure of wall	along line of closure of wall	either side of wall. Lower Surface up. → pocket of walls. → Destructive.	Both surfaces upper > lower. max embolization most destructive

One liners -

→ Dilated Cardiomyopathy - a/w Largest Titin (30,000).

→ (R) wall.

→ Arrhythmogenic (R) ventricular Cardiomyopathy.

a/w plakoglobin protein mutation.

→ LV hypertrophy ↑ osteoglycin (OGN).

HOCM: m/c cause of Sudden death in athletes. HOCM m/c mutation — β MHC gene (myosin heavy chain).

Cysts

- Banana like cavity.

m/E: Disarray of myofibres.
Helter-Skelter disarray

Myocarditis - Viral infections.

m/c - Parvovirus - B19 > Herpes > Coxsackie.

Parasite - m/c - Trichinella Spiralis

m/c Cause of Hemorrhagic pericarditis → malignancy
m/c Cause - Lung Ca.

m/c Splen tumour → Benign Cavernous Hemangioma
Rest all → malignancies.

m/c Tumour of Heart - metastasis
m/c - 1° lung.
m/c site - pericardium.
Cardiac tumours

Pediatric

m/c 1° malignancy
Rhabdomyosarcoma.

1° Benign Rhabdomyoma.

Adults

m/c 1° malignancy - Angiosarcoma.

m/c Benign 1° - Cardiac myxoma.