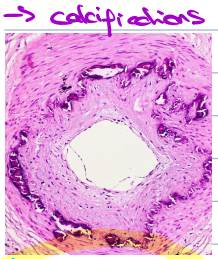
S02: Vascular Disease Pathology

Age-Related Vascular d's
Aneurysms
HTN d's
Diabetic vascular disease
Vasculitis

Age-Related vascular d's

occur in: [Aorta / AA. / Arterioles]
not clinically relevant before 40 y/o
Most common post 70 y/o
NB d's: fibrous thickening of intima
: fibrosis & scarring of media (muscular → elastic)
: ↑ mucopolysaccharide (Gravd Sub.)
: fragmentation of elastic laminae
d's = ↓ strength/Elasticity of vessel walls
= progressive dilation may occur in coronary AA.
= Aortic Valve Ring can stretch = AI (Regurg.)
Arteriosclerosis [Muscular aa.] (Hardening)
∴ ↑ freq of cardiac
Renal
cerebral
Colonic ischaemia (Elderly)
⇒ calcifications [NB not Atherosclerosis]
∴ no foam macrophages
→ calcifications + Intima thickening



Aneurysms:

Localised Abnormal dilation of a blood vessel / heart
↳ congenital / Acquired

True Aneurysm

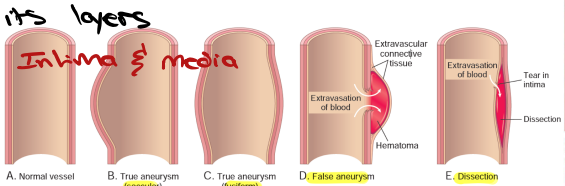
involves intact arterial / ventricular wall
(most common)
→ Atherosclerotic (complicated)
→ Syphilitic (almost ∅ see anymore)
→ congenital
→ Ventricular (following transmural MI)

False Aneurysm:

Defect in vascular wall = Extravascular Haematoma
in comm. with Intravascular space

Dissection:

Blood enters into vascular wall (via defect)
& tunnels through its layers
↳ usually between Intima & media
↳ usually in Aorta



Aneurysm Types

Table 13.1 Clinical effects of aneurysms

Type of aneurysm	Site	Clinical effects
Atherosclerotic ↳ can develop in Elderly ↳ impair blood flow to lower limbs ↳ usually occurs at Bifurcation of Abdominal AA into Iliac AA.	Lower abdominal aorta and iliac arteries	Pulsatile abdominal mass Lower limb ischaemia Rupture, with massive retroperitoneal haemorrhage
Aortic dissection ↳ blood into vessel defect → between intima & media ⇒ can track back into pericardium = Massive Haemopericardium [Tamponade] ⇒ can track into Lumen = "double-barrel" Aorta ↳ usually same degenerative process involved ↳ mucoid degen. of media with elastic fibre fragmentation ⇒ Marfan syndrome [FBN 1 – fibrillin gene mutation] ⇒ Genetics can play a role	Aorta and major branches	Loss of peripheral pulses (e.g. radials) Haemopericardium External rupture (retroperitoneal haemorrhage) Re-entry from dissected media to lumen causing 'double-barrelled' aorta
Berry ↳ Saccular Aneurysms found @ Branching points	Circle of Willis	Subarachnoid haemorrhage (most MS complication)
Microaneurysms (Charcot–Bouchard)	Intracerebral capillaries	Intracerebral haemorrhage, associated with hypertension
Syphilitic ⇒ proximal Aneurysm ↳ previously common	Ascending and arch of aorta	Aortic incompetence
Mycotic (infective) ↳ d/t weakening of aa. wall → bacterial → fungal reach aorta via blood stream (enter via vasa vasorum)	Root of aorta (direct extension from aortic valve endocarditis) Any vessel	Thrombosis or rupture, causing cerebral infarction or haemorrhage

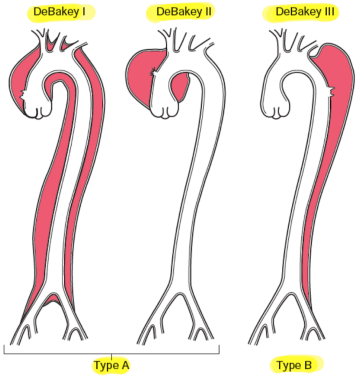


Figure 11-22 Classification of dissections. Type A (proximal) involves the ascending aorta, either as part of a more extensive dissection (DeBakey I) or in isolation (DeBakey II). Type B (distal or DeBakey III) dissections arise after the take-off of the great vessels. Type A dissections typically have the most serious complications and greatest associated mortality.

↳ Type A = proximal involvement ⇒ Asc. Aorta
↳ Type B = Distal involvement ⇒ Asc. Aorta (no involve)

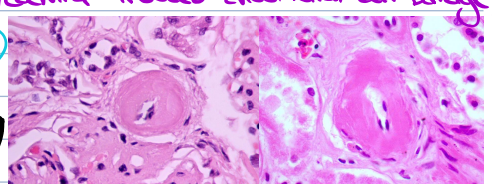
3° Syphilis Rare in developed world
underdeveloped = lack Antibiotics
Aneurysm d/t Ischaemic damage to media
= fibrosis & loss of elasticity
↳ obliterative endarteritis (incl. vasa vasorum)
⇒ Tree Bark appearance
↳ most common in cerebral aa.
↳ Bacterial Endocarditis = most common underlying infection
⇒ vegetations on valves embolise = Distal Mycotic Aneurysms

Hypertensive d's → Major risk for Atherosclerosis

↳ causes thickening of muscular aa. → Smooth muscle Hyperplasia
→ collagen deposition

Mainly affects smaller aa. & arterioles
↳ Hyaline Arteriolosclerosis:
→ Arterioles = pink Hyaline thickening = narrowing of lumen
→ plasma proteins leak across injured endothelial cells & ↑ smooth Mm. cell matrix
↳ seen in Elderly & DM
↳ [Hyperglycaemia - induced endothelial cell damage]

↳ Hyperplastic Arteriolosclerosis



↳ Severe HTN
↳ concentric "laminated" thickening of walls with luminal narrowing
⇒ Lamellations = smooth Mm. cells with reduplicated BM (thickened)
↳ in Malignant HTN = accompanied fibrinoid deposition + vessel wall necrosis (necrotising arteriolitis)

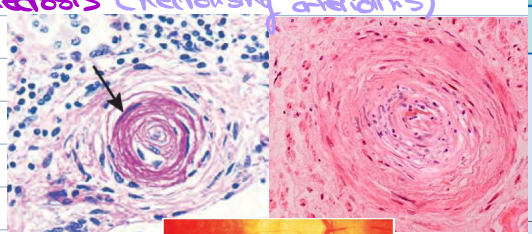


Table 13.2 Pathogenesis of systemic hypertension

Aetiological classification	Causes
Essential (primary) hypertension	Unknown, but probably multifactorial involving: • Genetic susceptibility • Excessive sympathetic nervous system activity • Abnormalities of Na/K membrane transport • High salt intake • Abnormalities in renin–angiotensin–aldosterone system
Secondary hypertension	Renal disease • Chronic kidney disease, especially in diabetes • Renal artery stenosis • Glomerulonephritis Endocrine causes • Adrenal tumours (cortical or medullary) • Cushing's syndrome Coarctation of aorta Drugs, e.g. corticosteroids, oral contraceptives

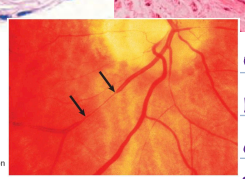


Fig. 13.10 Hypertensive fundus. Ocular fundus from a patient with hypertension. The outline of the blood vessels is caused by the reflection of light from the column of blood (the light reflex). Because the wall of the arteriole is thickened in hypertension, the lumen of the vessel is narrowed and the light reflex is reduced (between the arrows).

NB
Always do
a
Histology.

Diabetic Vascular Disease:

Patients with DM may develop 3 forms of vascular disease

Atherosclerosis → male & female

Develops premature, severe Atherosclerosis

Hypertensive Vasculature Disease:

frequent complication, Esp if diabetic renal disease is present

Capillary Microangiopathy: → NB

Most NB d in DM → found in fundoscopy

Alterations found throughout systemic circulation

Small arterioles & capillaries affected:

↳ Diabetic Retinopathy

↳ Diabetic Glomerulosclerosis

↳ Peripheral Neuropathy

⇒ Biochemical d are complex

[Abnormal Glycosylation of proteins within vessel wall]

Vasculitis:

Inflammatory Diseases of blood vessels

↳ Mostly immune-Mediated

- immune complex formation [SLE / Drug Hypersensitivity]
- Anti-neutrophil cytoplasmic Ab (ANCA'S)
- Anti-endothelial cell Ab
- Autoreactive T-cells (Giant cell / Temporal Arteritis)

↳ occasionally infective Microorganisms

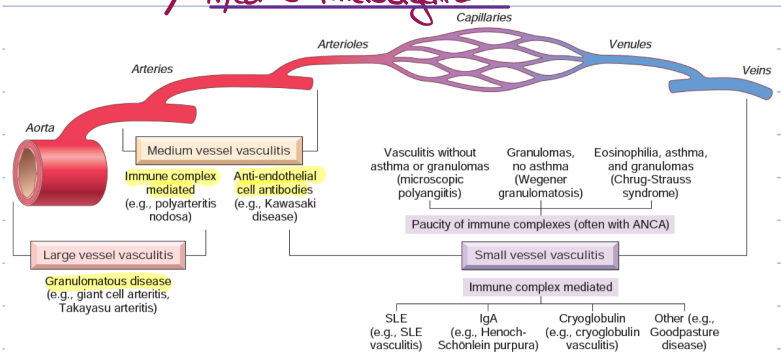


Figure 11-23 Vascular sites typically involved with the more common forms of vasculitis, as well as their presumptive etiologies. Note that there is a substantial overlap in distributions. ANCA, Antineutrophil cytoplasmic antibody; SLE, systemic lupus erythematosus.

Giant cell Arteritis: [GCA]

↳ Affects aa. of Head & neck

can cause blindness → ophthalmic aa.

→ Post. ciliary aa.

Elderly = most common form of Vasculitis

In fatal cases the superficial temporal aa.

is hard, tender & pulseless

↳ pt = severe Headache

= ESR ↑ [indicative of inflammation]

Microscopically:

Intimal thickening

Dense Granulomatous inflammation

Chronic Infiam of wall

(Giant cells phagocytosing Elastic fibres)

thought be T-cell Mediated immune Response

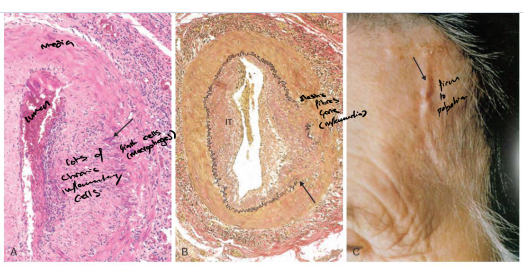


Figure 11-24 Giant cell (temporal) arteritis. **A,** Hematoxylin and eosin stain of section of temporal artery showing giant cells at the degenerated internal elastic lamina in active arteritis *(arrow)*. **B,** Elastic tissue stain demonstrating focal destruction of internal elastic lamina *(arrow)* and intimal thickening (IT) characteristic of long-standing or healed arteritis. **C,** The temporal artery of a patient with classic giant cell arteritis shows a thickened, nodular, and tender segment of a vessel on the surface of head *(arrow)*. (**C,** From Salvarani C, et al. Polymyalgia rheumatica and giant-cell arteritis. N Engl J Med 347:261, 2002.)

Takayasu Arteritis

(pulseless disease)

Rare Inflam disorder of Aorta & its proximal branches

Mostly young / middle-aged females

↳ present with Ischaemic sym of arms

Renal aa. involvement = Hypertension

Similar Histology compared to GCA

Buerger's Disease

(Thrombo Angiitis obliterans)

↳ Rare (+) associated with smoking

↳ Male + jewish

⇒ peripheral gangrene → fingers → Toes

d's are progressive & serial

Amputation often needed

⇒ mainly small aa. in arms & lower legs

⇒ Show intimal fibrosis

⇒ Thrombus formation (+ Recanalisation)

Acute & chronic inflammation Extends into contiguous Vv. & Aa.

↳ surrounds = very painful