CONGENITAL ANOMALIES OF BLOOD VESSELS 🧬

General Overview

Congenital vascular anomalies are usually asymptomatic, but they carry major clinical and surgical significance.

Why they matter clinically ⚠️

- Abnormal vessel location
  → Increased risk of accidental injury during surgery
- Variant anatomy
  → Challenges in interventional cardiology & cardiac surgery

📌 Important Fact:
Coronary artery variants occur in ~1–5% of individuals

Major Congenital Vascular Anomalies

1. Berry (Saccular) Aneurysms 🧠

Definition

- Thin-walled arterial outpouchings
- Occur in cerebral arteries

Common Location

- Branch points around the Circle of Willis

Pathogenesis (Flowchart)

Congenital attenuation of arterial media → Weak vessel wall → Formation of saccular outpouching → Increased risk of rupture → Intracerebral / subarachnoid hemorrhage ☠️

Clinical Significance

- Often asymptomatic until rupture
- Rupture can cause:
    - Sudden severe headache
    - Fatal intracerebral hemorrhage

📌 High-Yield Association:

Berry aneurysms → Associated with Autosomal Dominant Polycystic Kidney Disease (ADPKD)

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2. Arteriovenous (AV) Fistulas 🔁

Definition

- Abnormal direct connection between artery and vein
- No intervening capillary bed

Etiology

- Congenital (most common)
- Acquired causes:
  - Rupture of arterial aneurysm into vein
  - Penetrating trauma
  - Inflammation and necrosis of adjacent vessels

📌 Iatrogenic Use:

AV fistulas are surgically created for:

- Hemodialysis vascular access

Hemodynamic Consequences (Flowchart)

Arterial blood → Direct shunting into venous system → Bypass of capillary bed → ↓ Peripheral resistance → ↑ Venous return → ↑ Cardiac output → High-output cardiac failure (if large/multiple AV fistulas) ❤️‍🔥

📌 Exam Phrase:
AV fistulas cause high-output cardiac failure, not low-output failure.

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3. Fibromuscular Dysplasia (FMD) 🩺

Definition

- Non-atherosclerotic, non-inflammatory disease
- Characterized by focal irregular thickening of arterial walls

Affected Vessels

- Medium- and large-sized muscular arteries
- Renal arteries involved in 75–90% of cases

Epidemiology

- Predominantly affects women
- Median age at diagnosis: 52 years
- Etiology: Unknown

Pathogenesis

- Combination of:
    - Medial hyperplasia
    - Intimal hyperplasia
    - Fibrosis

Structural Consequences (Flowchart)

Medial & intimal hyperplasia → Focal wall thickening → Luminal narrowing (stenosis) → Reduced blood flow → End-organ ischemia

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Renal Involvement & Hypertension 🔥

Reduced renal perfusion → Activation of juxtaglomerular apparatus → ↑ Renin release → Activation of RAAS → Vasoconstriction + Na^+ retention → Renovascular hypertension 🩸

📌 Very High-Yield Mechanism:
FMD → renal artery stenosis → secondary hypertension

Additional Complications

Between thickened segments:

- Medial attenuation occurs
- Leads to:
    - Vascular outpouchings
    - Increased risk of rupture

📌 Classic Teaching Point:
Alternating stenosis and dilation predispose to vessel rupture.

Quick Comparison Table 📊

Condition	Vessel Type	Key Feature	Major Complication
Berry aneurysm	Cerebral arteries	Thin-walled saccular outpouching	Intracranial hemorrhage
AV fistula	Artery → vein	No capillary bed	High-output heart failure
FMD	Muscular arteries	Focal wall thickening	Renovascular hypertension

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One-Line Exam Pearls 💡

- Berry aneurysms rupture at Circle of Willis branch points
- AV fistulas → ↑ cardiac output

- Fibromuscular dysplasia → renal artery stenosis in women
- Congenital vascular anomalies may be silent but surgically dangerous

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-> The End <-