Aneurysms

⍰ Aneurysms are localized, irreversible dilatation of blood vessel lumen that exceeds the diameter of the adjacent normal segment.

Comparison: True Aneurysm vs. False Aneurysm

False Aneurysm (Pseudoaneurysm)	True Aneurysm	Feature
Does NOT involve all layers; there is a **wall defect** → blood is contained only by **surrounding tissues** or outer layer	Involves **all three layers** of the arterial wall (intima, media, adventitia), though they are thinned/attenuated	**Layers involved**
The "wall" is formed by a **hematoma** outside the vessel, not by the vessel wall	The aneurysm wall is formed by the **actual vessel wall**	**Wall structure**
A tear in the vessel wall allows blood to escape and form an **extravascular hematoma** that still communicates with the vessel lumen	- Atherosclerosis- Congenital defects- Ventricular aneurysm after **transmural MI**	**Cause / Pathogenesis**
Higher (no real wall—only contained by tissues)	**Lower** (wall still intact but weak)	**Risk of rupture**
- Femoral artery after catheterization- Anastomotic sites- Trauma sites	- Abdominal aorta (atherosclerosis)- Thoracic aorta (Marfan, Ehlers-Danlos)- Left ventricle (post-MI aneurysm)	**Common sites**
Loculated, sac-like hematoma outside the vessel	Uniform, smooth dilation of the vessel wall	**Appearance**
- Pseudoaneurysm after arterial puncture or trauma	- Atherosclerotic aortic aneurysm- Congenital aneurysm- Left ventricular aneurysm after MI	**Example**
Wall is ruptured; hematoma is contained externally	**Wall is intact but weak**	**Key Concept**

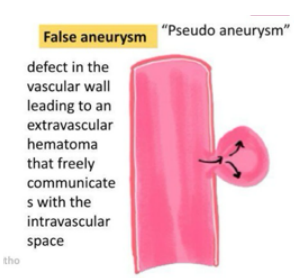
- However, the blood does not freely leak; instead, it becomes contained by:
  - surrounding tissues,
  - or the outer layer only,
  - forming an extravascular hematoma (a collection of blood outside the vessel).

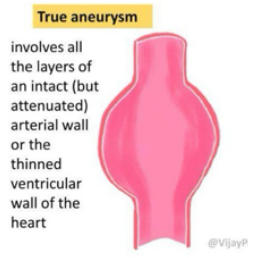
meaning:

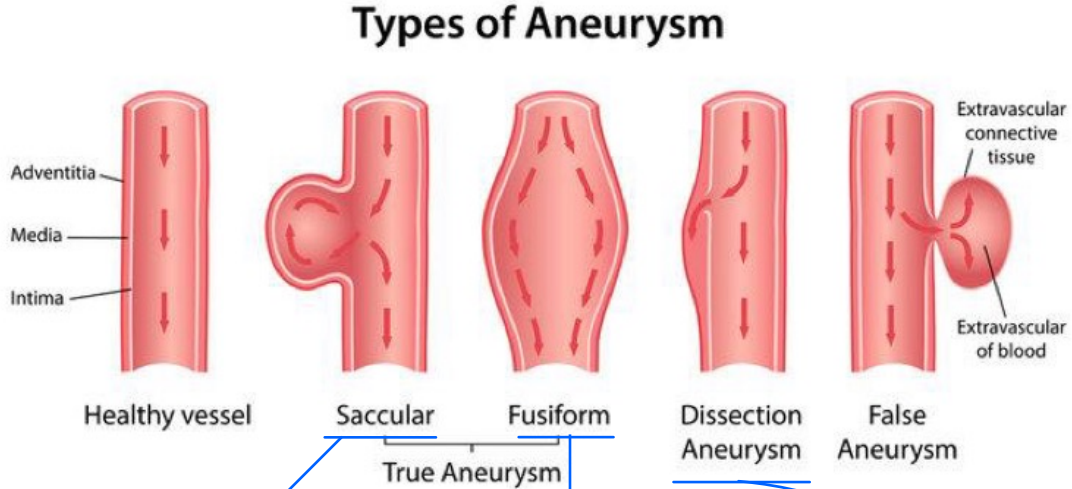
- it stays connected to the inside of the vessel through the tear,
  - so blood flows into it,
  - making it look like an aneurysm.

Ventricular aneurysms after transmural MI occur because the full-thickness infarction heals as a thin, fibrotic scar that bulges outward, forming a true aneurysm.

ventricular aneurysms





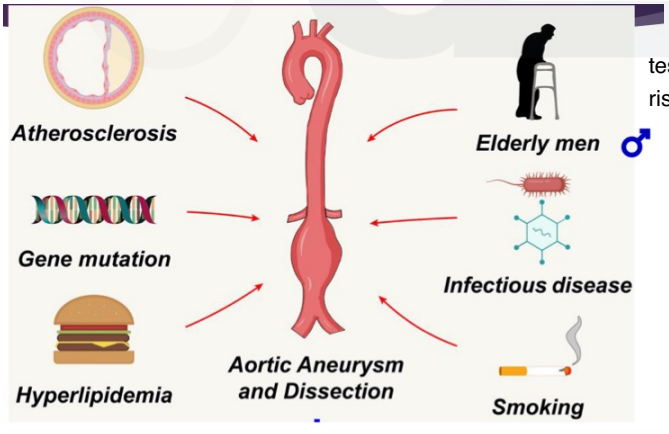


Saccular aneurysms:
• are discrete outpouchings ranging from 5 to 20 cm in diameter, often with a contained thrombus.

Fusiform aneurysms :
• are circumferential dilations up to 20 cm in diameter; these most commonly involve the aortic arch, the abdominal aorta, or the iliac arteries.

A dissection aneurysm occurs when blood enters the wall of the artery through a tear in the intima (the inner layer), and then:
- splits the media (middle layer)
- creating a false channel (called a false lumen) between the layers of the vessel wall.

Risk factors



testosterone is considered one of the reasons males have a higher cardiovascular risk compared to females during reproductive age.

Estrogen (in females) – Protective effect

Pathogenesis

✓ Inadequate or abnormal connective tissue synthesis:
• mutations in TGF-β receptors result in defective elastin and collagen synthesis.
• Loss of SMCs or change in the SMC synthetic phenotype: e.g. in :
Atherosclerosis, systemic hypertension , both of them leading to ischemia resulting in SMC loss as well as aortic “degenerative changes” .
✓ Excessive connective tissue degradation:
• Increased matrix metalloprotease expression by macrophages in atherosclerotic plaque.

Atherosclerotic

Atherosclerotic refers to atherosclerosis, a chronic disease in which lipids, cholesterol, inflammatory cells, and fibrous tissue accumulate inside the intima of arteries.

High yield ⚠️
You should know

Key points:

- Forms atherosclerotic plaques.
- Narrows the artery → reduced blood flow.
- Weakens the wall → can lead to aneurysm, thrombosis, or dissection.
- Common in large and medium arteries (aorta, coronary arteries).

Ischemia

Ischemia means insufficient blood flow to a tissue → leading to lack of oxygen and nutrients.

Causes of ischemia:

- Most commonly caused by atherosclerotic narrowing.
- Can also occur from thrombosis, embolism, vasospasm, or compression.

Consequences of ischemia:

- Pain (like angina)
- Tissue dysfunction
- If severe or prolonged → cell death (infarction)

Connection between them

Atherosclerosis is one of the most common causes of ischemia.

Example:

- Atherosclerotic plaque in a coronary artery → ↓ blood flow → myocardial ischemia → possibly myocardial infarction.

Excessive Connective Tissue Degradation معلم

This phrase refers to too much breakdown of structural proteins (like collagen and elastin) in the vessel wall.

Why does it happen?

Inside an atherosclerotic plaque, macrophages become activated and start releasing matrix metalloproteases (MMPs).

Matrix Metalloproteases (MMPs)

MMPs are enzymes that digest components of the extracellular matrix, including:

- Collagen
- Elastin
- Proteoglycans

When macrophages express too many MMPs, they break down these structural proteins faster than they can be replaced.

What is the result?

1. Weakening of the vessel wall

Because collagen/elastin provide strength, their degradation causes the arterial wall to become:

- thinner
- weaker
- more prone to dilation

→ leading to aneurysm formation, especially in the abdominal aorta.

2. Plaque instability

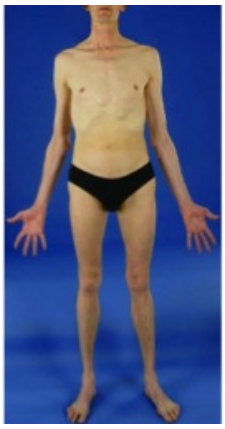
Excess MMP activity degrades the fibrous cap of the plaque → making it thin and prone to rupture → leading to thrombosis and acute MI.

In summary:

Macrophages in atherosclerotic plaques secrete high levels of MMPs, which excessively break down connective tissue (collagen and elastin), weakening the vessel wall and contributing to aneurysm formation or plaque rupture.

Marfan Syndrome

Marfan syndrome results from a defect in the synthesis of fibrillin, which is a major *scaffolding protein* required for the formation and maintenance of elastic fibers.

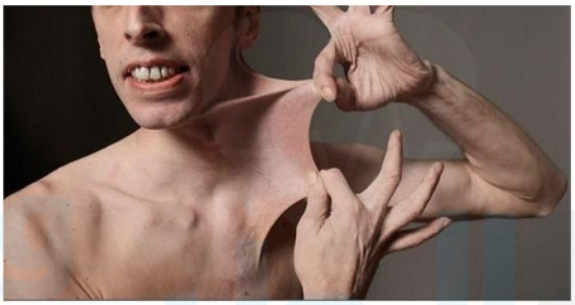


What happens because of fibrillin deficiency?

- Elastic tissue becomes weaker and progressively lost.
- The walls of large arteries—especially the aorta—lose their strength and elasticity.
- This leads to progressive dilation, particularly aortic root dilation and aortic aneurysm.
- These patients are at high risk for aortic dissection.

Ehlers-Danlos Syndrome (Vascular Type)

Ehlers-Danlos (vascular type) results from a defect in type III collagen synthesis.



Why is this important?

Type III collagen is a major structural protein in:

- blood vessel walls
- intestines
- uterus

Consequences of defective type III collagen:

- Vessel walls become very weak.
- This predisposes to spontaneous aneurysm formation, arterial rupture, and fragility of hollow organs.

Summary

Result	Effect on Vessel Wall	Defect	Disorder
Aortic dilation & aneurysm	Loss of elastic tissue	Defective **fibrillin**	**Marfan syndrome**
Aneurysm & rupture	Weak vessel structure	Defective **type III collagen**	**Ehlers-Danlos (vascular)**

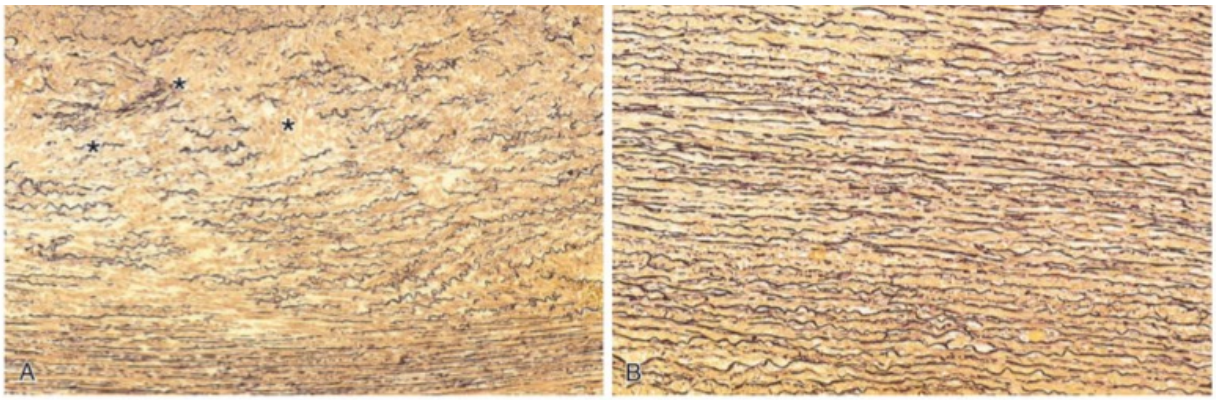


Fig. 10.18 Cystic medial degeneration. (A) Cross-section of aortic media from a patient with Marfan syndrome, showing marked elastin fragmentation and areas devoid of elastin that resemble cystic spaces *(asterisks)*. (B) Normal media for comparison, showing the regular layered pattern of elastic tissue. In both (A) and (B), elastin is stained black.

Cystic medial degeneration is a histologic term used to describe the structural changes that occur in the tunica media of large arteries (especially the aorta) in conditions like Marfan syndrome, Ehlers-Danlos, hypertension, and aging.

What does it look like histologically?

- Loss of smooth muscle cells
- Fragmentation of elastic fibers
- Accumulation of basophilic, mucoid (myxoid) ground substance in the media

These areas look like empty spaces or clefts under the microscope because the normal structure of the media is disrupted.

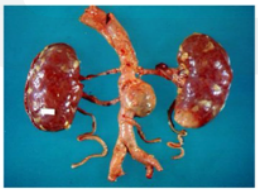
Why is it called “cystic”?

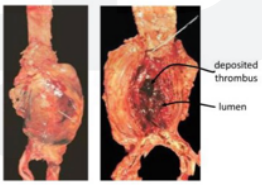
Because the degenerative spaces resemble cysts under the microscope — small, rounded, clear areas.

However:

- No real cysts are formed
- There is no epithelial lining
- They are simply areas of degeneration filled with mucoid material

So the name “cystic” is descriptive but not literal.





Abdominal Aortic Aneurysm (AAA)

Definition & Etiology

- Usually occurs as a consequence of **atherosclerosis**.
- Involves the **abdominal aorta,** typically located **between the renal arteries and the aortic bifurcation**, and may involve the **common iliac arteries.**
- Occurs more frequently in **old smoker males (>50 years).**

Pathogenesis

- In most cases, AAA results from **ECM degradation** mediated by **proteolytic enzymes** released from inflammatory infiltrates within atherosclerotic lesions.

Morphology

- The aneurysmal sac usually contains:
- **Bland, laminated, poorly organized mural thrombus,** which may fill much of the dilated segment.
- **Extensive atherosclerosis** is usually present.

Clinical Consequences of AAA

- **Ischemia** due to obstruction of a vessel branching off the aorta (e.g., renal or iliac arteries).
- **Embolism** of atheromatous material.
- **Compression** of adjacent structures.
- **Misdiagnosis** as an abdominal mass.
- **Rupture** into the peritoneal cavity → massive, fatal hemorrhage (especially if aneurysm is **>5 cm).**

Mycotic Thoracic Aortic Aneurysm (MTAA)

Definition

- A mycotic thoracic aortic aneurysm is an aneurysm caused by **infection** of the aortic wall through microbial inoculation of diseased endothelium.
- Most commonly caused by **bacteria or fungi.**

Why Intima Normally Resists Infection

- The vascular intima is usually highly resistant.
- Infection occurs when intimal integrity is compromised due to:
- Embolization of a septic embolus, often from infective endocarditis.
- **Extension of an adjacent suppurative process.**
- **Direct infection** of the arterial wall by circulating organisms.

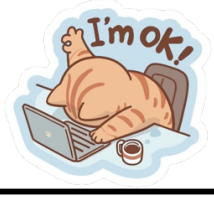
Thoracic Aortic Aneurysm

Associated Conditions

- Hypertension
- Bicuspid aortic valves
- Marfan syndrome

Clinical Manifestations

- **Compressive symptoms:**
- Respiratory difficulties (airway compression)
- Feeding difficulties (esophageal compression)
- **Persistent cough** due to irritation of the recurrent laryngeal nerve.
- **Pain** from erosion of ribs or vertebral bodies.
- Risk of **aortic dissection or rupture.**



Thrombophlebitis and Phlebothrombosis

Definitions

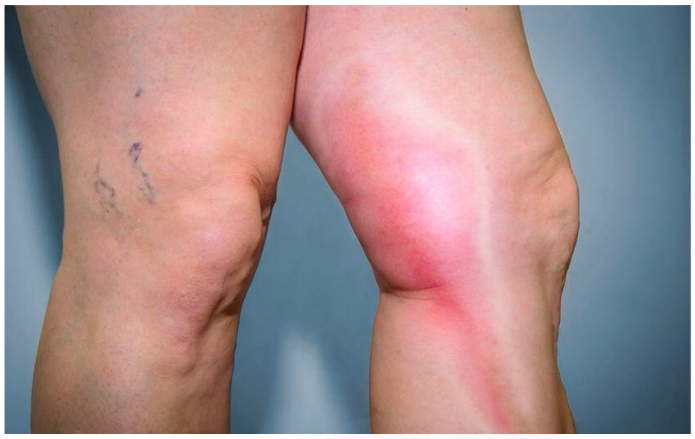
- **Thrombophlebitis**: Inflammation of the vein wall that **precedes** thrombus formation.
- **Phlebothrombosis**: A thrombus in a vein **without** associated inflammation.

Most Common Site

- **Deep leg veins (DVT)** → account for **>90%** of cases.

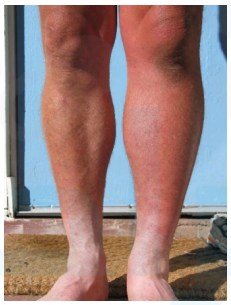
Risk Factors

- Prolonged immobilization
- Congestive heart failure
- Pregnancy
- Oral contraceptive use
- Malignancy
- Obesity
- Male sex
- Age > 50 years



Signs and Symptoms

- Hotness
- Tenderness
- Redness
- Swelling



Complication

- In many cases, the **first manifestation** is a **pulmonary embolus** due to fragmentation or detachment of the venous thrombus.

Lab Test

- D-DIMER

Treatment

- Anticoagulation
- Supportive measures

Pulmonary Embolus as First Manifestation of Thrombophlebitis

In many patients with deep vein thrombosis (DVT), there are no obvious symptoms in the leg.
Sometimes, the first clinical sign of DVT is a pulmonary embolus (PE).

This happens because:

- A piece of the venous thrombus breaks off (fragmentation or detachment),
- Travels through the venous system → right heart,
- Lodges in the pulmonary arteries, causing PE.

This is the most serious complication of DVT.

Laboratory Test: D-Dimer

D-Dimer = elevated

D-dimer is a breakdown product of cross-linked fibrin.
It is high when there is ongoing clot formation and lysis, as in:

- **DVT**
- **Pulmonary embolism**

Interpretation:

- High D-dimer: Suggests possible DVT/PE → requires imaging.
- Normal/negative D-dimer: Helps rule out DVT/PE in low-risk patients.

Treatment of DVT / Thrombophlebitis

1. **Anticoagulation (first-line)**
   - Heparin (IV unfractionated heparin or LMWH like enoxaparin)
   - Then switch to oral anticoagulants:
   - Warfarin, or
   - DOACs (direct oral anticoagulants): apixaban, rivaroxaban, dabigatran

2. **Thrombolysis (selected severe cases)**

Used only for:

- Massive PE
- Limb-threatening DVT (phlegmasia cerulea dolens)

Drugs: tPA (alteplase)

3. IVC Filter

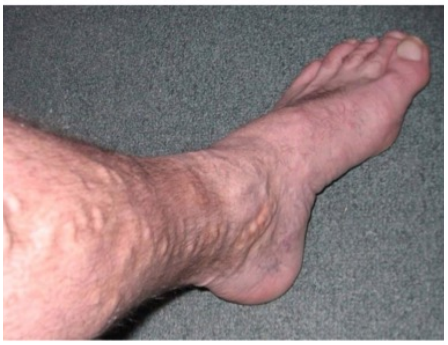
Used only when anticoagulation is contraindicated (e.g., active bleeding).

4. Compression & Support
   - Compression stockings
   - Elevation of limb
   - Mobilization

Varicose Veins of the Extremities

Definition

- Abnormally dilated, tortuous veins caused by **chronically increased intraluminal pressure** and **weakened vessel wall support.**
- Usually involve the **superficial veins** of the upper and lower legs.

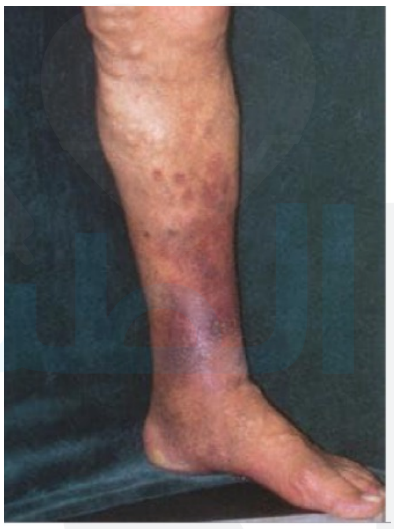


Risk Factors

- Obesity
- Pregnancy
- Familial tendency

Clinical Features

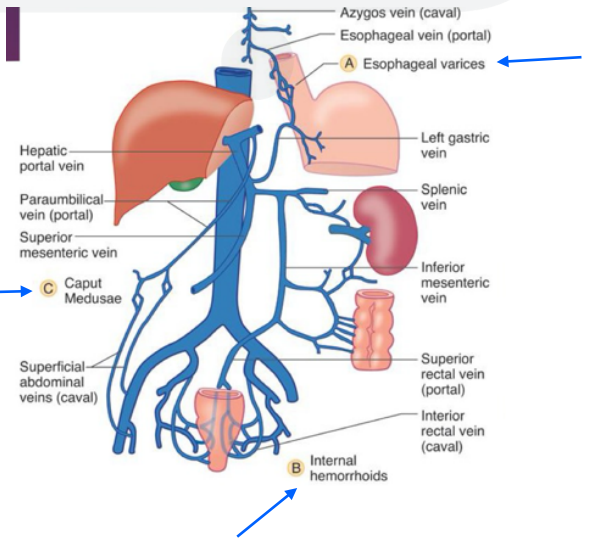
- Due to **venous valve incompetence** → lower-extremity stasis:
- Congestion
- Edema with secondary ischemic skin changes:
- Stasis dermatitis
- Ulcerations
- Poor wound healing
- Superimposed infections
- Pain
- Thrombosis

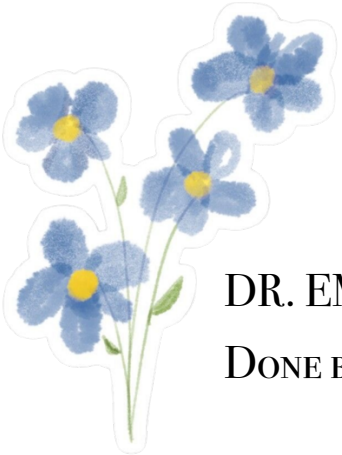


Varicosities in Other Sites

Due to Liver Cirrhosis–related Portal Hypertension

- **Gastroesophageal junction** (esophageal varices)
- **Rectum** (hemorrhoids)
- **Periumbilical veins,** forming **caput medusae**





DR. EMAN KREISHAN

DONE BY : RAGHAD MRAYAT

إلي طلاب وطالبات الطب :

« كل كرسي من كراسي كلية الطب هو بمثابة أحد سدود الواجب الكفائي المفروض على الأمة ... أنت على ثغر عظيم فأحسن القيام على ثغرك » 💙