

# ANEURYSMS AND DISSECTIONS

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## I. Definitions

Term	Definition	Layers Involved	Example
True Aneurysm	Localized dilation of a vessel/heart wall	All 3 layers (intima, media, adventitia)	Atherosclerotic aortic aneurysm, congenital aneurysms, post-MI ventricular aneurysm
False (Pseudo) Aneurysm	Wall defect → extravascular hematoma communicating with lumen ("pulsating hematoma")	Not all vessel layers	Ventricular rupture contained by pericardium, vascular graft leak
Dissection	Blood enters wall through defect → separates layers	Intima + media	Aortic dissection

Clinical importance:

- Cause stasis → thrombosis
  - Prone to rupture → catastrophic hemorrhage ⚠️
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## 2. Classification by Shape

Shape	Description	Common Sites
Saccular	Discrete outpouching, often contains thrombus	Cerebral arteries (berry aneurysms)
Fusiform	Circumferential dilation	Aortic arch, abdominal aorta, iliac arteries

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## 3. Pathogenesis of Aneurysms

Underlying problem: Weakening of vessel wall → imbalance between ECM synthesis & degradation

Mechanisms:

## 1. Defective connective tissue synthesis

- Genetic disorders → abnormal ECM
- TGF- $\beta$  signaling defects → impaired SMC proliferation & matrix synthesis
- Marfan syndrome: Fibrillin defect → ↑ TGF- $\beta$  → elastic tissue loss → aortic dilation

## 2. Excessive ECM degradation

- Inflammation → macrophages release matrix metalloproteases (MMPs) → elastin/collagen breakdown
- Seen in atherosclerotic aneurysms

## 3. Smooth muscle cell (SMC) loss / phenotypic change

- Intimal thickening → medial ischemia
- Hypertension → vasa vasorum narrowing → outer media ischemia
- Histology → Cystic medial degeneration (fibrosis, loss of elastic tissue, amorphous proteoglycans)
- Occurs in Marfan syndrome, scurvy, ECM defects

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## 4. Predisposing Factors

Factor	Type	Typical Association
Atherosclerosis	Acquired	Dominant in abdominal aortic aneurysms
Hypertension	Acquired	Ascending aortic aneurysms
Smoking	Lifestyle	↑ risk of abdominal aortic aneurysms
Trauma	Acquired	Vessel wall injury → aneurysm formation
Vasculitis	Acquired	Inflammation → wall weakening
Congenital anomalies	Genetic	E.g., connective tissue disorders
Infections (Mycotic aneurysm)	Acquired	Septic embolus, adjacent suppurative process, direct infection

Tertiary syphilis	Rare	Obliterative endarteritis of vasa vasorum → ascending aorta & aortic valve annulus involvement
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### Key Points:

- Abdominal aorta: atherosclerosis + smoking dominant
- Ascending aorta: hypertension dominant
- Mycotic aneurysms = infectious cause

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### 5. Flowchart: Pathogenesis of Aortic Aneurysm

Defective ECM / SMC loss / inflammation → Medial weakening → Vessel dilation →

- True aneurysm (all 3 layers)
- False aneurysm (pseudo) (wall defect → pulsating hematoma)

Contributing factors:

- Genetic defects → ECM abnormalities (Marfan, TGF- $\beta$  mutations)
- Chronic inflammation → MMP-mediated degradation
- Hemodynamic stress (hypertension, trauma)
- Infectious causes → mycotic aneurysm

Consequences:

- Stasis → thrombosis
  - Rupture → life-threatening hemorrhage
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### High-Yield Exam Points

- True aneurysms involve all 3 layers; false aneurysms do not
- Saccular vs. fusiform: shape guides clinical suspicion & site
- Cystic medial degeneration = hallmark of Marfan / ECM disorders
- Abdominal aortic aneurysms: atherosclerosis + smoking

- Ascending aortic aneurysms: hypertension; tertiary syphilis rare
  - Mycotic aneurysms arise from infection → embolic, contiguous, or direct
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## Aortic Aneurysms: Abdominal and Thoracic

### I. Abdominal Aortic Aneurysm (AAA)

#### Epidemiology & Risk Factors

- Most common site: abdominal aorta & common iliac arteries
- Less commonly: aortic arch, descending thoracic aorta
- Predominantly men & smokers
- Rare before 50 years of age
- Major cause: atherosclerosis
- Other contributing factors: ECM imbalance, inflammation, genetics

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## Morphology

- Location: between renal arteries and aortic bifurcation
- Shape: saccular or fusiform
- Size: up to 15 cm diameter, 25 cm length
- Thinning & focal destruction of media
- Mural thrombus: bland, laminated, poorly organized
- Often associated with iliac artery aneurysms

## Special Subtypes:

Subtype	Features	Notes
Inflammatory AAA	Dense periaortic fibrosis, chronic inflammatory cells (lymphocytes, plasma cells, macrophages, giant cells)	5-10% of AAAs, younger patients

IgG4-related AAA	Fibrosis + IgG4 plasma cells	Can affect pancreas, thyroid, biliary system; responds to steroids & anti-B-cell therapy
Mycotic AAA	Infection of aneurysm wall or thrombus	Rapid dilation & rupture; often secondary to bacteremia or infective endocarditis

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## Clinical Features

- Obstruction of branch vessels: renal, iliac, vertebral, mesenteric → ischemia
- Embolism: atheromatous material or thrombus
- Compression of adjacent structures: ureter, vertebrae
- Palpable abdominal mass: often pulsatile
- Rupture: peritoneal or retroperitoneal → massive hemorrhage 

Risk of Rupture by Size:

- $\leq 4$  cm  $\rightarrow$  almost never rupture
- $\geq 5.5$  cm  $\rightarrow$  high risk  $\rightarrow$  surgical repair indicated

Key Point: AAA patients usually have systemic atherosclerosis  $\rightarrow$   $\uparrow$  risk of IHD & stroke

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### Flowchart: AAA Pathogenesis & Consequences

Atherosclerosis + ECM imbalance  $\rightarrow$  Medial weakening  $\rightarrow$   
Abdominal aortic dilation  $\rightarrow$

- Chronic expansion  $\rightarrow$  Compression of nearby structures, obstruction of branch vessels  $\rightarrow$  ischemia
  - Mural thrombus  $\rightarrow$  embolism
  - Rupture  $\rightarrow$  massive hemorrhage  $\rightarrow$  emergency surgery
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## 2. Thoracic Aortic Aneurysm (TAA)

### Etiology / Risk Factors

- Hypertension → major cause
  - Bicuspid aortic valve → structural predisposition
  - Marfan syndrome → cystic medial degeneration
  - Other causes: tertiary syphilis, TGF- $\beta$  pathway mutations (e.g., Loeys-Dietz syndrome)
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## Clinical Features

- Respiratory / feeding issues: airway or esophagus compression
  - Persistent cough: recurrent laryngeal nerve irritation
  - Pain: erosion of ribs or vertebral bodies
  - Cardiac manifestations:
    - Valvular insufficiency
    - Narrowing of coronary ostia
    - Heart failure from aortic incompetence
  - Life-threatening: aortic dissection or rupture 
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Flowchart: Thoracic Aortic Aneurysm Consequences 

Thoracic aortic dilation →

- Compression of airway/esophagus → breathing/swallowing difficulties
  - Nerve irritation → persistent cough
  - Bone erosion → chest/back pain
  - Aortic valve / coronary ostia → valvular heart disease, heart failure
  - Dissection / rupture → acute life-threatening event
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### High-Yield Exam Points

- AAA: atherosclerosis + smoking; size >5.5 cm → repair
- Inflammatory / IgG4 AAA: younger patients, immune-mediated, steroid-responsive
- Mycotic AAA: infection accelerates rupture
- TAA: hypertension, Marfan, bicuspid valves; syphilis rare

- Compression symptoms common in TAA, rupture is catastrophic
  - Always consider systemic atherosclerosis in AAA → check for IHD & cerebrovascular disease
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## Aortic Dissection

### 1. Definition

Aortic dissection occurs when blood enters a tear in the intima and splits the medial layers, creating a false lumen.

- Can rupture through adventitia → catastrophic hemorrhage 
  - Commonly affects ascending aorta; may propagate distally
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### 2. Epidemiology & Risk Factors

Feature	Details
Age group 1	Men 40–60 years, antecedent hypertension (>90% of cases)
Age group 2	Younger patients with connective tissue disorders (Marfan, Ehlers-Danlos type IV, copper metabolism defects)
Iatrogenic	Arterial cannulation, cardiopulmonary bypass
Pregnancy	Rare (~10–20 cases/million births), usually 3rd trimester or perinatal; hormone & hemodynamic stress
Other triggers	Abrupt BP spikes (cocaine use), ECM abnormalities

Note: Dissection is less likely in severe atherosclerosis due to medial fibrosis limiting propagation.

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### 3. Pathogenesis

Main cause: Hypertension → medial degeneration

## Mechanisms:

1. Hypertension → vasa vasorum narrowing → medial ischemia → SMC loss → weak media
2. Connective tissue disorders → abnormal ECM → medial weakness
3. Acute trigger: intimal tear → blood dissects along laminar planes of media
4. Rare: disruption of vasa vasorum → intramural hematoma without intimal tear

Key concept: Aggressive antihypertensive therapy may slow dissection progression.

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## 4. Morphology

- Tear location: ascending aorta, within 10 cm of aortic valve
- Tear features: transverse/oblique, 1-5 cm, jagged edges
- Dissection plane: middle → outer third of media

- Propagation: retrograde to heart or distal to iliac/femoral arteries
- Double-barreled aorta: blood re-enters lumen via second tear → false lumen becomes endothelialized → chronic dissection
- Histology: cystic medial degeneration → SMC dropout, elastic fragmentation, amorphous proteoglycans; inflammation absent

## 5. Classification of Dissections

Type	Location	DeBakey Classification	Management
Type A / Proximal	Ascending aorta ± descending aorta	DeBakey I (ascending + descending) / II (ascending only)	Emergency surgery + antihypertensives

Type B / Distal	Begins beyond subclavian artery	DeBakey III	Medical management: antihypertensives
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## 6. Clinical Features

Classic symptom:

- Sudden tearing/stabbing chest pain → radiates to back between scapulae → may move downward with progression

Life-threatening complications:

- Rupture: pericardial, pleural, or peritoneal cavity → death
- Retrograde dissection: aortic root → valvular damage, coronary compression → tamponade, aortic insufficiency, MI

Other complications:

- Extension → great arteries, renal, mesenteric, iliac → ischemia
  - Spinal artery compression → transverse myelitis
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## 7. Flowchart: Aortic Dissection Pathophysiology

Hypertension / ECM defect / connective tissue disorder → Medial weakening → Intimal tear →

- Blood dissects media → False lumen formation →
  - Propagation proximal → Type A → cardiac complications, tamponade, aortic insufficiency → surgical emergency
  - Propagation distal → Type B → renal/mesenteric/iliac ischemia → usually managed medically
  - Rupture → massive hemorrhage → death 
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## High-Yield Exam Points

- Most common site of origin: ascending aorta near aortic valve
  - Two major patient groups: older hypertensive men, young with connective tissue disorders
  - Classic pain: sudden, tearing, radiates to back
  - Type A = surgical emergency, Type B = medical management
  - Cystic medial degeneration is hallmark in connective tissue disease-related dissections
  - Acute triggers: BP spikes, iatrogenic injury, pregnancy-related stress
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-> The End <-