ANEURYSMS AND DISSECTIONS

Aneurysm

- Iocalized abnormal dilation of artery or heart
- Types:
- 1-"true" aneurysm
- all three layers of arterial wall or attenuated wall of heart
- → e.g. Atherosclerotic, syphilitic, congenital aneurysms, ventricular aneurysms following transmural MI



2- "false" aneurysm

- →(a.k.a pseudo-aneurysm)
- → a breach in vascular wall leading to hematoma communicating with intravascular space ("pulsating hematoma")
- →E.g. ventricular rupture after MI contained by pericardial adhesion
 →E.g. a leak at the junction of a vascular graft with a natural artery.



- aneurysms are classified by macroscopic shape and size:
 Note: shape and size are not specific
- for any disease or clinical manifestations

1- Saccular aneurysms - spherical outpouchings -involving only a portion of vessel wall

- often contain thrombi.



2- Fusiform aneurysms

- diffuse, circumferential dilation of a long vascular segment
- they vary in diameter and length and can involve extensive portions of artery





Aortic aneurysms

- > The two most important causes are:
- 1- atherosclerosis :
- most common cause
- intimal plaques compress underlying media
- >compromise nutrient and waste diffusion into arterial wall
- →media degeneration and necrosis
- thinning and weakening of media
 dilation of vessel

2- cystic medial degeneration of arterial media.

causes include: trauma; congenital defects

 (e.g., *berry* aneurysms); hereditary defects in
 structural components (Marfan); infections
 (*mycotic* aneurysms); vasculitis.

Abdominal Aortic Aneurysm

- Atherosclerotic aneurysms occur most frequently in
 <u>abdominal</u> aorta (= AAA)
- common iliacs, arch, and descending parts of thoracic aorta can also be involved
- Pathogenesis
- ▶ m/c men
- rarely < age 50.</p>
- Atherosclerosis is a major cause of AAA

- other contributors include: hereditary defects in structural components of the aorta (e.g., defective fibrillin production in Marfan disease affects elastic tissue synthesis)
- an altered balance of collagen degradation and synthesis mediated by local inflammatory infiltrates and the destructive proteolytic enzymes

AAA- Morphology

- Usually below renal arteries and above bifurcation of aorta
- can be saccular or fusiform
- as large as 15 cm in diameter, and as long as 25 cm.
- Microscopically: atherosclerosis; thinning of media
- frequently contains a laminated mural thrombus

Abdominal aortic aneurysm and complications A: rupture B: thrombosis



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Mycotic aneurysms

- Infection of a major artery that weakens its wall is called a *mycotic aneurysm*
- can originate from:
- (1) embolization of a septic thrombus (infective endocarditis)
- (2) extension of adjacent suppurative process
- (3) circulating organisms infecting arterial wall

Syphilitic Aneurysm

- *Caused by* The spirochetes *T. pallidum*
- A rare complication (early recognition and treatment of syphilis)
- Tertiary stage of syphilis can cause *obliterative endarteritis* of vasa vasorum of aorta
- ischemic medial injury
- aneurysmal dilation of aorta and aortic annulus
- eventually valvular insufficiency.

The clinical consequences of AAA

- **Rupture** \rightarrow massive hemorrhage
 - risk is directly related to size ($\geq 5 \text{ cm}$)
 - mortality for unruptured aneurysms =5%,
 - if rupture mortality rate > 50%
- ► Obstruction of downstream vessel→ ischemic injury
- **Embolism** \rightarrow mural thrombus
- compression on adjacent structures (e.g. ureter or vertebrae)
- **abdominal mass** (often pulsating)

Arterial dissection

- Extravasation of blood that enters the wall of artery through an intimal tear, as a hematoma dissecting between its layers.
- often but not always aneurysmal.
- Both true and false aneurysms as well as dissections can rupture, often with catastrophic consequences



Aortic dissection

- A catastrophic event whereby blood dissects apart the media to form a blood-filled channel within aortic wall
- Complications are :
 - massive hemorrhage
 - cardiac tamponade (hemorrhage into the pericardial sac).

Aortic dissection



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Pathogenesis of Aortic dissection

- <u>1- Hypertension</u> is *the* major risk factor
- pressure-related mechanical injury and/or ischemic injury.
- <u>2- inherited or acquired connective tissue</u> <u>disorders causing abnormal vascular ECM</u>
- (e.g., Marfan syndrome, Ehlers-Danlos syndrome, vitamin C deficiency, copper metabolic defects)

Marfan syndrome

- the most common <u>among inherited or acquired</u> <u>connective tissue disorders</u> assosiated with aortic dissection
- autosomal dominant disease of fibrillin, an ECM scaffolding protein required for normal elastic tissue synthesis.
- skeletal abnormalities (elongated axial bones); ocular findings (lens subluxation); cardiovascular manifestations

Aortic dissections are generally classified into two types:

I- The more common (and dangerous) proximal lesions (called *type A dissections*), involving either the ascending aorta only or both the ascending and descending aorta (types I and II of the DeBakey classification)



2- Distal lesions not involving the ascending part and usually beginning distal to the subclavian artery (called *type B* dissections or **DeBakey type III**





Figure 9–21 Classification of dissections. Type A (proximal) involves the ascending aorta, either as part of a more extensive dissection (DeBakey type I), or in isolation (DeBakey type II). Type B (distal, or DeBakey type III) dissections arise after the takeoff of the great vessels.

Clinical course

- Previously, aortic dissection was typically fatal, but the prognosis has markedly improved.
- Rapid diagnosis and institution of intensive antihypertensive therapy, coupled with surgical procedures involving plication of the aorta permits survival of 65% to 75% of patients