



# Cardiovascular System - Pathology Lab

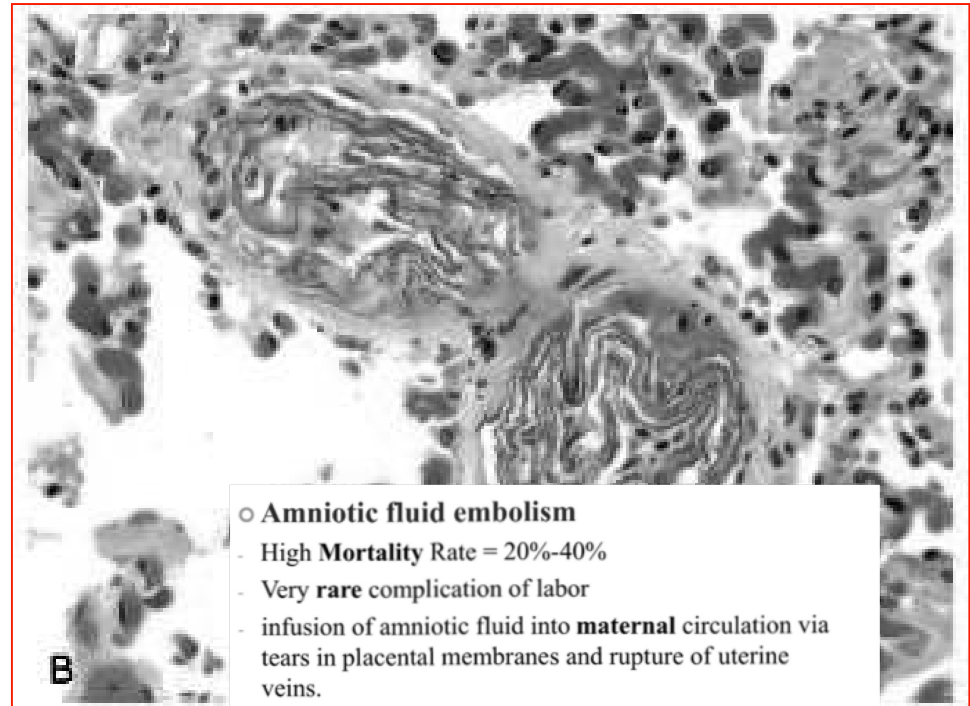
**3<sup>rd</sup> year medical students**

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**Answered file- Ayat Mohsen & Noor Momani**

A 26 years old pregnant woman had a complicated C/S. 12 hours post her C/S, she started to develop shortness of breath, decreased consciousness, And seizures before she died. This is a microscopic section from her lung.

Amniotic fluid embolus: keratin and fetal squamous cells in pulmonary arterioles

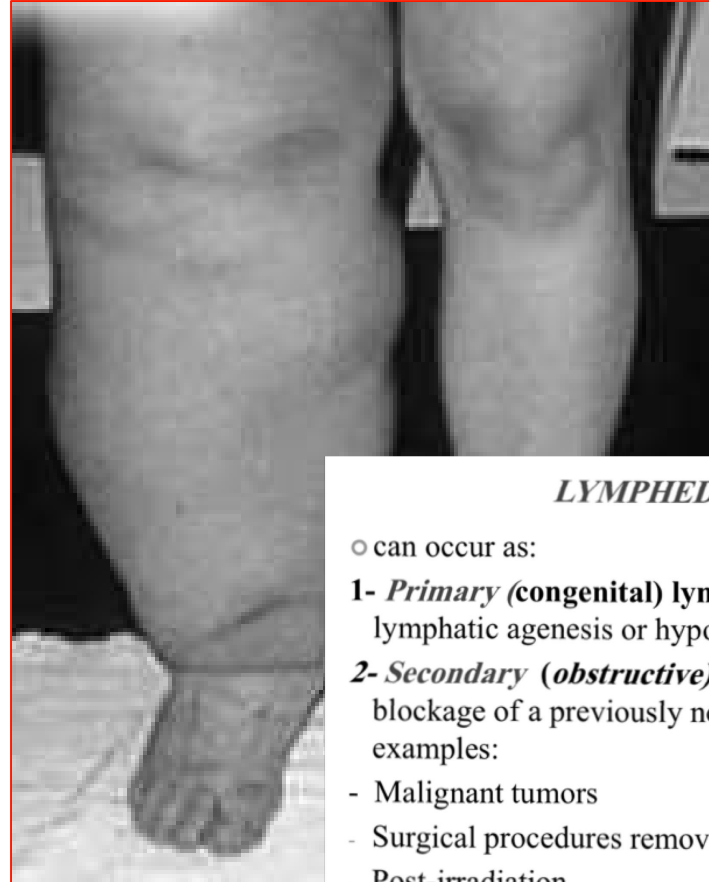


○ **Amniotic fluid embolism**

- High **Mortality Rate** = 20%-40%
- Very **rare** complication of labor
- infusion of amniotic fluid into **maternal** circulation via tears in placental membranes and rupture of uterine veins.
- **Symptoms:** sudden severe dyspnea, cyanosis, ARDS, and hypotensive shock, followed by seizures, DIC and coma
- **Microscopic Findings** upon autopsy:  
fetal squamous cells, lanugo hair, fat, mucin .....etc within the maternal pulmonary microcirculation

# Lymphedema

- Name major types.
- Give examples on causes



## *LYMPHEDEMA*

○ can occur as:

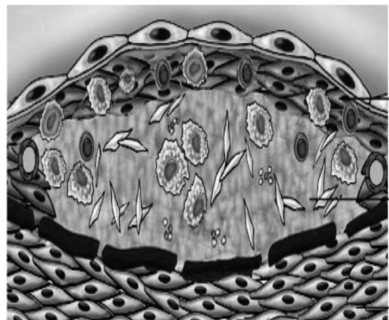
- 1- Primary (congenital) lymphedema** → lymphatic agenesis or hypoplasia.
- 2- Secondary (obstructive) lymphedema** → blockage of a previously normal lymphatic examples:
  - Malignant tumors
  - Surgical procedures removing lymph nodes
  - Post-irradiation
  - Fibrosis
  - Filariasis
  - Postinflammatory thrombosis and scarring

It's an atheroma

## Atherosclerosis

- Name parts of this lesion (**red** and **black** arrows)
- Describe the composition of each part

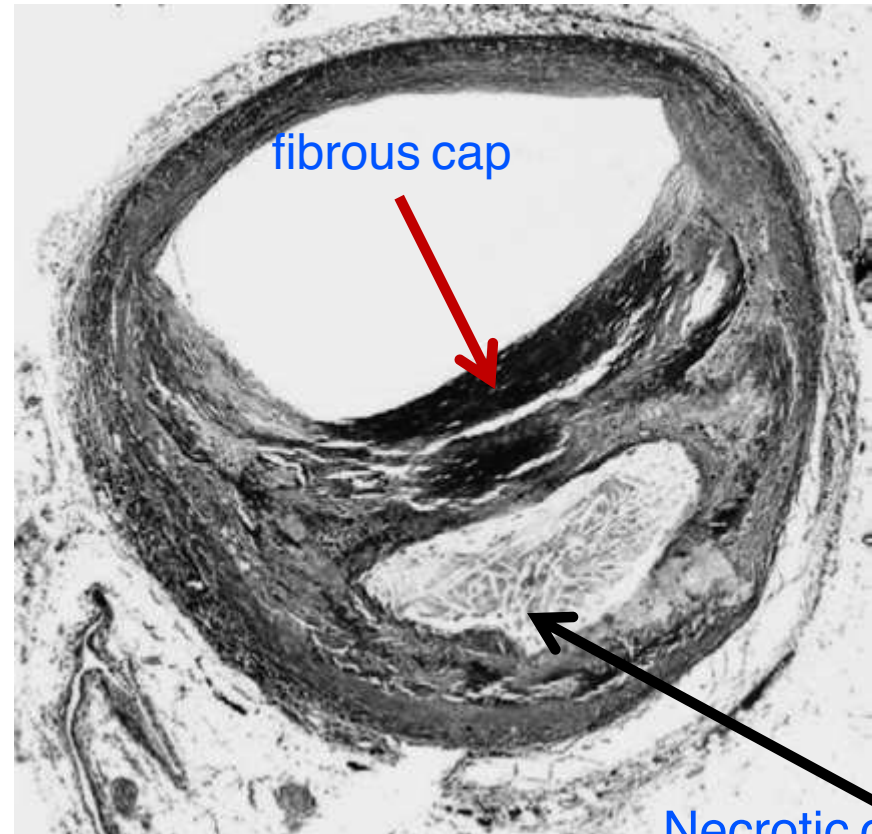
The major components of a well-developed intimal atheromatous plaque



FIBROUS CAP  
(smooth muscle cells, macrophages,  
foam cells, lymphocytes, collagen,  
elastin, proteoglycans, neovascularization)

NECROTIC CENTER  
(cell debris, cholesterol crystals,  
foam cells, calcium)

MEDIA



Necrotic core

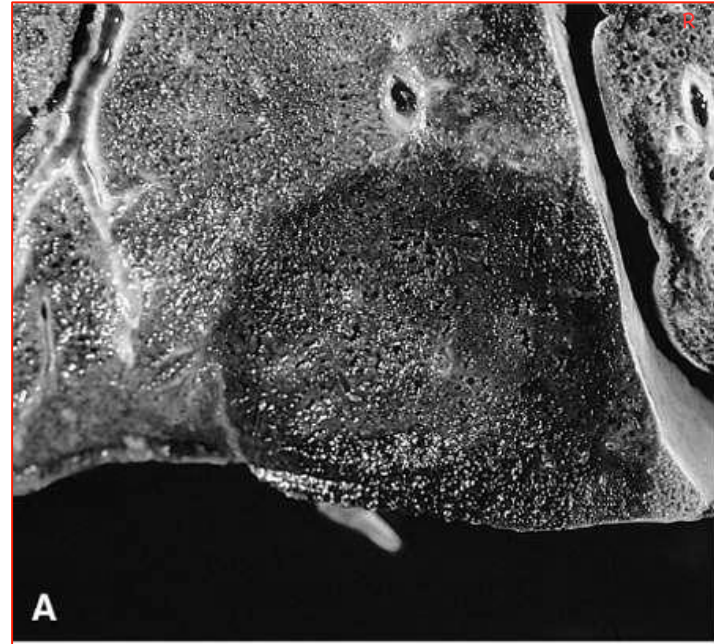
The dye used here is: Masson Trichrome, stains collagen with blue ( fibrous cap), and necrotic core with red.

# Infarction

- lung (A), and spleen (B) Solid organ

1- what type of infarct do you see in A & B? A: Red  
B: White

2- microscopic features you expect to see in A & B?



- infarct = an area of **ischemic necrosis** caused by occlusion of arterial supply or venous drainage in a tissue
- 99% of infarcts result from thromboi/emboli
- other mechanisms:

### **Vasospasm**

**extrinsic compression** (e.g., by tumor)

vessel **twisting** (e.g. testicular torsion, volvulus)

traumatic vessel **rupture**

### **RED INFARCTS:**

- **occur in any of the following scenarios:**
  - (1) **venous** occlusions (e.g. ovarian torsion)
  - (2) **loose** tissues (e.g. lung) that allow blood to collect in the infarcted zone
  - (3) tissues with **dual** circulations (e.g. lung and small intestine)
  - (4) previously congested tissues because of **sluggish venous** outflow
  - (5) when flow is **re-established** to a site of previous arterial occlusion and necrosis

### **WHITE INFARCTS**

- occur with:
  - 1) **arterial** occlusions
  - 2) **solid** organs (such as heart, spleen, and kidney).

### **MORPHOLOGY OF INFARCTS**

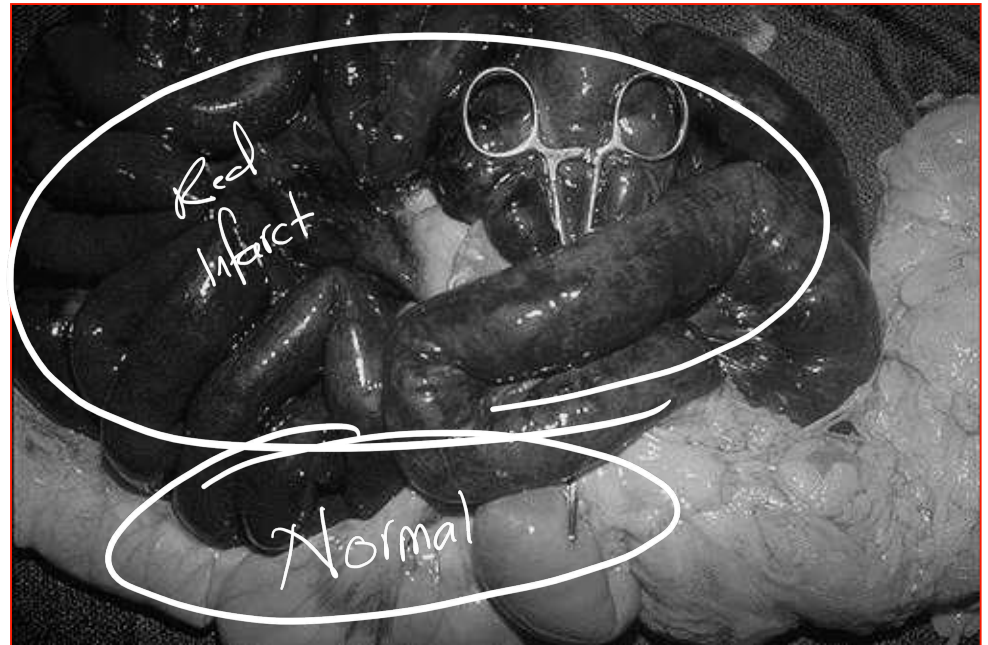
- infarcts may be either **red (hemorrhagic)** or **white (anemic)** and may be either septic or bland
- tend to be wedge shaped (occluded vessel at the apex and the periphery of the organ forming the base)
- margins of infarcts tend to become better defined with time
- histologic hallmark of infarction is **ischemic coagulative necrosis**
- note: The brain is an **exception** (liquefactive necrosis)
- most infarcts are ultimately replaced by scar

A 74 years old man was brought to ER with severe abdominal pain and rectal bleeding. Laparotomy was performed. This is a picture of His small intestines.

## Infarction

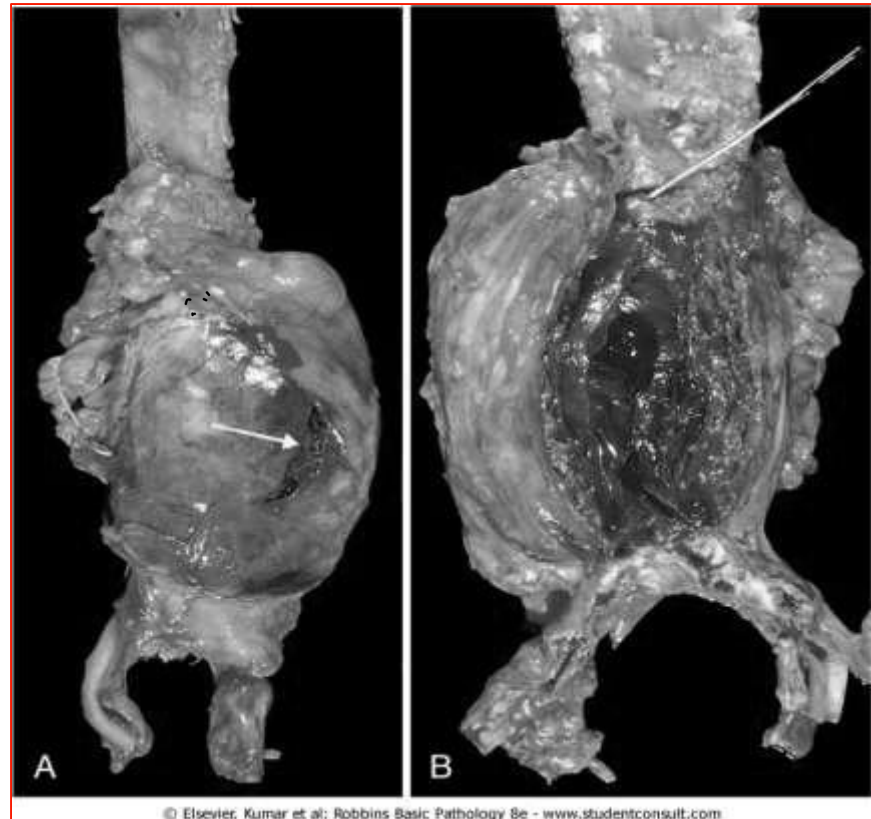
- **A case of sudden mesenteric artery occlusion.**
- **What type of infarcts is it? Why?**

Red infarct; dual blood supply



## Aortic aneurysm

- What part of the aorta is involved ?  
Abdominal, AAA
- Picture A, white arrow? rupture
- picture B? Mural thrombosis





# Abdominal Aortic Aneurysm

- ▶ (Atherosclerotic aneurysms) occur most frequently in **abdominal** aorta (= AAA)
- ▶ common iliacs, arch, and descending parts of thoracic aorta can also be involved

## ▶ Pathogenesis

- ▶ m/c in men
- ▶ rarely < age 50
- ▶ Atherosclerosis is a major cause of AAA

- ▶ other contributors include:

## 1- Hereditary defects in structural components of the aorta:

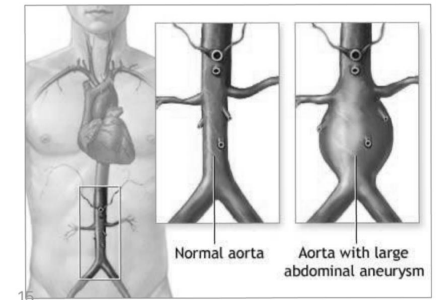
(e.g., **Marfan disease** by defective fibrillin production affects elastic tissue synthesis)

2- An altered balance of collagen degradation and synthesis mediated by local inflammatory infiltrates and the destructive proteolytic enzymes

- (e.g. vasculitis)

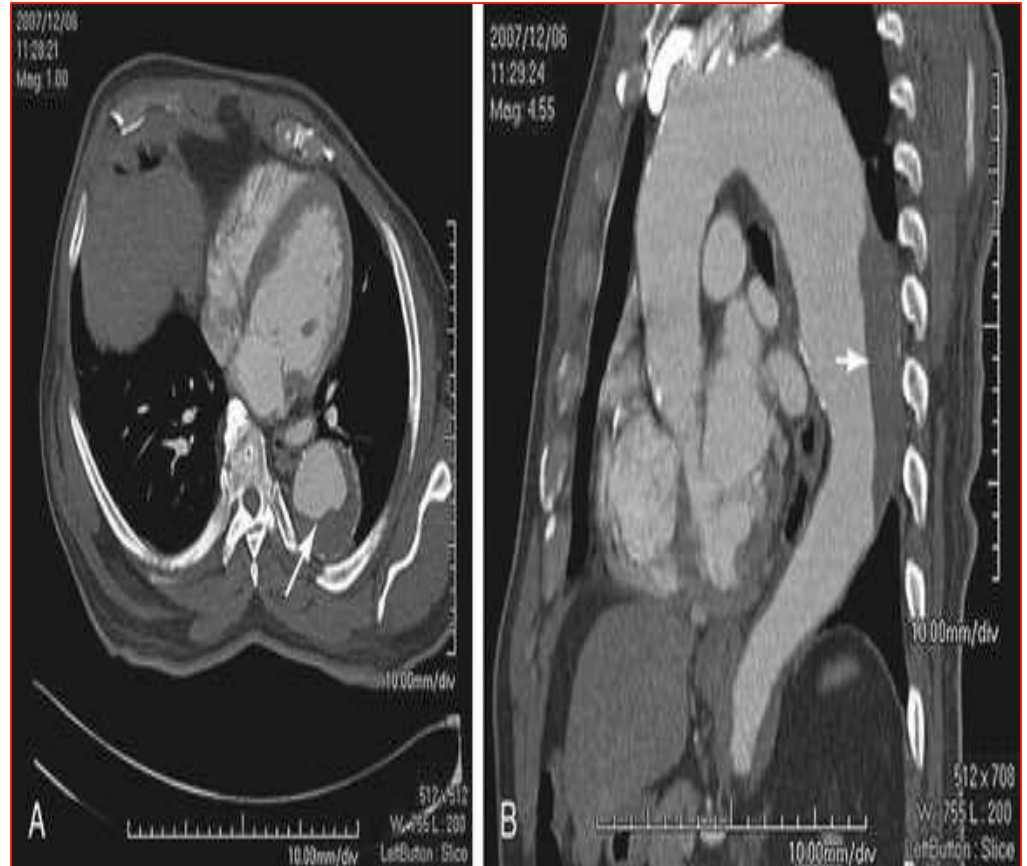
## AAA- Morphology

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



## Aortic aneurysm

- What part of the aorta is involved?  
**descending**
- white arrows represents?  
**Thrombosis**



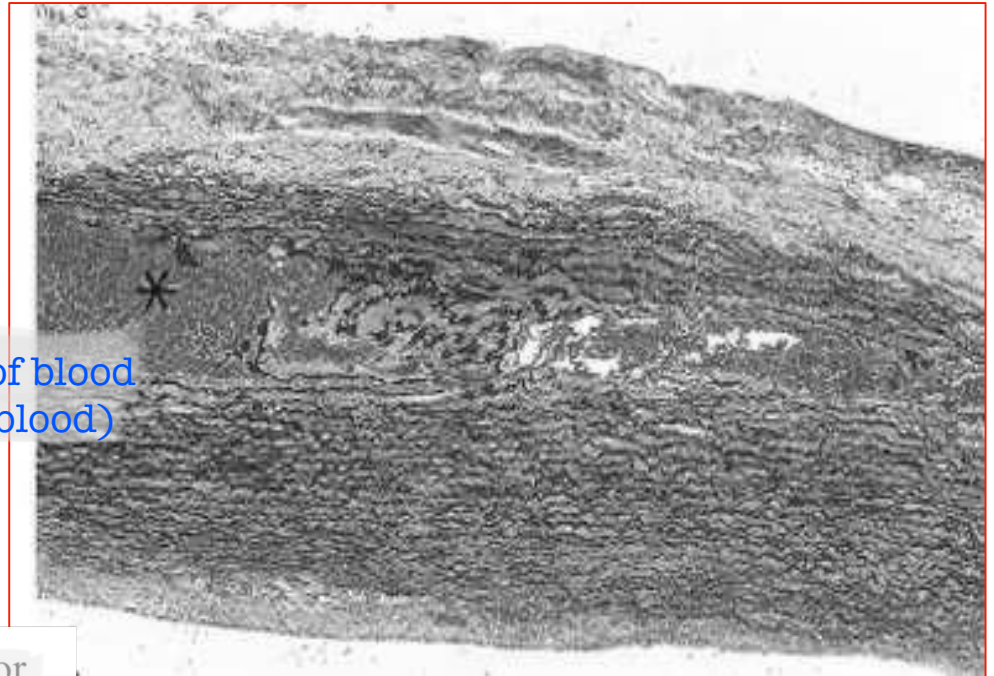
## Aortic dissection

- The special histochemical stain in this microscopic section of aorta shows elastic fibers in black color. What does the black star represents? *Collection of blood (dissected blood)*

-Name a major precipitating factor *Hypertension*

-other causes?

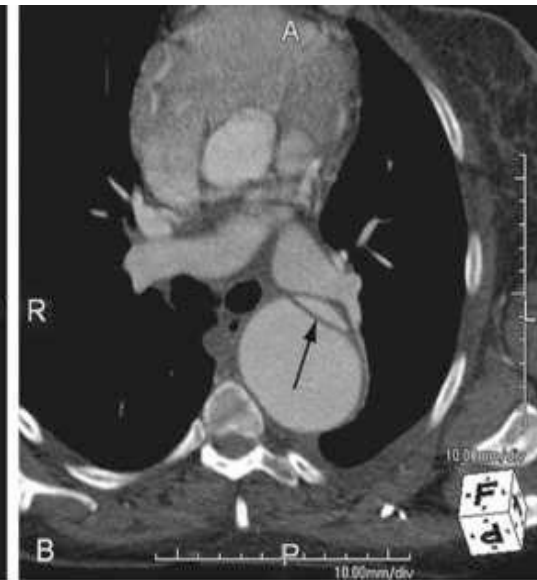
- ▶ 1- Hypertension is *the* major risk factor
- ▶ pressure-related mechanical injury and/or ischemic injury.
- ▶ 2- inherited or acquired connective tissue disorders causing abnormal vascular ECM
- ▶ (e.g., Marfan syndrome, Ehlers-Danlos syndrome, vitamin C deficiency, copper metabolic defects)



## Aortic dissection

-black arrows  
represents?

Aneurysm with dissection

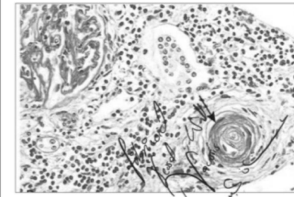


## Arteriolosclerosis

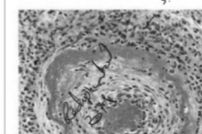
- types? A, B
- causes?

- Ass. with benign hypertension
- homogeneous pink hyaline thickening of arteriolar walls
- luminal narrowing
- leakage of plasma components across injured endothelial cells into vessel walls
- increased ECM production by smooth muscle cells in response to chronic hemodynamic stress

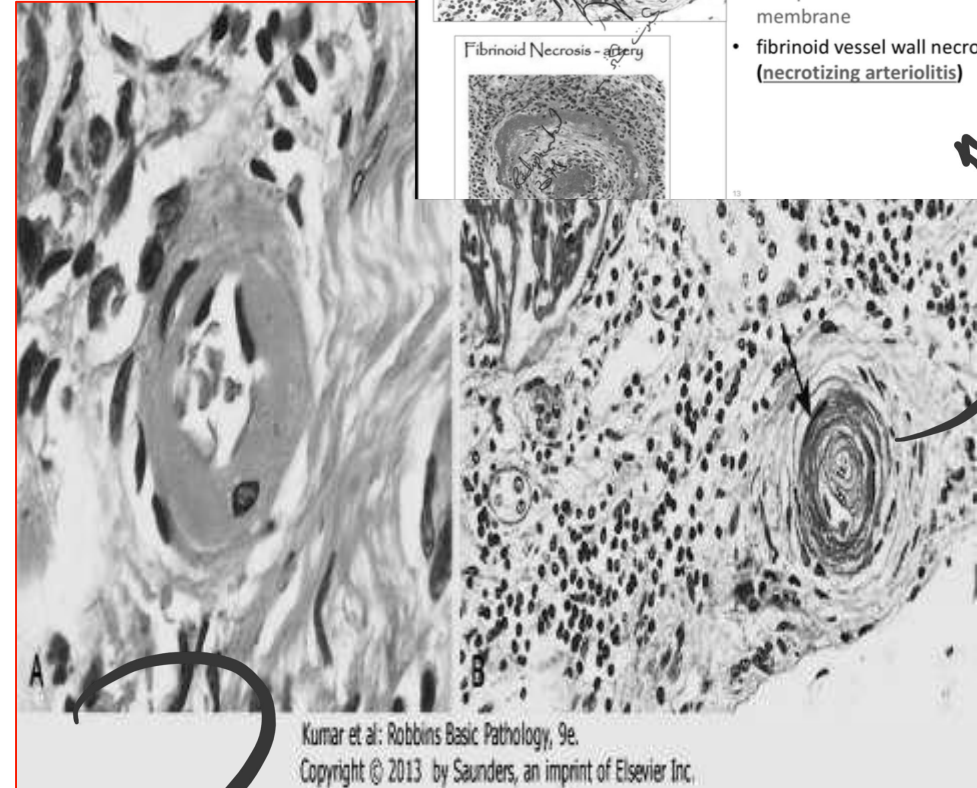
## 2- Hyperplastic arteriolosclerosis



Fibrinoid Necrosis - artery



- With severe (malignant) hypertension
- "onionskin" concentric laminated thickening of arteriolar walls
- luminal narrowing
- reduplicated basement membrane
- fibrinoid vessel wall necrosis (necrotizing arteriolitis)



## Complications of MI

**-what complication is seen in this picture (white arrow)?**

Rupture in the free wall of the Ventricle.

**-Mention the consequences that may follow this.**

Hemopericardium  
and Cardiac  
Tamponade (fatal)

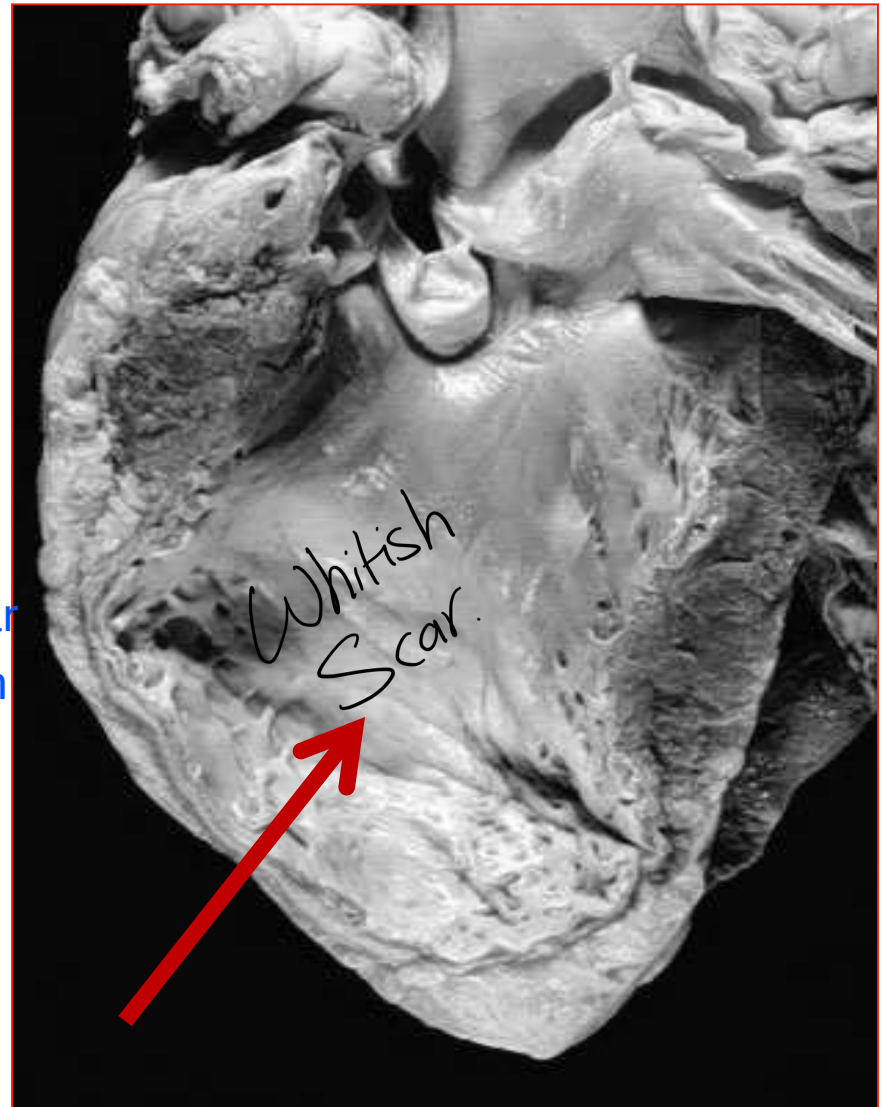


What type of aneurysm is this?  
**FALSE ANEURYSM**

## Complications of MI

- A recent or old MI? (red arrow) Old (scar).
- What post-MI complication is seen? Ventricular Aneurysm
- potential clinical consequences? Ventricular dilation.

- Complications of ventricular aneurysms include:
- 1-mural thrombus
  - 2-arrhythmias
  - 3-heart failure



Type of aneurysm?  
**TRUE ANEURYSM**

## Complications of MI

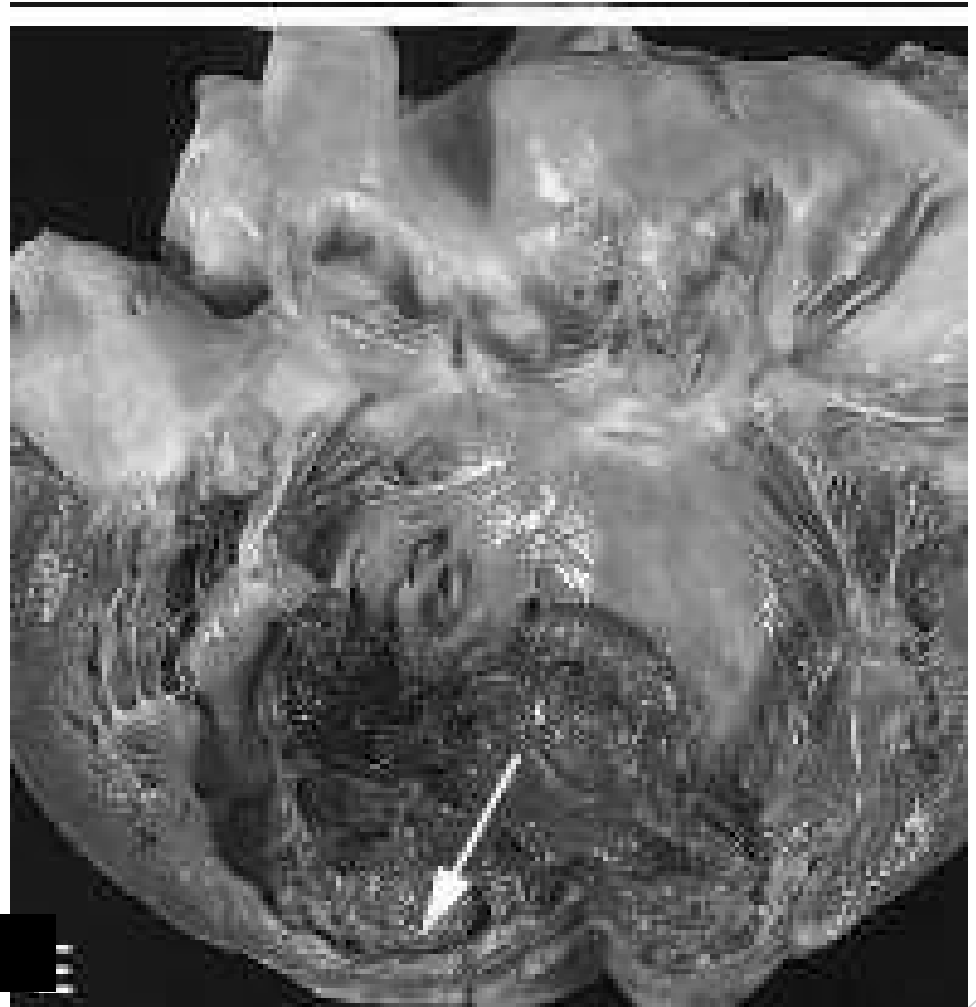
- what complication of acute MI do you see?  
rupture of papillary muscle.
- potential adverse effects ? Regurgitation if it happens in Mitral valve





## Complications of MI

-what significant complication of acute MI do you see?  
**Mural Thrombosis**



## Acute rheumatic heart disease

- **Aschoff bodies are *pathognomonic* for rheumatic fever**
- **Composition?  
Collections of T-lymphocytes, plasma cells, and macrophages**

Seen in acute phase of rheumatic fever

