

Clinical Features of MI

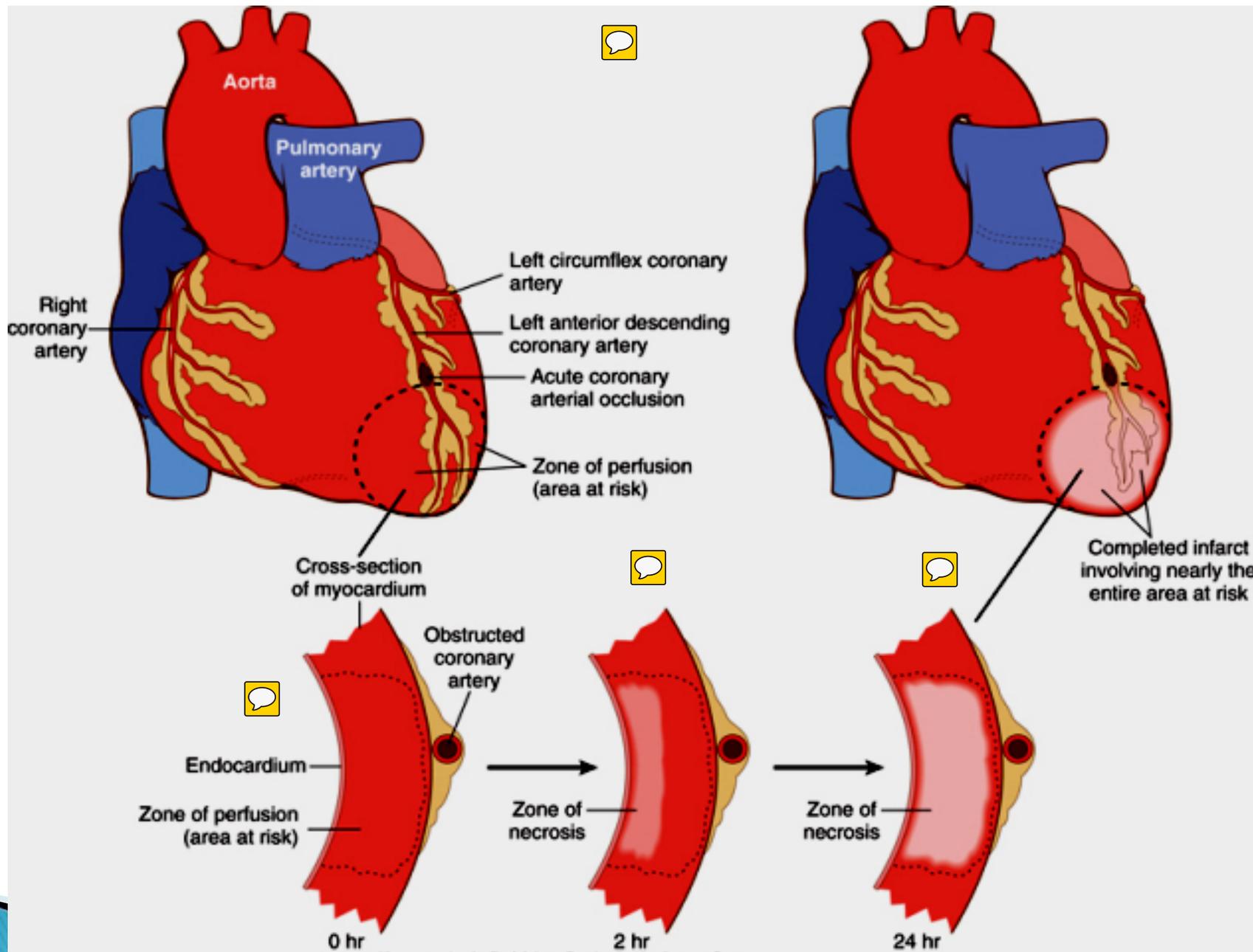
- 1) Severe, crushing substernal chest pain 
- 2) radiate to the neck, jaw, epigastrium, or left arm.
- 3- rapid and weak pulse
- 4- nausea (posterior MI).
- 5- cardiogenic shock (massive MIs >40% of the left ventricle)
- 6- dyspnea (pulmonary congestion and edema)

- ▶ 10% to 15% of MIs are asymptomatic (**silent infarcts**) → particularly in patients with:
 - 1- DM (peripheral neuropathies)
 - 2- the elderly

Myocardial Infarction

- MI = *heart attack*
- *necrosis of heart muscle due to ischemia.*
- A very significant cause of death worldwide.
- 33% -50% die before hospital → lethal arrhythmia → Sudden Cardiac Death
- Arrhythmias are caused by electrical abnormalities of the ischemic myocardium and conduction system. 

- ▶ Acute occlusion of the proximal left anterior descending (**LAD**) artery is the cause of 40% to 50% of all MIs
- *frequency of MIs rises with increasing age and presence of risk factors such as hypertension, smoking, and diabetes* 



Evaluation of MI

- ▶ *Clinical signs and symptoms*
- ▶ *Electrocardiographic(ECG) abnormalities*
- ▶ ***Laboratory evaluation:***
blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes.

Cardiac enzymes in MI

1-myoglobin.

2-cardiac troponins T and I (TnT, TnI)

3-creatine kinase (CK, and more specifically the myocardial-specific isoform, CK-MB)

4- lactate dehydrogenase

- **Cardiac troponins T and I (TnT, TnI), are the best markers for acute MI.** 
- **creatine kinase CK-MB** is the **second best** marker after the cardiac-specific troponins.

Microscopic changes of MI and its repair.

(<24 hr) → coagulative **necrosis** and **wavy fibers**. 

Necrotic cells are separated by edema fluid.

2- to 3-day old - infarct → Dense **neutrophil** infiltrate 

(7 to 10 days) → complete removal of necrotic myocytes by phagocytic **macrophages** 

up to 14 days → **Granulation tissue** characterized by loose connective tissue and abundant capillaries. 

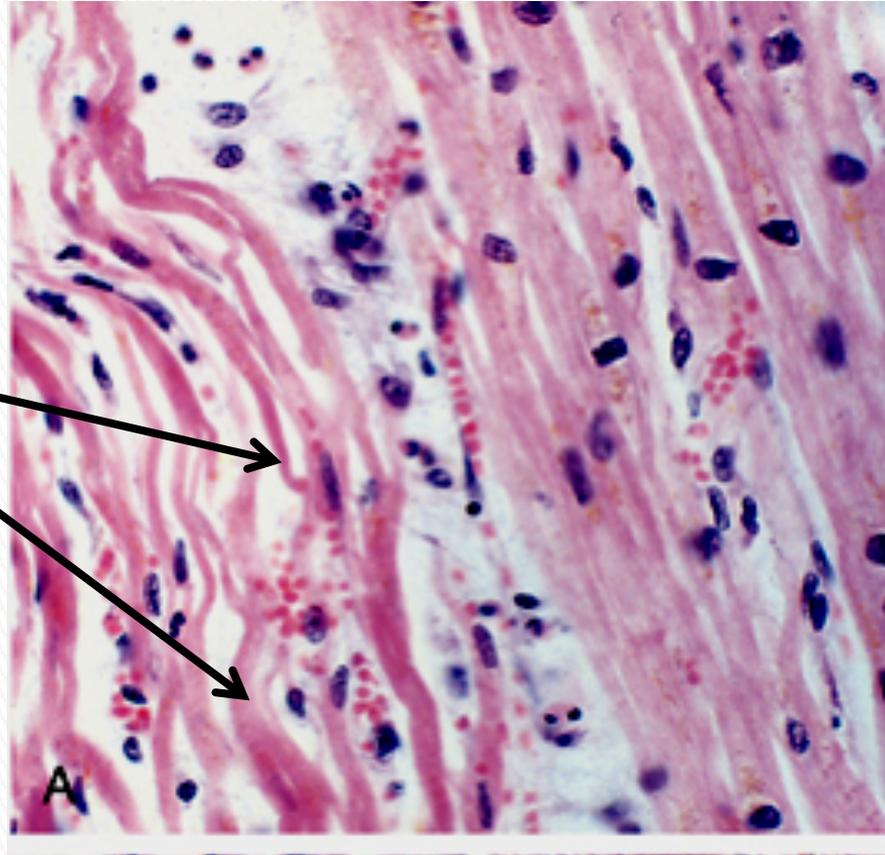
several weeks → Healed myocardial infarct consisting of a dense collagenous **scar**.

Microscopic features of myocardial infarction and its repair.

(**<24 hr**) →

coagulative **necrosis**
and **wavy fibers**

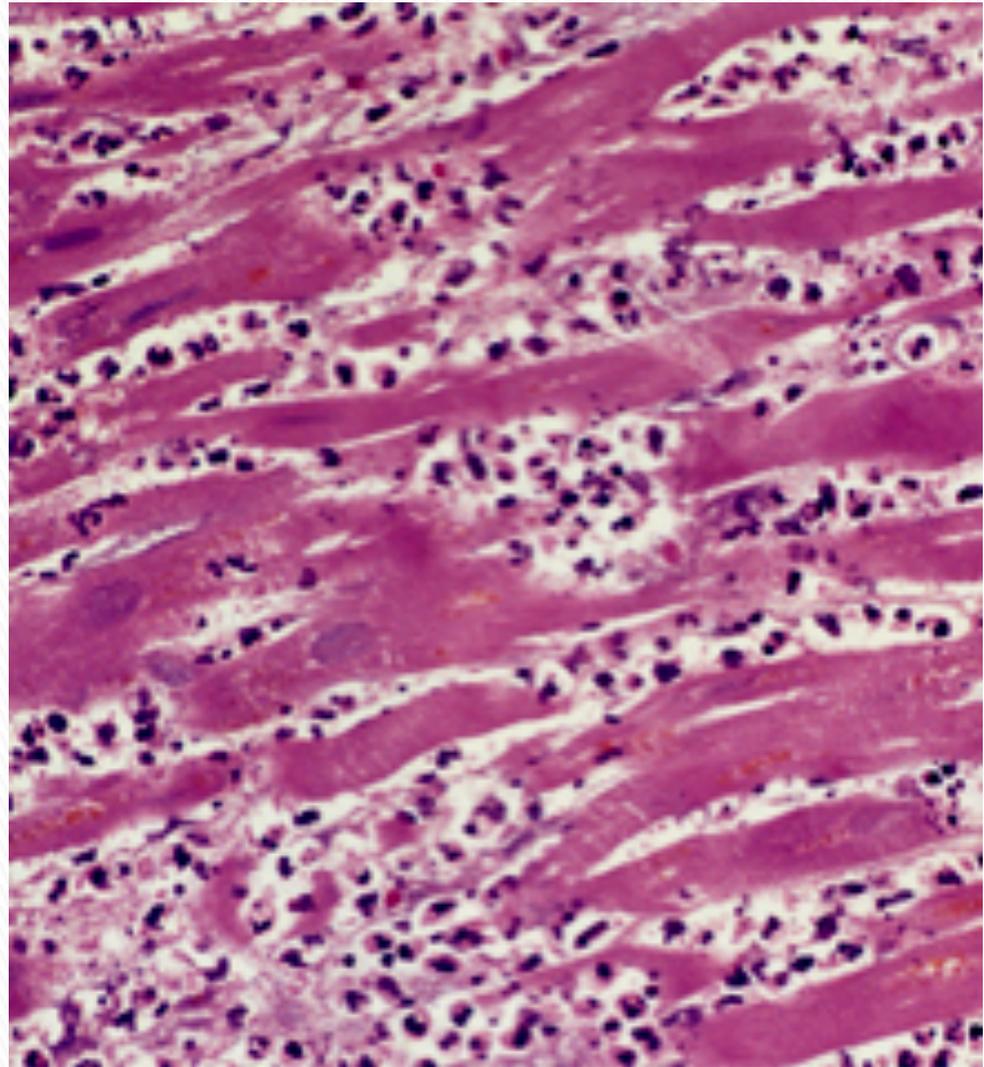
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Microscopic features of myocardial infarction and its repair.

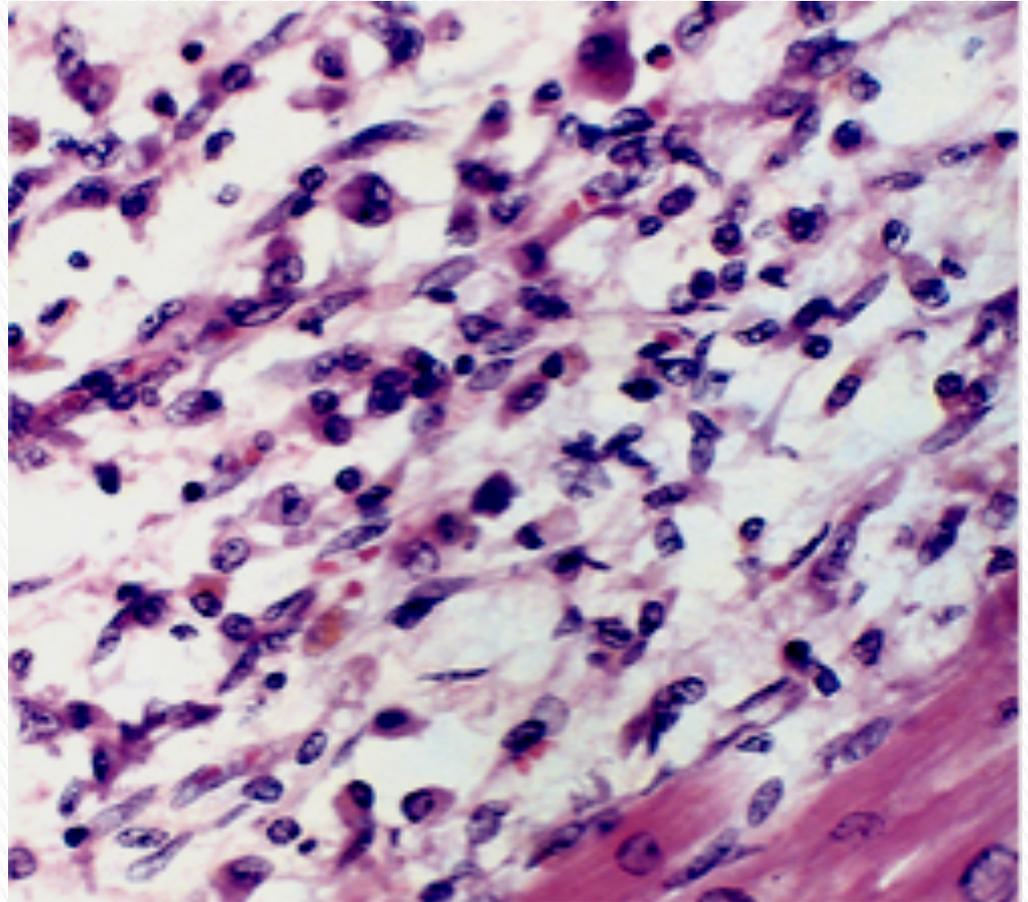
2- to 3-day old - infarct → Dense neutrophil infiltrate

• in case of reperfusion → contraction bands



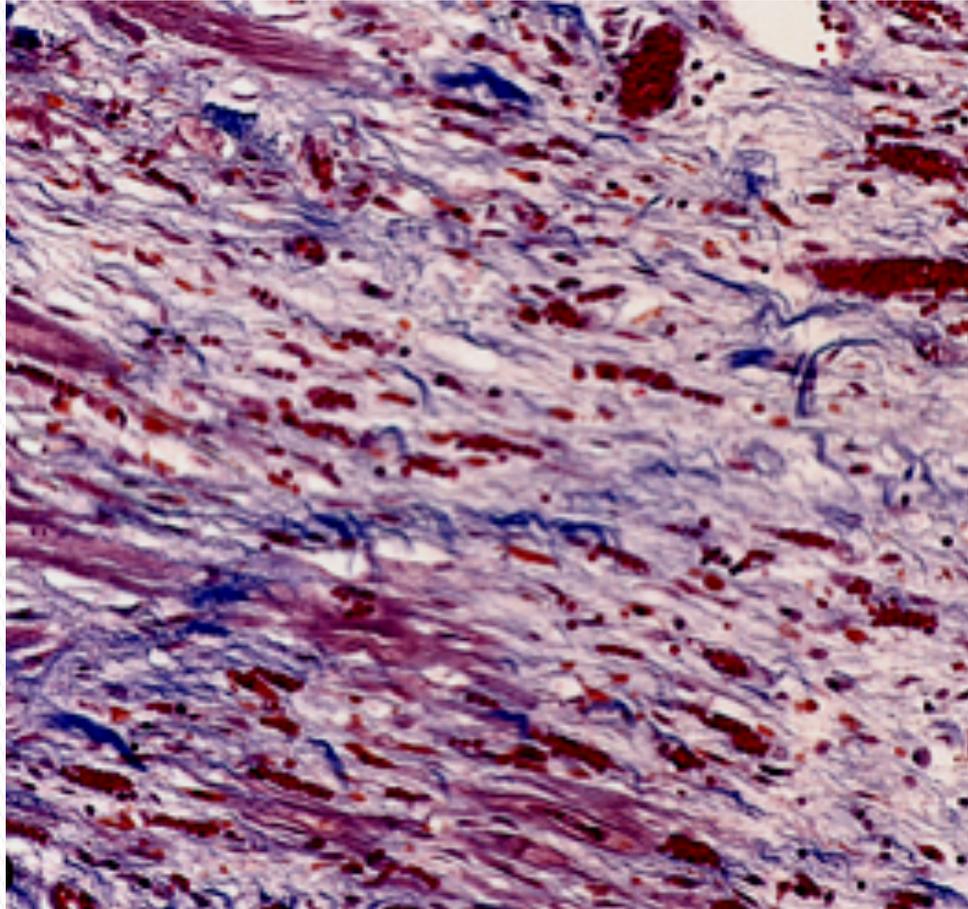
Microscopic features of myocardial infarction and its repair.

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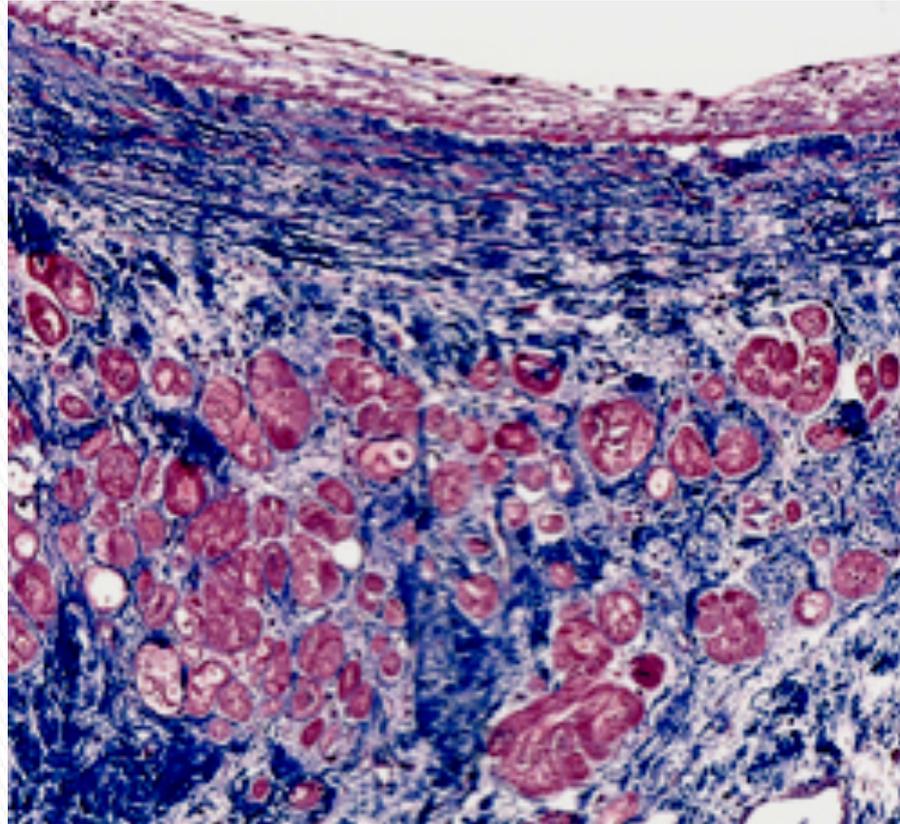
Microscopic features of myocardial infarction and its repair.

up to 14 days →
Granulation tissue
= loose connective
tissue (blue) and
abundant capillaries
(red)



Microscopic features of myocardial infarction and its repair.

several weeks →
Healed infarct
consisting of a
dense collagenous
scar (blue)



Consequences and Complications of MI

- **1- Death:** Unfortunately, 50% of the deaths associated with acute MI occur in individuals who never reach the hospital (within 1 hour of symptom onset-usually as a result of arrhythmias)
- Extraordinary progress has been made in patient outcomes subsequent to acute MI (*their-hospital death rate* has declined from approximately 30% to an overall rate of between 10% and 13%).

Consequences and Complications of MI

- 2- cardiogenic shock.
 - (10% to 15%) of patients after acute MI
 - with a large infarct (>40% of the Left ventricle).
 - 70% mortality rate; 2/3 of in-hospital deaths.
- 3-Myocardial rupture
- 4-Pericarditis.
- 5-Infarct expansion
- 6-Ventricular aneurysm
- 7-Progressive late heart failure



Complications of myocardial rupture include:



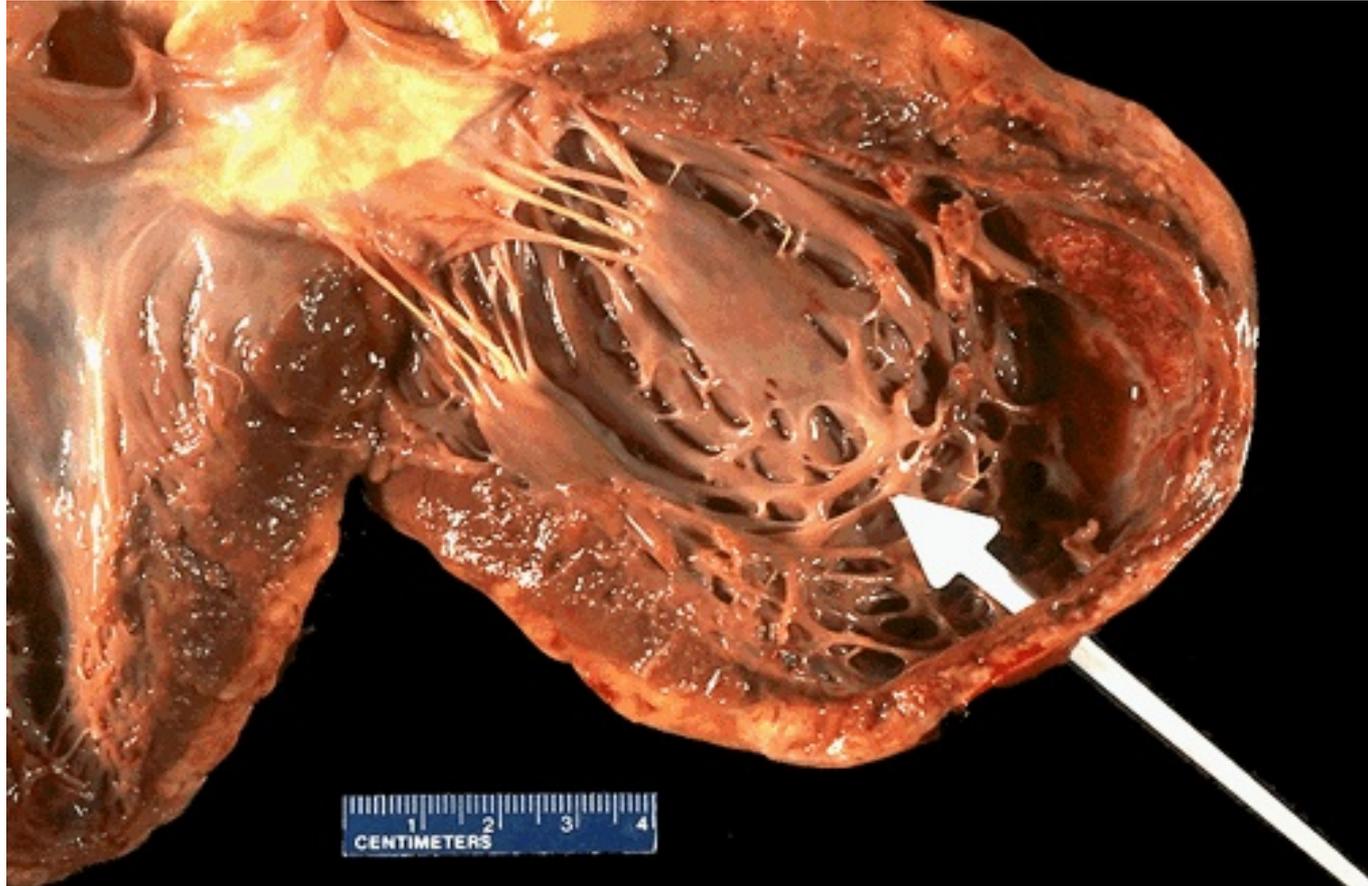
(1) rupture of the ventricular free wall → hemopericardium and cardiac tamponade (usually fatal)



(2) rupture of the ventricular septum → VSD and left-to-right shunt

(3) papillary muscle rupture → severe mitral regurgitation

myocardial rupture



- ***4-Pericarditis.*** 

- fibrinous or hemorrhagic pericarditis
- usually 2 to 3 days of a transmural MI
- typically spontaneously resolves with time (immunologic mechanism).

- ***5-Infarct expansion.*** 

Because of the weakening of necrotic muscle, there may be disproportionate stretching, thinning, and dilation of the infarct region (especially with anteroseptal infarcts)

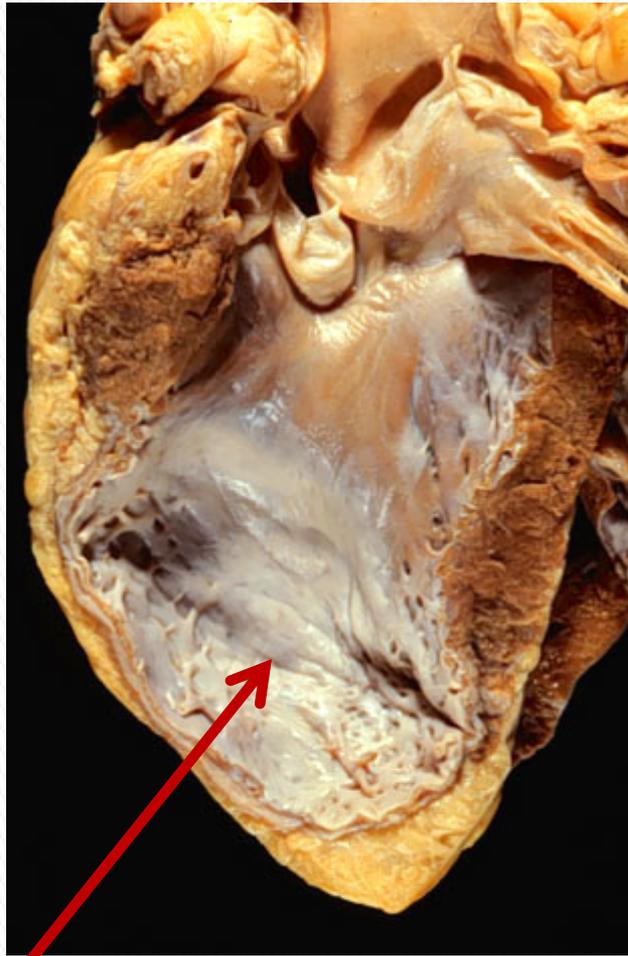
- ***6-Mural thrombus.***

- the combination of a local loss of contractility (causing stasis) + endocardial damage (causing a thrombogenic surface) → *thromboembolism*

- ***7-Ventricular aneurysm.***

- A late complication

- most commonly result from a large transmural anteroseptal infarct that heals with the formation of thin scar tissue



Ventricular aneurysm

Complications of ventricular aneurysms include:

- 1-mural thrombus
- 2-arrhythmias
- 3-heart failure



- ***8-Papillary muscle dysfunction*** (post-infarct mitral regurgitation)
- dysfunction of a papillary muscle after MI occurs due to:
 - 1- rupture.
 - 2- ischemic dysfunction
 - 3- fibrosis and shortening
 - 4- ventricular dilation.
- ***9-Progressive late heart failure***

Long-term prognosis after MI

- depends on many factors, the most important of which are left ventricular function and the severity of atherosclerotic narrowing of vessels perfusing the remaining viable myocardium.
- Mortality rate within the first year =30%
- ▶ Thereafter, the annual mortality rate is 3% to 4%.

Chronic Ischemic Heart Disease

- ▶ Chronic IHD usually results from **post-infarction** cardiac decompensation that follows exhaustion of the hypertrophic viable myocardium.
- ▶ **progressive heart failure** as a consequence of ischemic myocardial damage; sometimes punctuated by episodes of angina or MI.
- ▶ Arrhythmias are common along with **CHF**

Sudden Cardiac Death (SCD)

- ▶ Affecting some 300,000 to 400,000 individuals annually in the United States
- ▶ SCD is most commonly defined as **unexpected death from cardiac causes either without symptoms or within 1 to 24 hours of symptom onset**
- ▶ **Coronary artery disease is the most common underlying cause**
- ▶ In many adults SCD is the first clinical manifestation of IHD.
- ▶ With **younger** victims, other **non-atherosclerotic** causes are more common:

Other non-atherosclerotic causes of SCD

- ▶ Congenital coronary arterial abnormalities
- ▶ Aortic valve stenosis
- ▶ Mitral valve prolapse
- ▶ Myocarditis
- ▶ Dilated or hypertrophic cardiomyopathy
- ▶ Pulmonary hypertension
- ▶ Hereditary or acquired abnormalities of the cardiac conduction system.
- ▶ Isolated myocardial hypertrophy.
- ▶ unknown causes.