ISCHEMIC HEART DISEASE-2

Acute Myocardial Infarction

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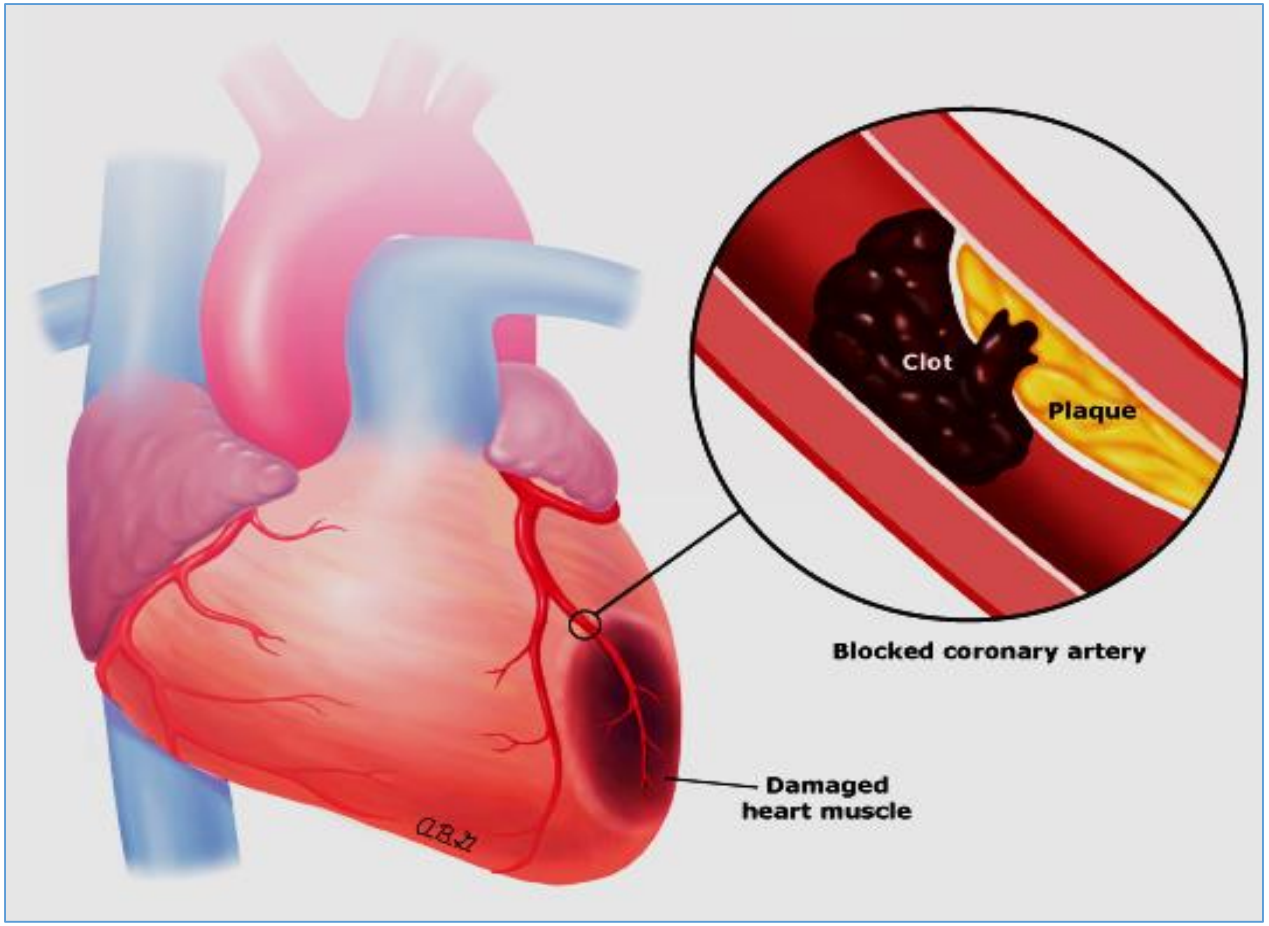
Acute Myocardial Infarction (MI)



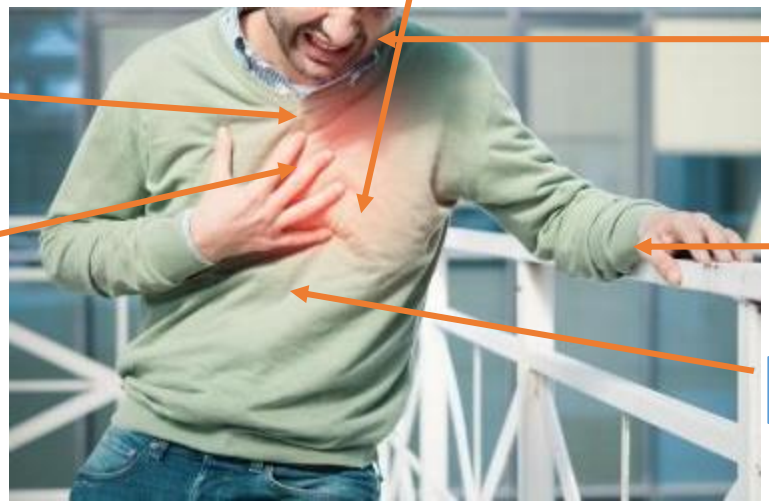
- MI = *heart attack.*
- *Necrosis of heart muscle due to ischemia.*
- A significant cause of death worldwide.

of acute MI ***Clinical Features***

Severe, crushing substernal chest pain that radiates to neck, jaw, epigastrium, or left arm

dyspnea (if pulmonary congestion and edema)

cardiogenic shock (in massive MIs >40% of left ventricle)



Dizziness; sweating

rapid and weak pulse

nausea (in posterior MI)

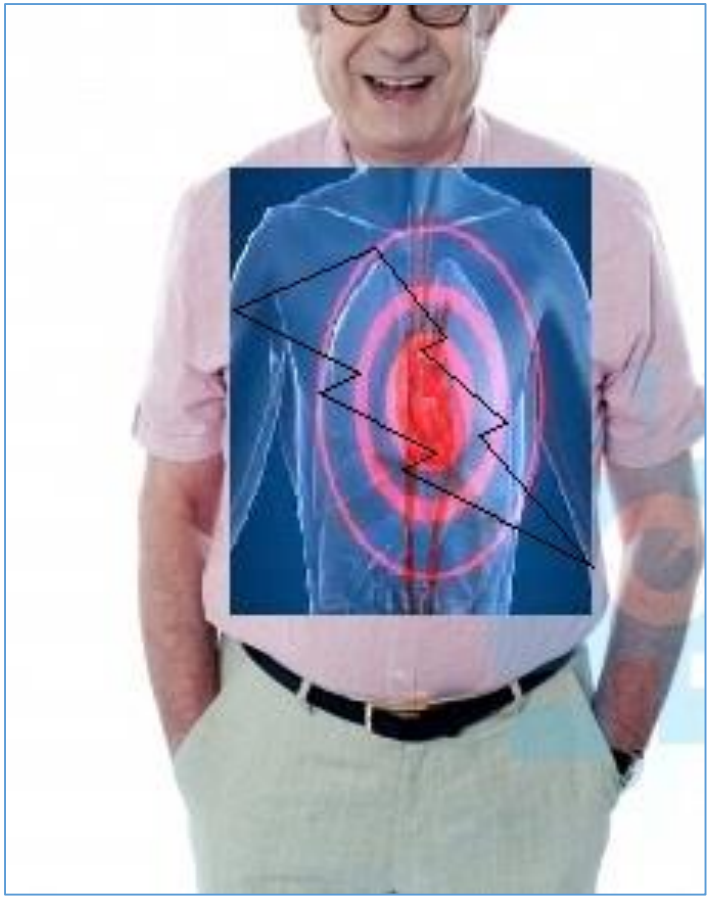
Sometimes: No typical symptoms (silent infarcts)

Silent infarcts:

- A variable percentage of MIs are asymptomatic
- Confirmed only on ECG and lab workup.
- particularly in:

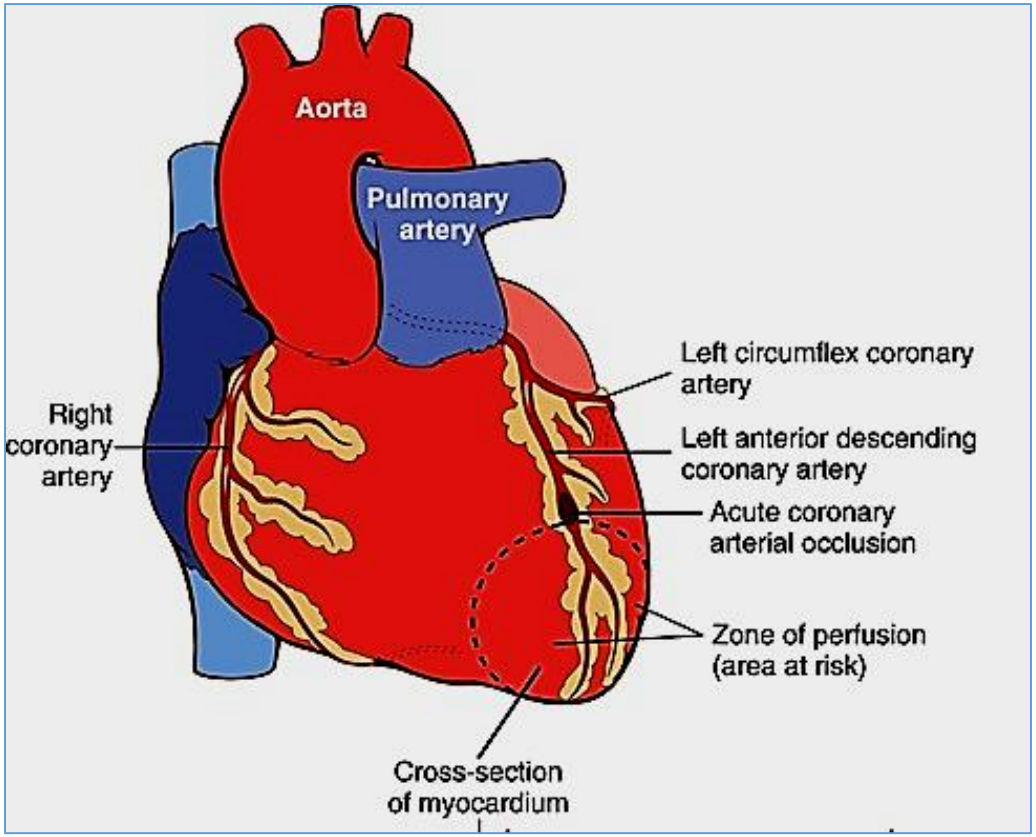
  1- DM (peripheral neuropathies)

  2- the elderly

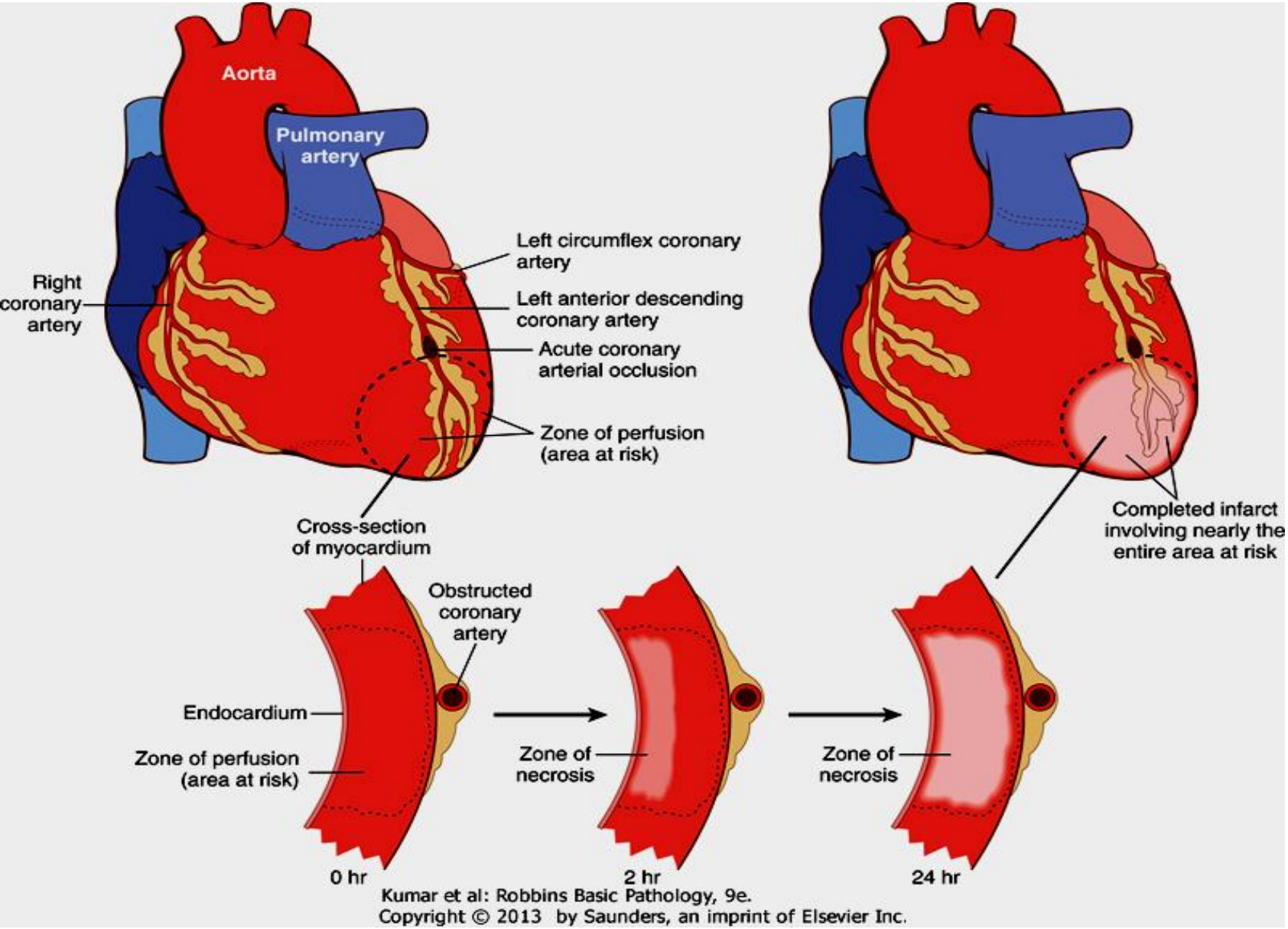


MI- Causes:

- Acute occlusion of the proximal left anterior descending (**LAD**) artery is the cause of *40% to 50% of all MI cases*



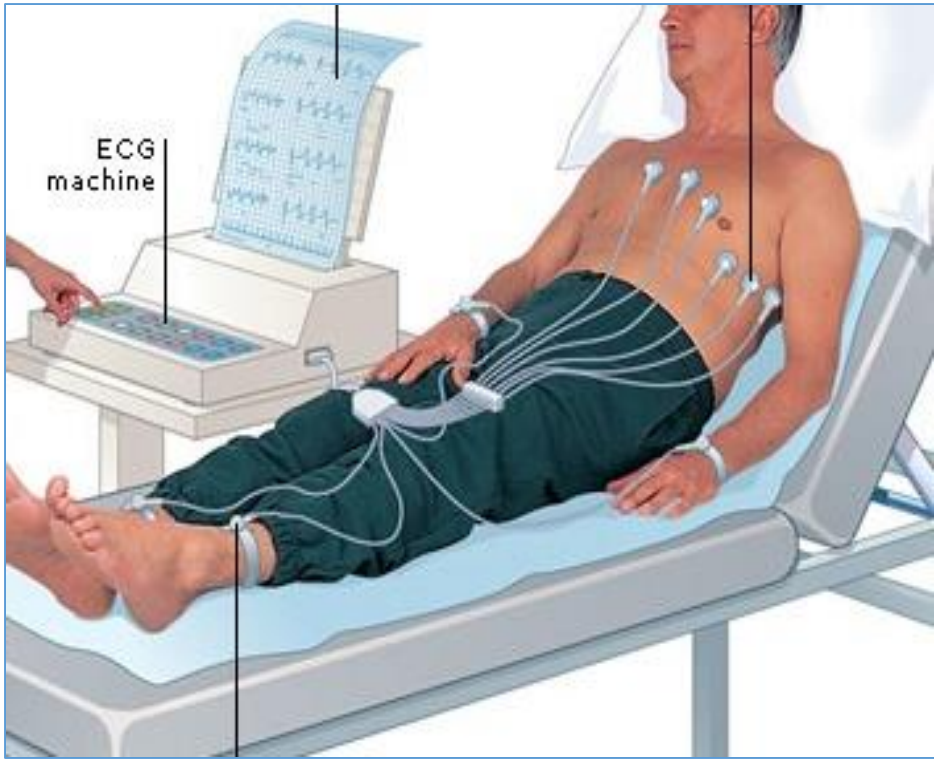
MI- Evolution



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

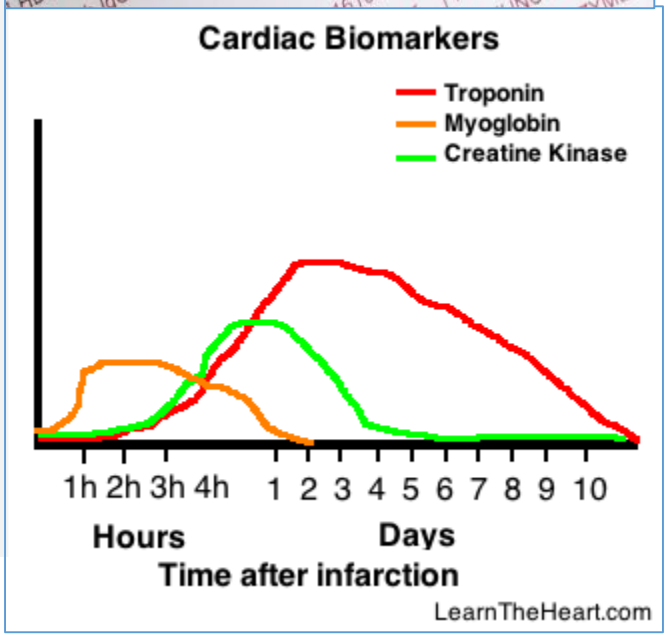
1-Myoglobin

2-Cardiac **Troponins** T and I (TnT, TnI)

3-Creatine kinase (CK); specifically the myocardial-specific isoform (CK-MB)

4- Lactate dehydrogenase

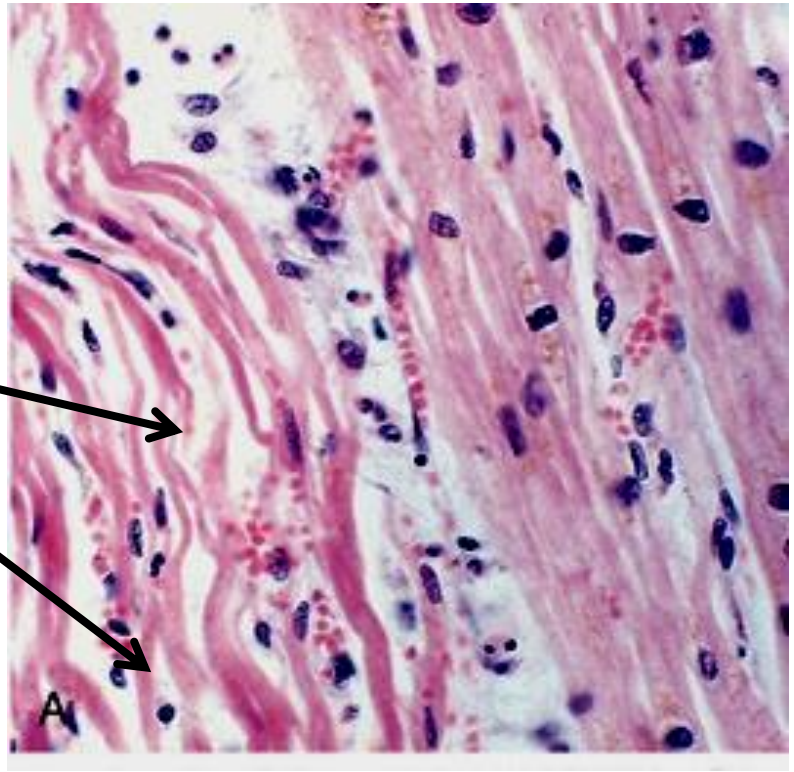
- Cardiac troponins T and I (TnT, TnI), are the best markers for acute MI.
- Creatine kinase CK-MB is the second best marker after the cardiac-specific troponins.



Microscopic features of myocardial infarction and its repair.

<24 hr:

coagulative **necrosis** and **wavy fibers** Necrotic cells are separated by edema fluid

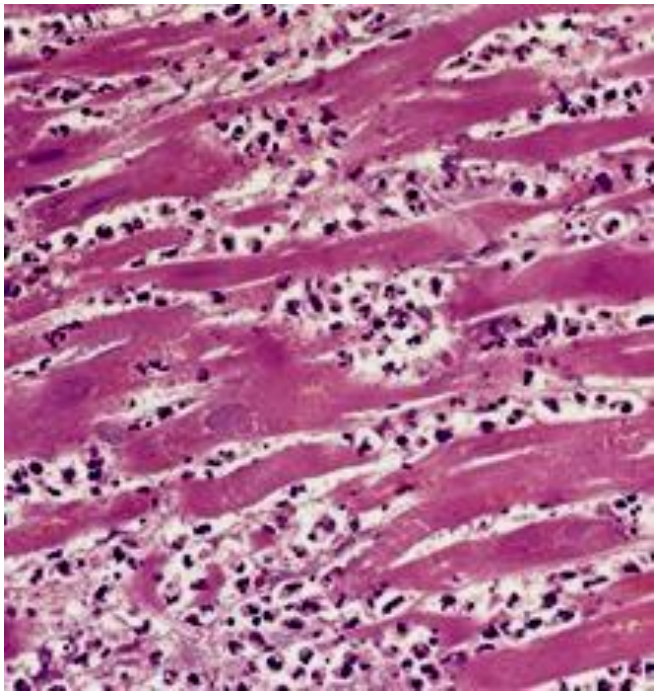


Stain: Hematoxylin & Eosin (H&E)

Microscopic features of myocardial infarction and its repair.

2 - 3 days:

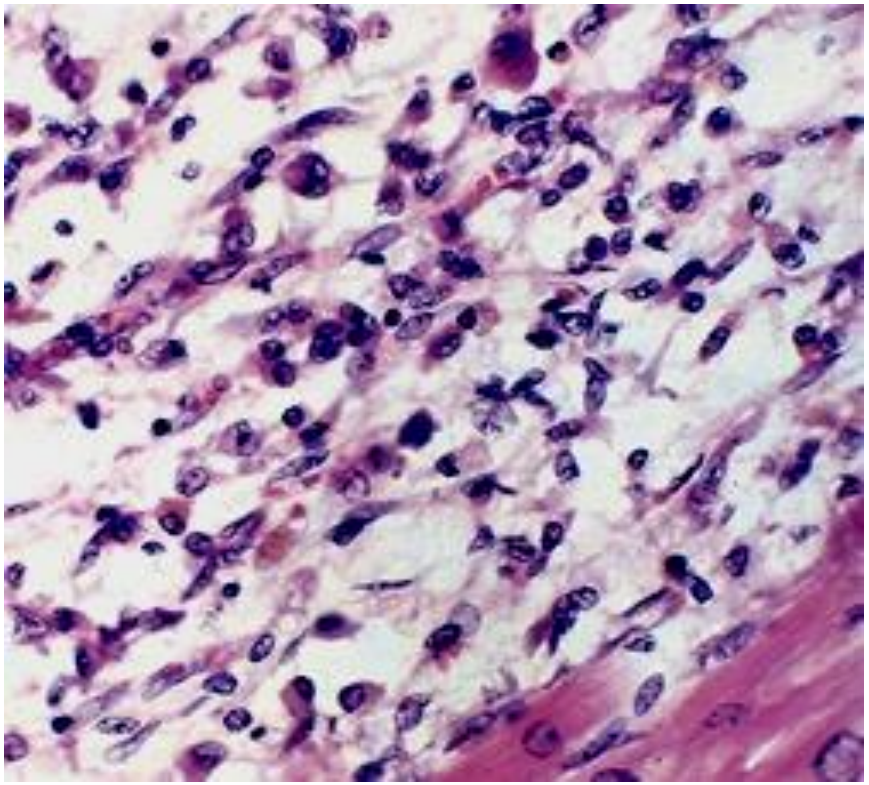
Dense **neutrophil** infiltrate



Stain: Hematoxylin & Eosin (H&E)

Microscopic features of myocardial infarction and its repair.

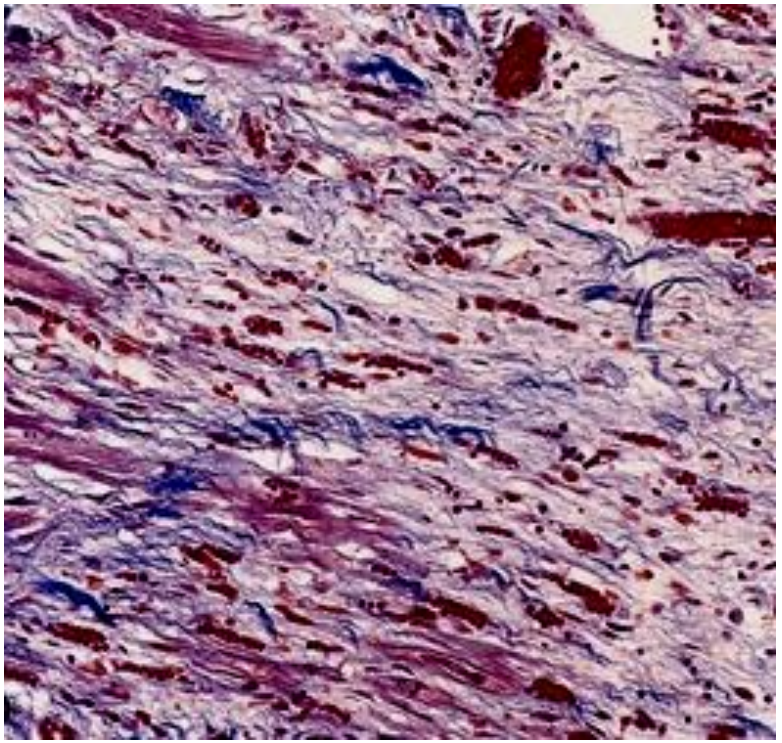
7 to 10 days:
complete removal of necrotic myocytes by **macrophages**



Stain: Hematoxylin & Eosin (H&E)

Microscopic features of myocardial infarction and its repair.

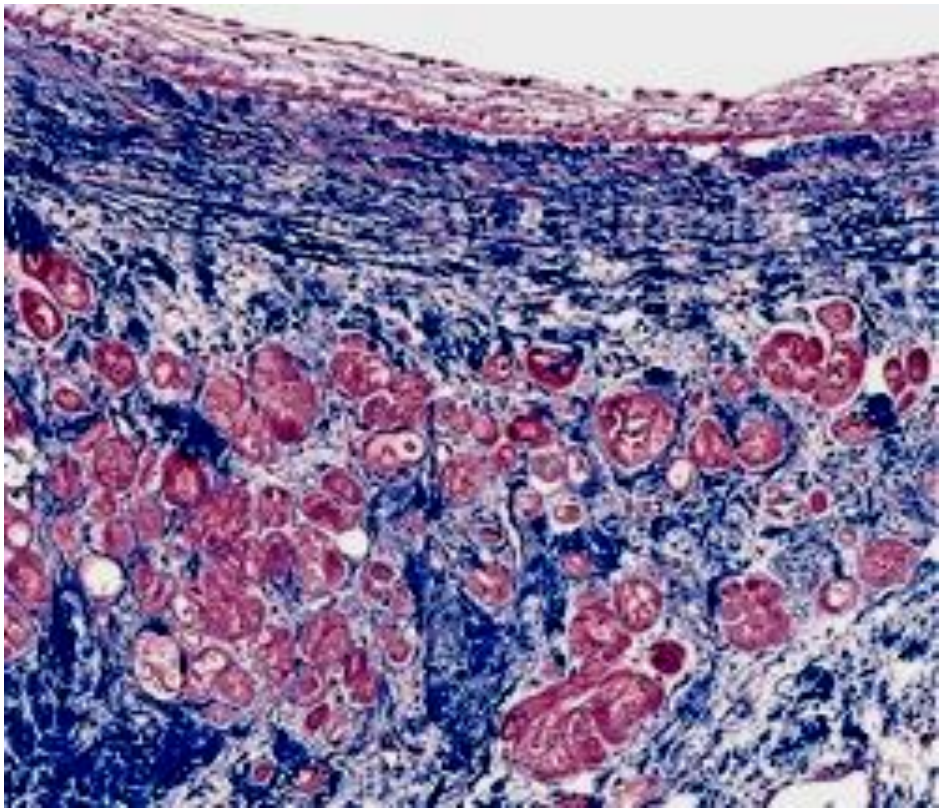
up to 14 days:
Granulation tissue
[loose connective tissue (blue) and abundant capillaries (red)]



Stain: Masson Trichrome (MT)

Microscopic features of myocardial infarction and its repair.

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



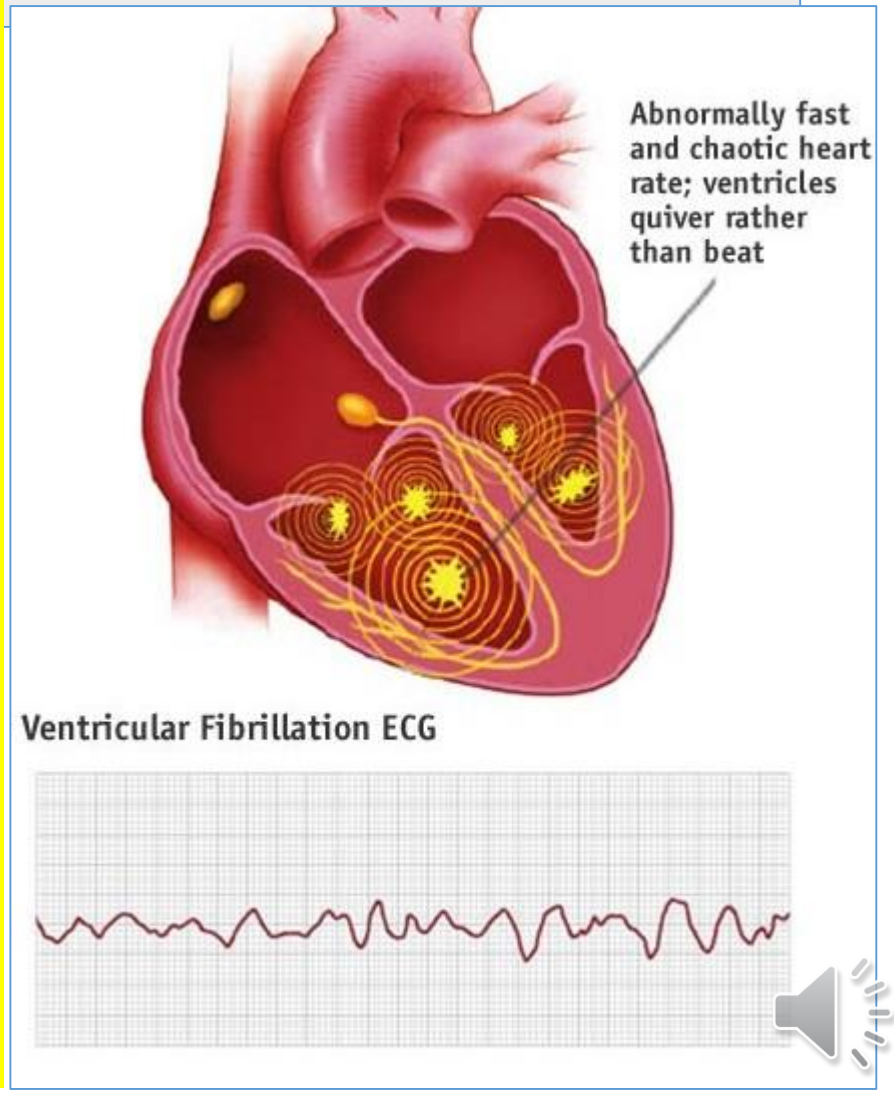
Stain: Masson Trichrome (MT)

Consequences & Complications of MI

1- Death:

- **50%** occur before reaching hospital (within 1 hour of symptom onset-usually as a result of lethal arrhythmias (Sudden Cardiac Death)

- **Arrhythmias are caused by electrical abnormalities of the ischemic myocardium and conduction system**
- With current medical care, patient outcome is better (*in-hospital death rate* has declined).



Consequences & Complications of MI

- ***2- Cardiogenic shock.***

- 15% - In large infarcts (>40% of Left ventricle).
- 70% mortality rate - important cause of in-hospital deaths.

- ***3-Myocardial rupture***
- ***4-Pericarditis***
- ***5-Infarct expansion***
- ***6- Mural thrombus***
- ***7-Ventricular aneurysm***
- ***8-Progressive late heart failure***

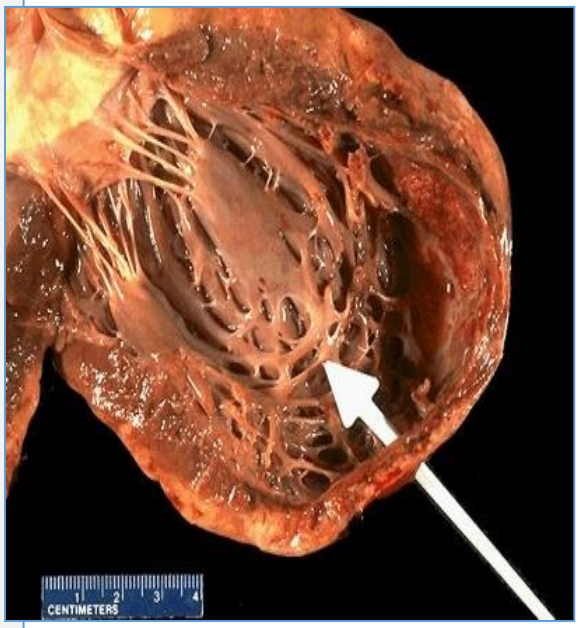
Complications of Myocardial Rupture Include:

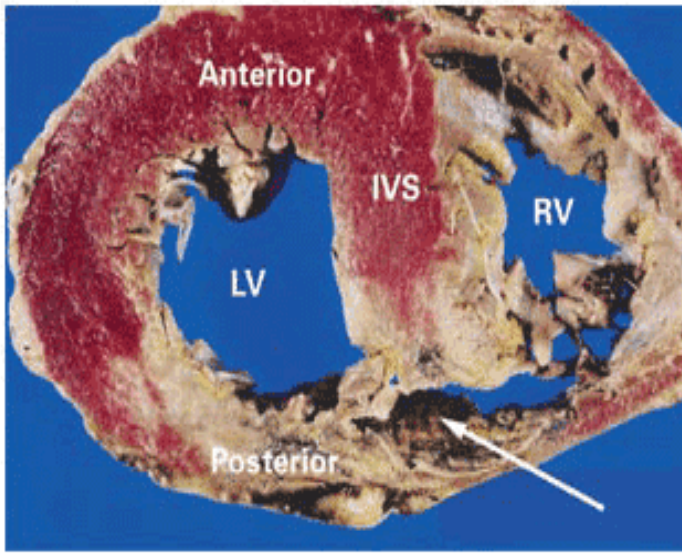
(1) rupture of the ventricular free wall: hemopericardium and cardiac tamponade (usually fatal)

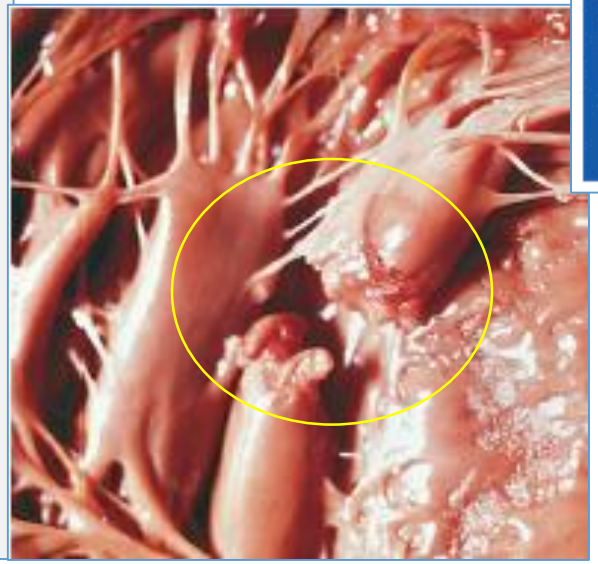
(2) rupture of the ventricular septum: VSD and left-to-right shunt

(3) papillary muscle rupture:

severe mitral regurgitation







4-Pericarditis.

- 2 to 3 days post a transmural MI
- spontaneously resolves (immunologic mechanism)

5-Infarct expansion.

disproportionate stretching, thinning, and dilation of the infarct region (especially with anteroseptal infarcts)

6-Mural thrombus.

loss of contractility (causing stasis) + endocardial damage → ***thromboembolism***

7-Ventricular aneurysm.

- A late complication

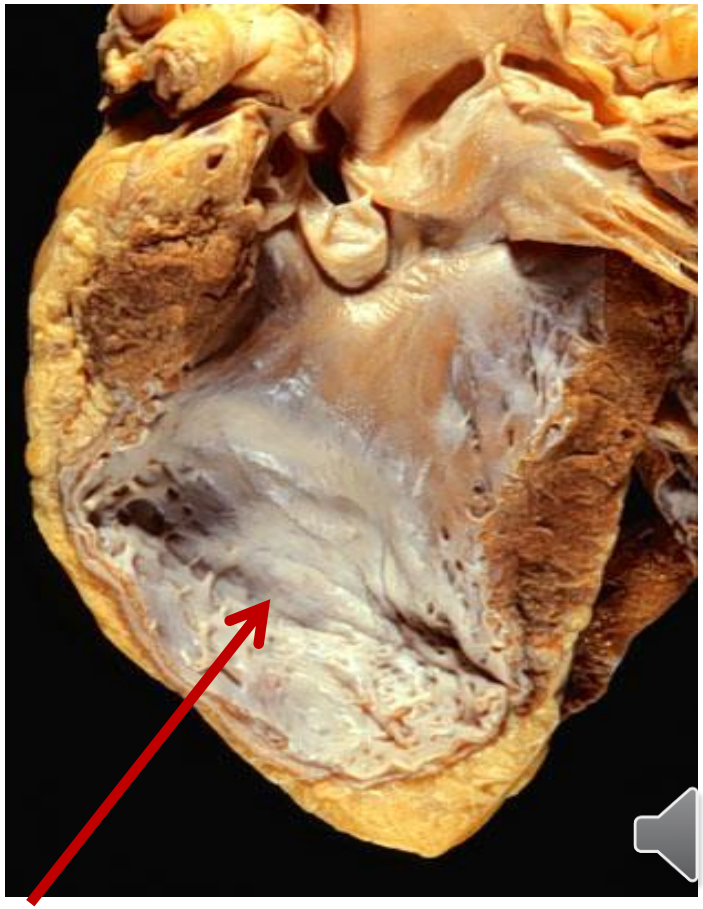
- most commonly result from a large transmural anteroseptal infarct that heals with the formation of thin scar tissue

- **Complications of ventricular aneurysms include:**

1-mural thrombus

2-arrhythmias

3-heart failure



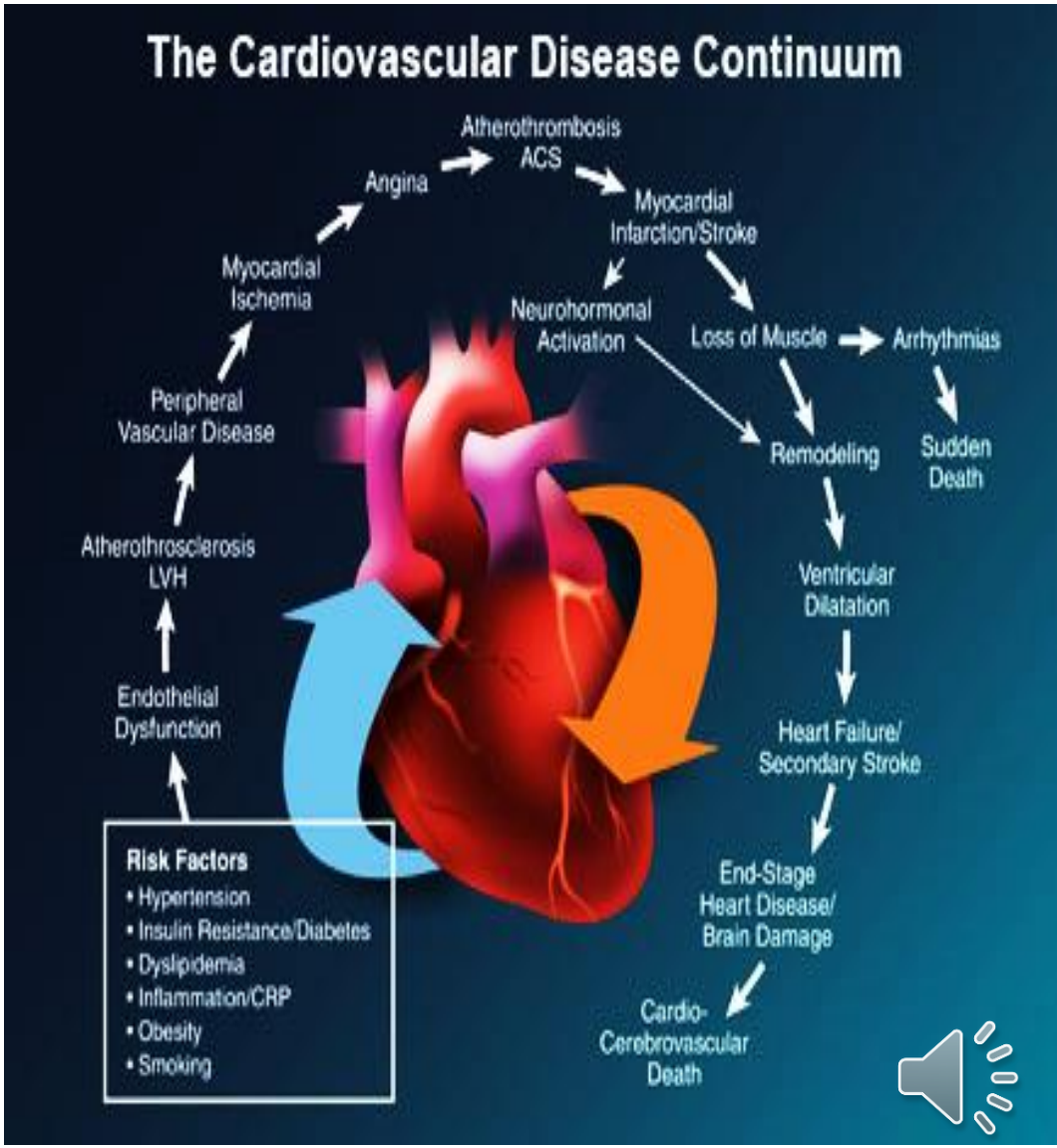
Long-term prognosis after MI

- depends on many factors: e.g. left ventricular function; severity of atherosclerosis in viable myocardium; etc...

- 1^{st} year mortality ≈ 30%.
- Thereafter, the annual mortality rate≈ 3%

Chronic Ischemic Heart Disease

- results from **post-infarction** cardiac decompensation that follows exhaustion of hypertrophic viable myocardium.
- **progressive heart failure**
- sometimes punctuated by episodes of angina or MI
- Arrhythmias are common



Sudden Cardiac Death (SCD)

- **Unexpected death from cardiac causes either without symptoms or < 24 hours of symptom onset**
- **CAD (atherosclerosis) is the most common underlying cause**
- **Lethal arrythmias (v. fibrillation) is the most common direct mechanism of death**
- With **younger** victims, other **non-atherosclerotic** causes are more common:

Non-atherosclerotic causes of SCD

- Congenital coronary arterial abnormalities
- Aortic valve stenosis
- Mitral valve prolapse
- Myocarditis
- Dilated/ hypertrophic cardiomyopathy
- Pulmonary hypertension
- Hereditary/ acquired abnormalities of cardiac conduction system
- unknown causes….