



ISCHEMIC HEART DISEASE - 2

Dr. Nisreen Abu Shahin
Associate Professor of Pathology
Pathology Department
University of Jordan

Clinical Features of acute MI



Clinical Features of MI

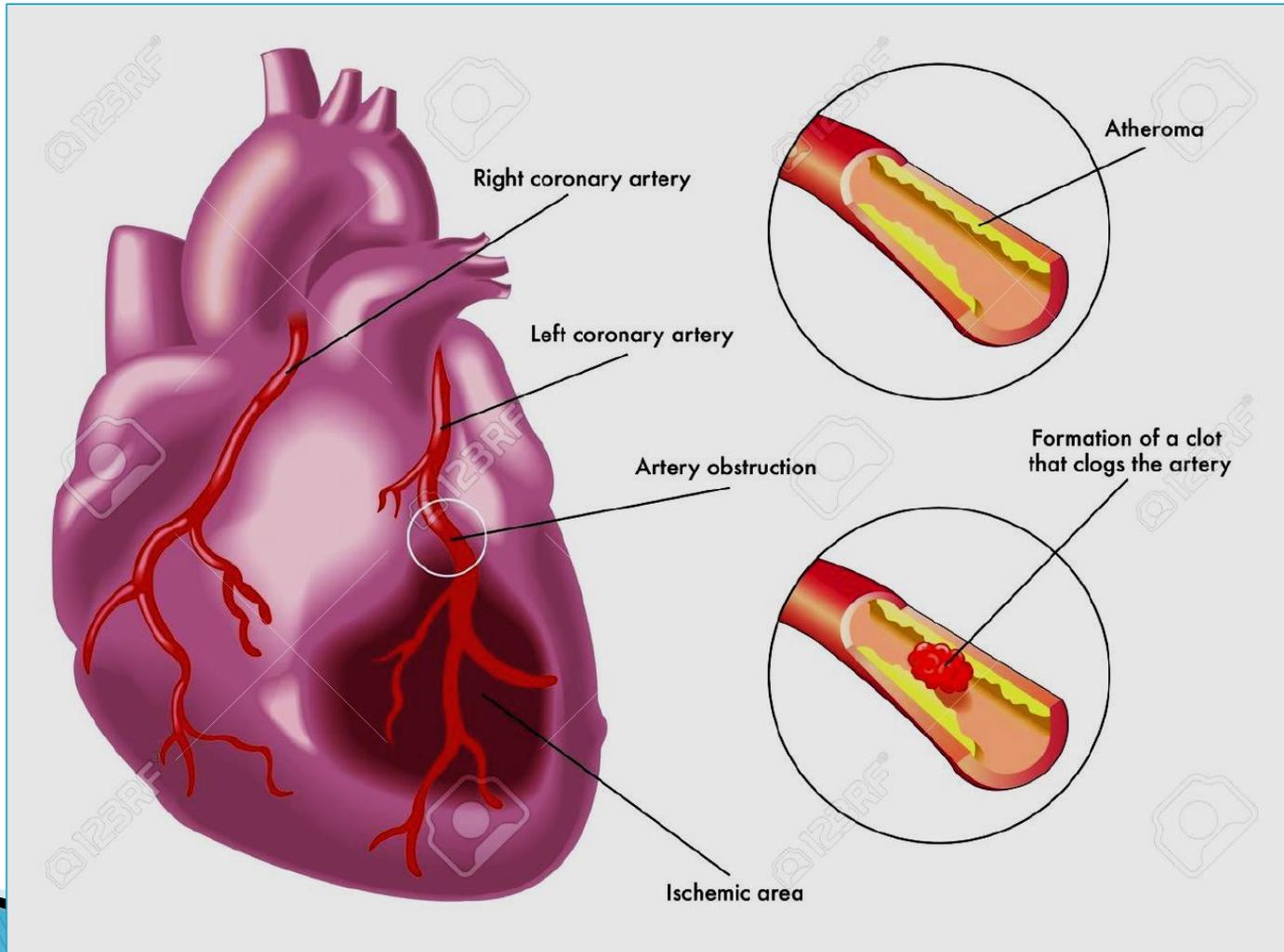
- ▶ Severe, crushing substernal chest pain
- ▶ radiates to neck, jaw, epigastrium, or left arm
- ▶ rapid and weak pulse
- ▶ nausea (posterior MI)
- ▶ cardiogenic shock (>40% of Lt ventricle)
- ▶ dyspnea (pulmonary congestion & edema)
- ▶ **silent infarcts:** (10% - 15% of MIs)

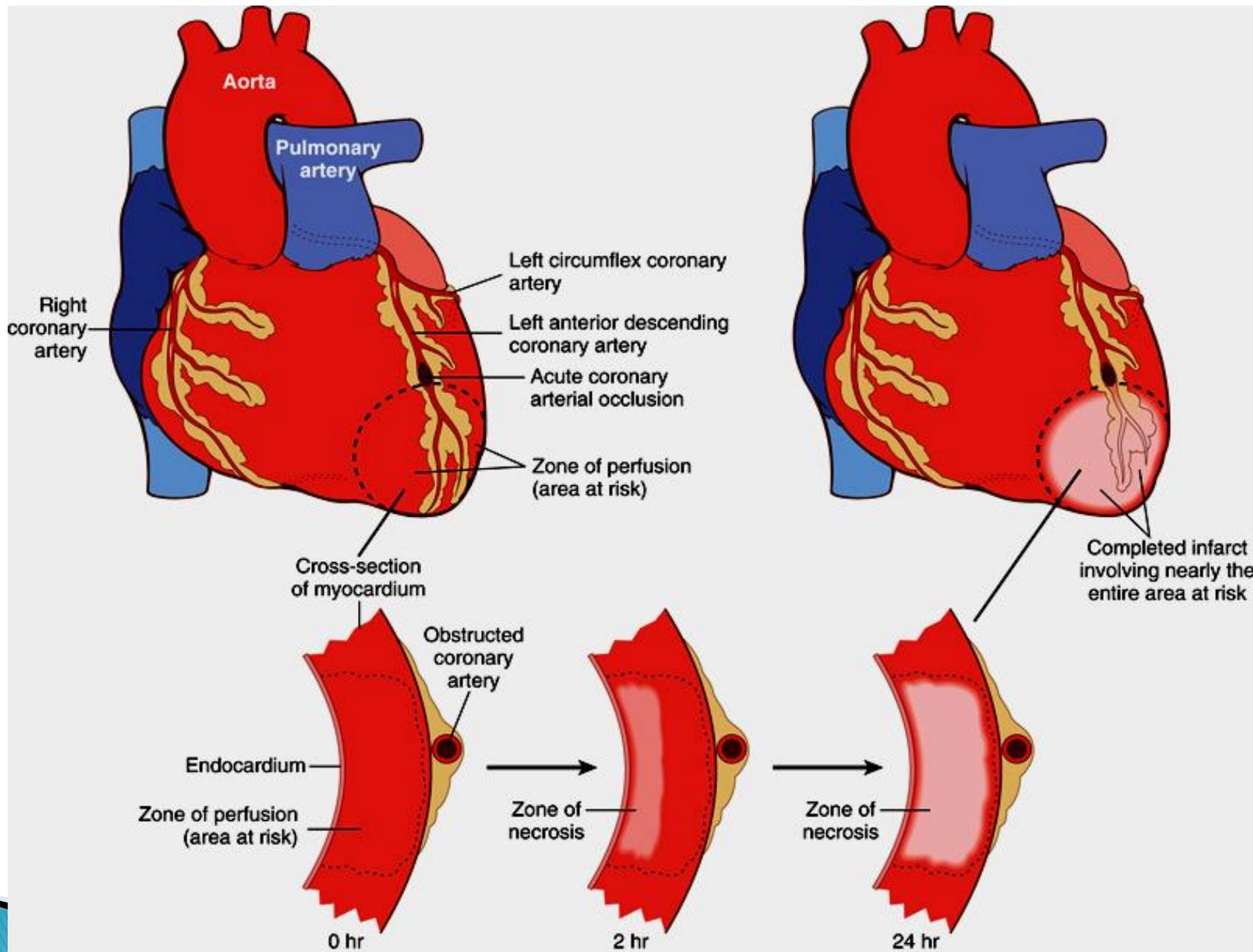
- ▶ **Silent infarcts:**
 - ▶ 10% -15% are asymptomatic
 - ▶ particularly in:
 - 1- DM (peripheral neuropathies)
 - 2- the elderly
- 

Myocardial Infarction

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

Acute occlusion of left anterior descending (LAD) artery is the cause of *50% of all MI*





Evaluation of MI

- ▶ *Clinical signs and symptoms*
- ▶ *Electrocardiographic(ECG) abnormalities*
- ▶ ***Laboratory evaluation:***

blood levels of certain cardiac intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes.

Cardiac enzymes in MI

1- Cardiac Troponins T and I (TnT, TnI): **the best markers for acute MI**

2- Creatine kinase (CK, and more specifically the myocardial-specific isoform, CK-MB): **the second best**

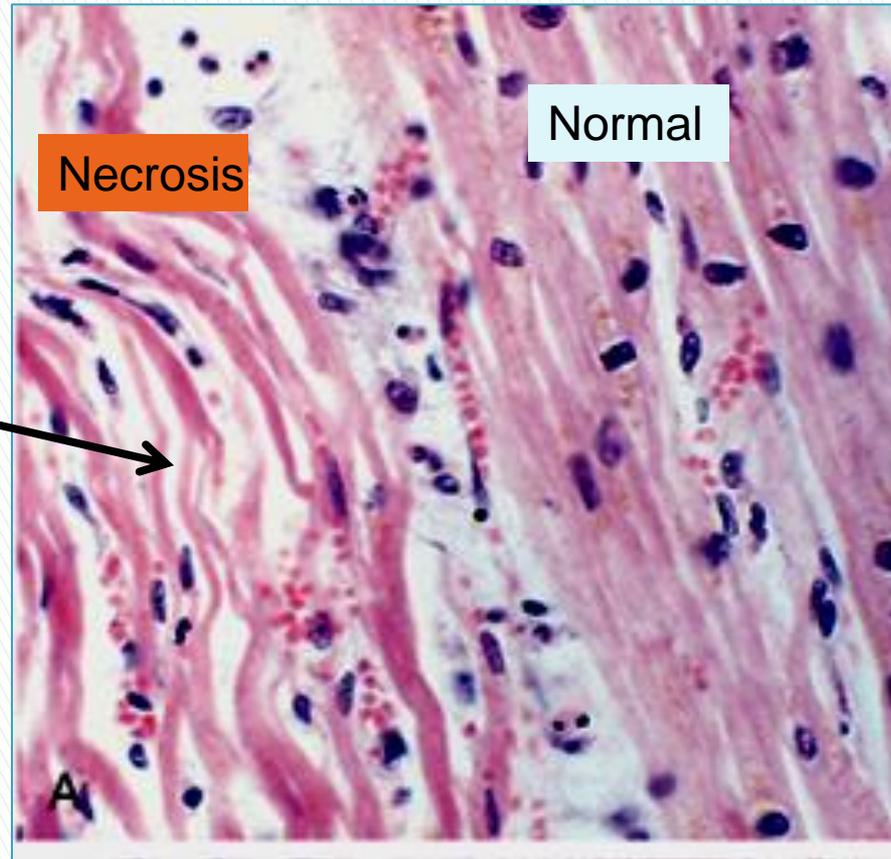
3- Lactate dehydrogenase

4- Myoglobin

Microscopic changes of MI & its repair

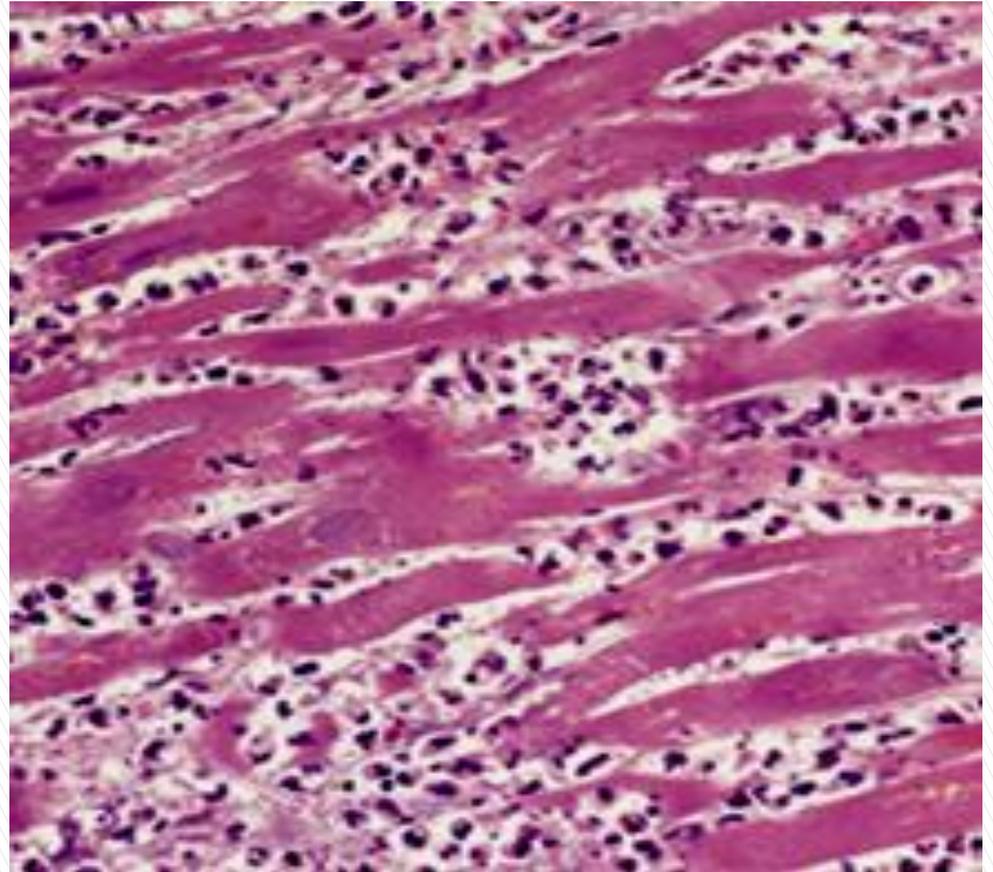
- (**<24 hr**) → coagulative **necrosis** and **wavy fibers**
- (**2- to 3-day**) **old - infarct** → **neutrophil** infiltrate
- (**7 to 10 days**) → phagocytic **macrophages** remove necrotic tissue
- **up to 14 days** → **Granulation tissue** (loose connective tissue and abundant capillaries)
- **several weeks** → Healed MI (collagenous **scar**)

(<24 hr):
coagulative
necrosis and **wavy**
fibers

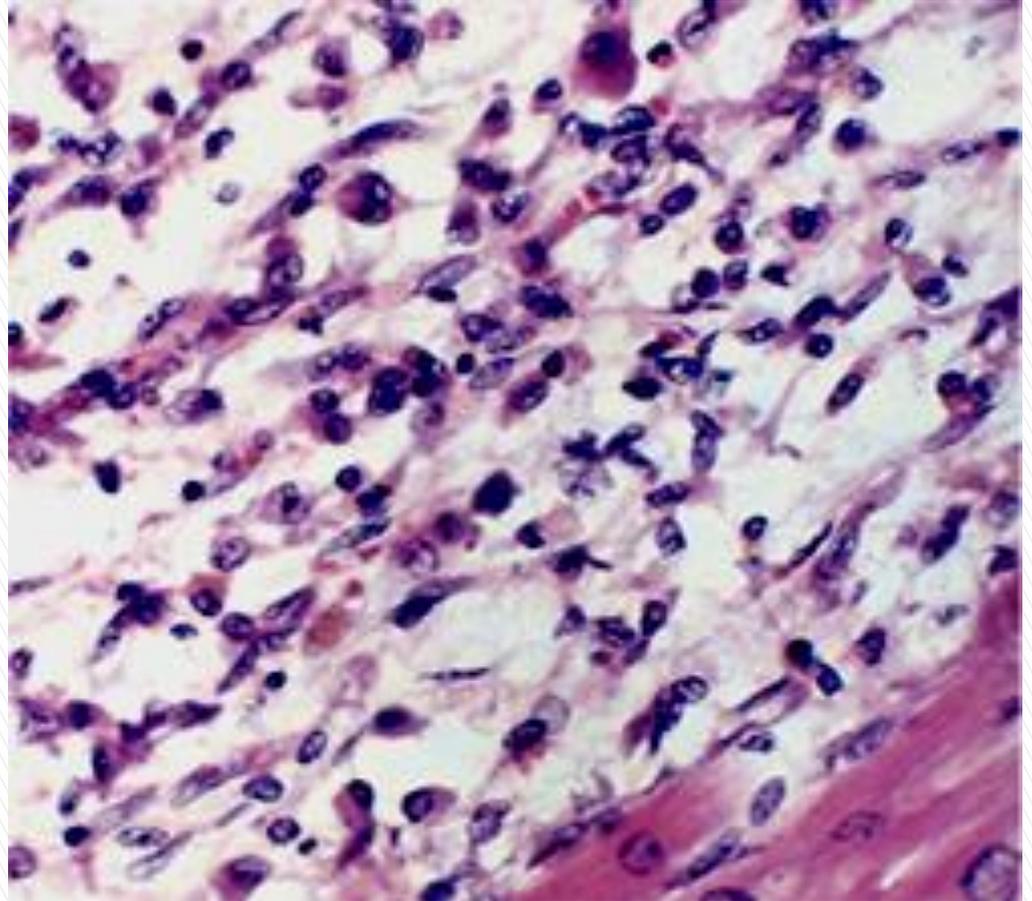


**2- 3 day old –
infarct:**

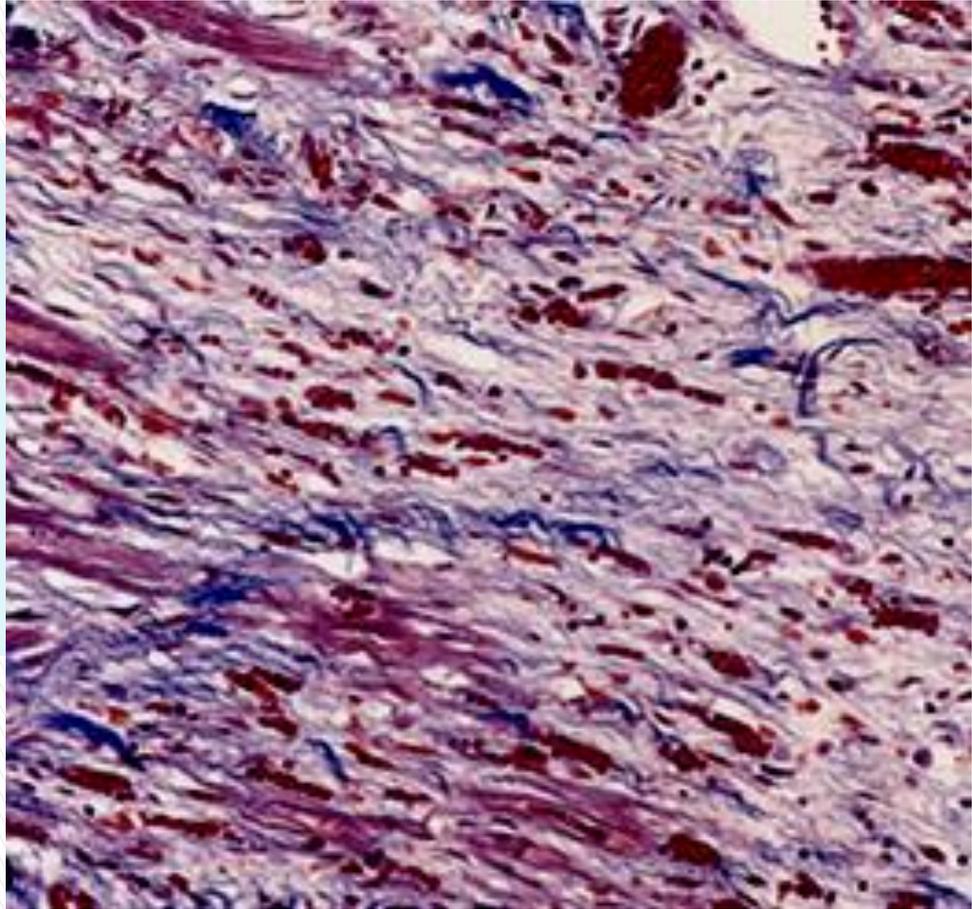
**Dense neutrophil
infiltrate**



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

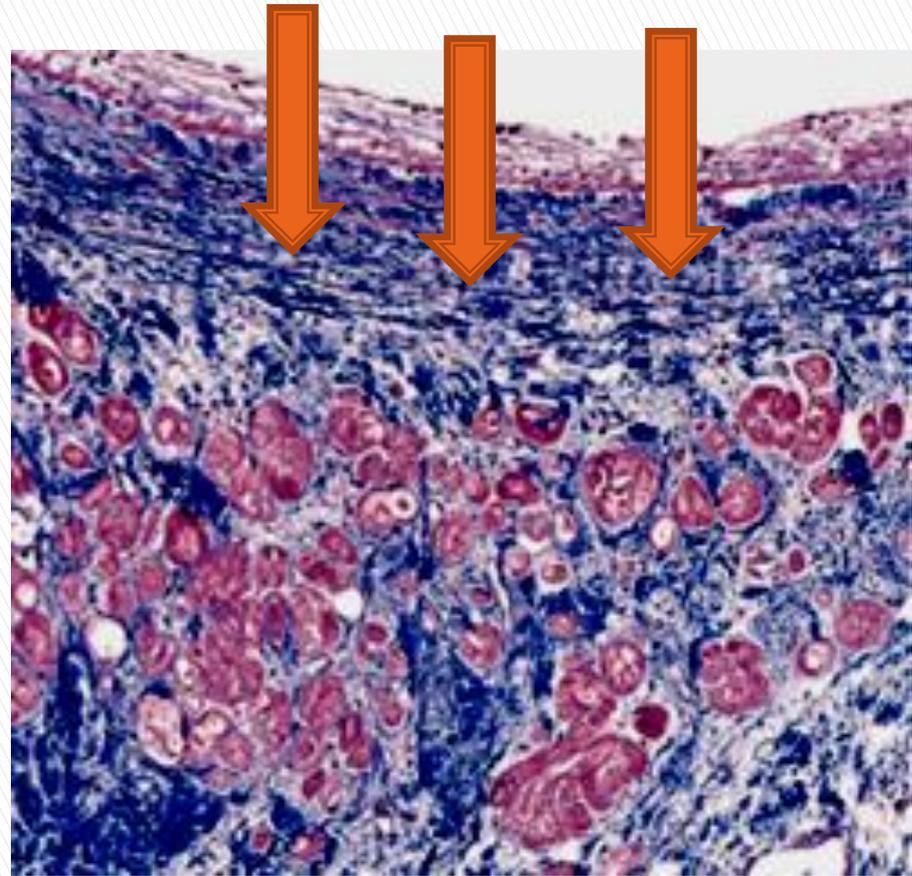


up to 14 days:
Granulation
tissue = loose
connective tissue (blue)
and abundant capillaries
(red)



several weeks:

Healed infarct with a
dense collagenous
scar (blue)



Consequences and Complications of MI

- **1- Death:**
- **50%:** before reaching hospital (<1 hour of symptom onset; due to arrhythmias)
- (*in-hospital*) *death rate* has declined (30% to 10%) thanks to extraordinary progress in medical care

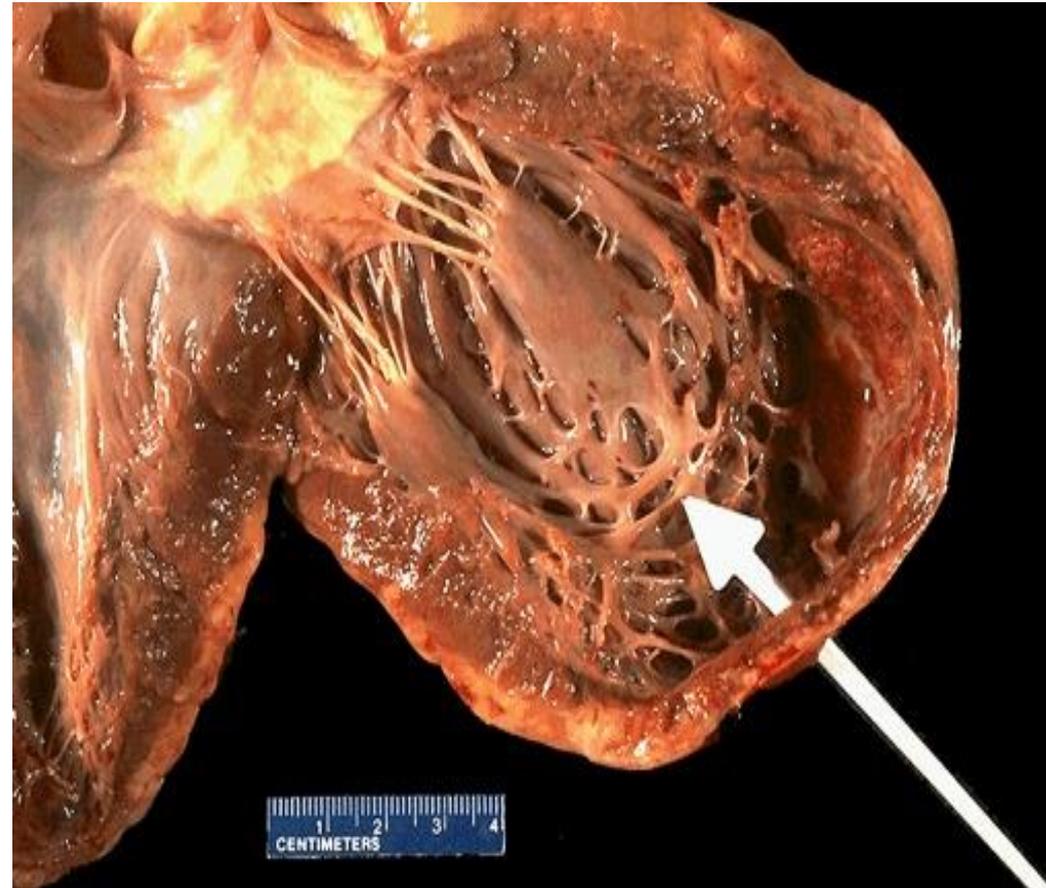
Consequences and Complications of MI

- ▶ ***Cardiogenic shock***
 - (10% to 15%)
 - In large infarcts (>40% of Left ventricle)
 - Mortality 70%; 2/3 of in-hospital deaths
- ▶ ***Myocardial rupture***
- ▶ ***Papillary Muscle Rupture***
- ▶ ***Pericarditis***
- ▶ ***Infarct expansion***
- ▶ ***Mural thrombus***
- ▶ ***Ventricular aneurysm***
- ▶ ***Progressive late heart failure***

Myocardial Rupture

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

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

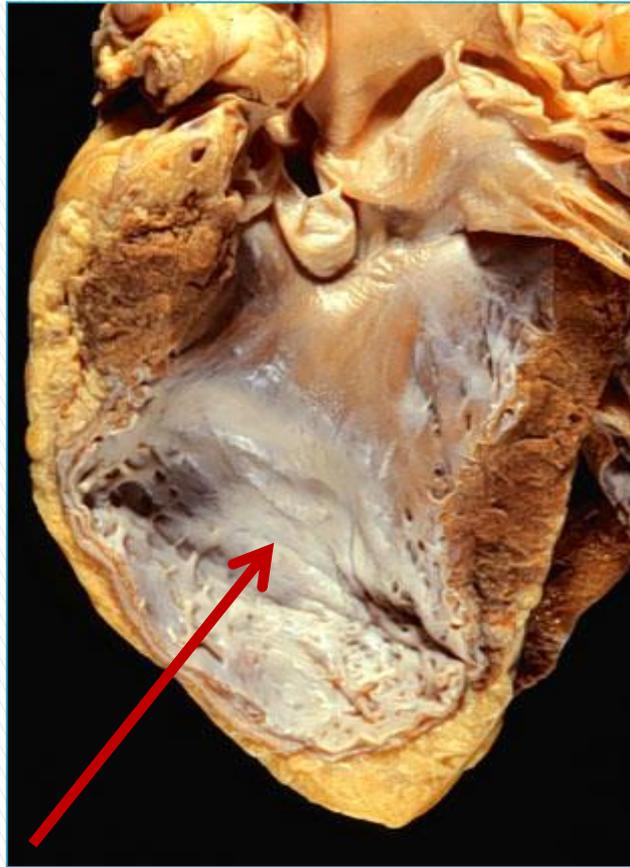


Papillary Muscle Rupture



Leads to post-infarct
mitral regurgitation

Ventricular aneurysm



Ventricular aneurysm

- A late complication
- result from a large transmural infarct with 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. LV function; severity of atherosclerosis in remaining viable myocardium;
- Mortality rate 1st year 30%
- annual mortality rate 3%

Chronic Ischemic Heart Disease

- ▶ **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 without symptoms or < 24 hours of symptom onset**
- ▶ **CAD (atherosclerosis) is the most common underlying cause**
- ▶ **Lethal arrhythmias (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 or hypertrophic cardiomyopathy
- ▶ Pulmonary hypertension
- ▶ Hereditary or acquired abnormalities of the cardiac conduction system
- ▶ Isolated myocardial hypertrophy
- ▶ unknown causes....

