# ISCHEMIC HEART DISEASE-1

myocardial ischemia occurs when: ↓ Blood supply.

If the Blood supply is decreased and Oxygen demand is increased we have imbalance

Examples: -Atherosclerosis -Coronary Vasospasm -Hypovolemia -Shock

↑O2 demand.

Factors that increase oxygen demand in the heart (increase workload on cardiac muscle) :

Examples: -exertion -hypertension -stress -tachycardia

- a group of related syndromes resulting from myocardial ischemial (an imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand)
- IHD ≈ coronary artery disease (CAD)

## Ischemia can result from:

1. reduction in coronary blood flow atherosclerosis (90 % of cases )
2. increased demand (e.g., tachycardia or hypertension)
3. diminished oxygen-carrying capacity (e.g., anemia, CO poisoning)

## There are four basic clinical syndromes of IHD

### 1 Angina pectoris

ischemia causes pain but is insufficient to lead to death of myocardium

### 2 Acute myocardial infarction (MI)

the severity or duration of ischemia is enough to cause cardiac muscle death

### 3 Chronic IHD

progressive cardiac decompensation (heart failure) following MI

### 4 Sudden cardiac death (SCD)

can result from a lethal arrhythmia following myocardial ischemia.

Angina pain. A crushing or squeezing substernal pain

## Angina pectoris vs MI

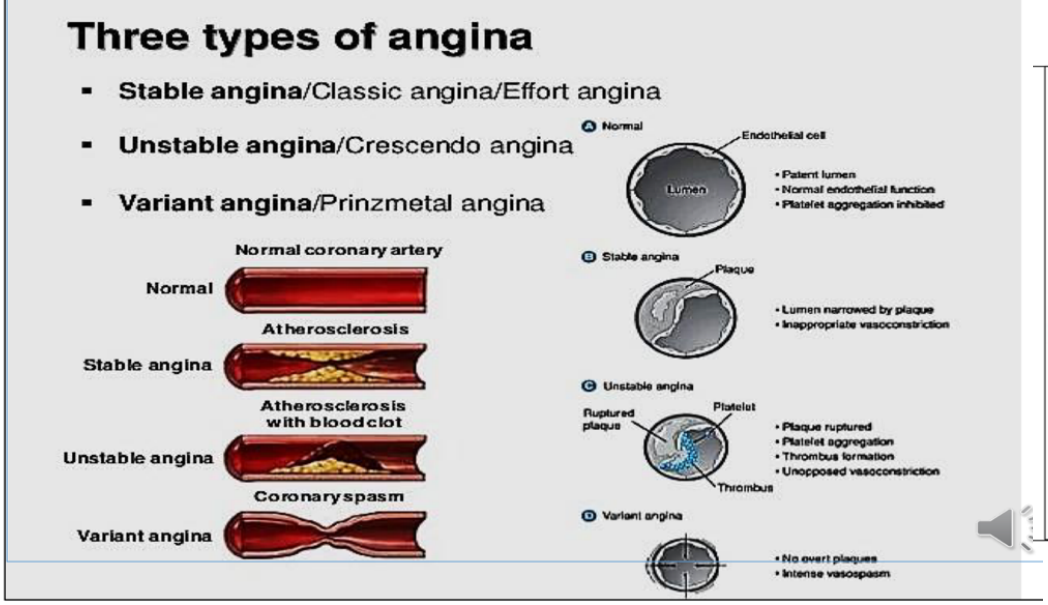
angina causes intermittent chest pain caused by transient reversible myocardial ischemia (ischemia causes pain but is insufficient to lead to death of myocardium)

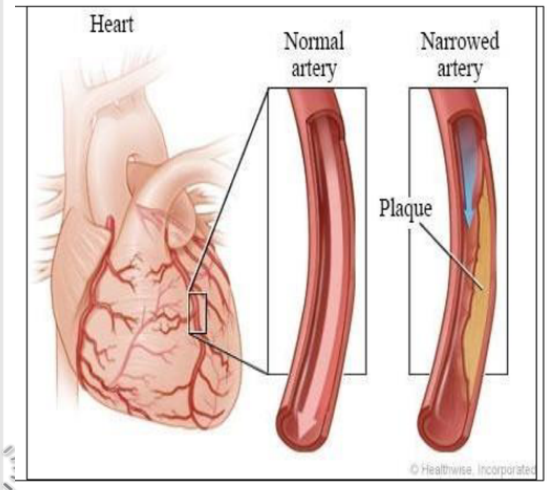
- angina pectoris: pain usually < 20 minutes and relieved by rest or nitroglycerin → pharma mid.
- MI: pain lasts usually > 20 minutes to several hours and is not relieved by nitroglycerin or rest

Coronary artery disease: plaque builds up in the artery.

Angina: it is harder for blood to get through the artery.

Heart attack: plaque cracks and a blood clot blocks the artery.




## Pathogenesis of stable angina: critical coronary

-episodic pain only with increased demand -forms of ↑ myocardial oxygendemand (e.g. exertion;tachycardia; hypertension;fever; anxiety;fear)
-associated with critical atherosclerotic narrowing
-relieved by rest (reducing demand) or by drugs (e.g. Nitroglycerin )

## Pathogenesis of Prinzmetal angina: severe coronary vasospasm

- occur at rest or sleep
- Vesselswithout atherosclerosis can be affected
- Etiology not clear
- Treatment: vasodilators (nitroglycerin or calcium channel blockers)

## Pathogenesis of unstable angina

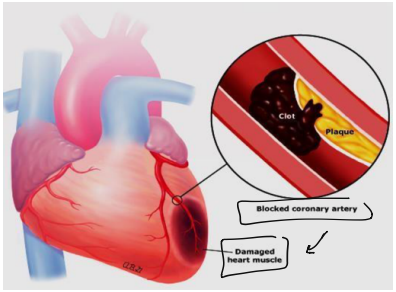
critical stenosis with superimposed Acute Plaque Change:
1-plaque disruption 2- partial thrombosis (non-occlusive)
3- distal embolization 4-vasospasm

- (crescendo angina)

- increasing frequency of pain, precipitated by less exertion.
- more intense and longer lasting than stable angina - Causes: plaque disruption; superimposed partial
thrombosis; distal embolization; vasospasm.
- Usually precedes more serious, potentially irreversible ischemia, thus it is called: pre-infarction angina

# ISCHEMIC HEART DISEASE-2

## Acute Myocardial Infarction (MI)




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

## Clinical Features of acute MI

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)

## Silent infarcts:

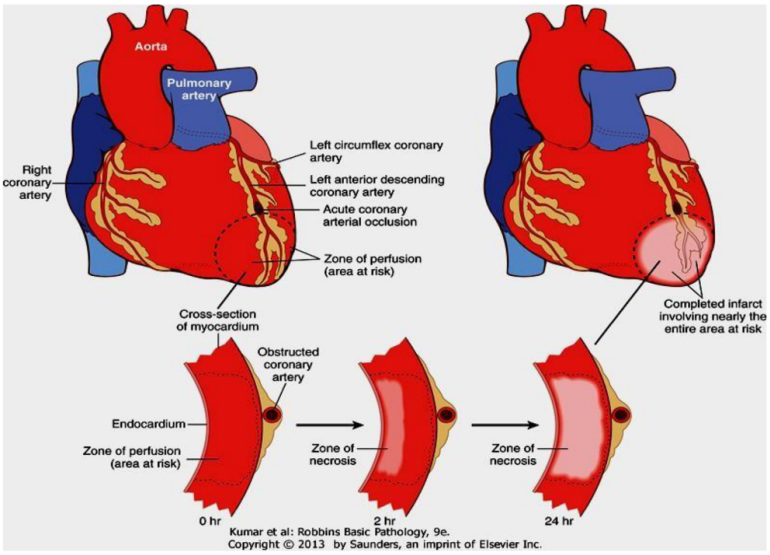
- A variable percentage of MIs are asymptomatic
- Confirmed only on ECG and lab workup.
- particularly in: 1DM (peripheral neuropathies) 2 the elderly

## MI- Causes:

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

## Evaluation of MI

- 1.Clinical signs and symptoms
- 2.Electrocardiographic (ECG) abnormalities -> Confirmatory of having acute MI
- 3.Laboratory evaluation: blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes



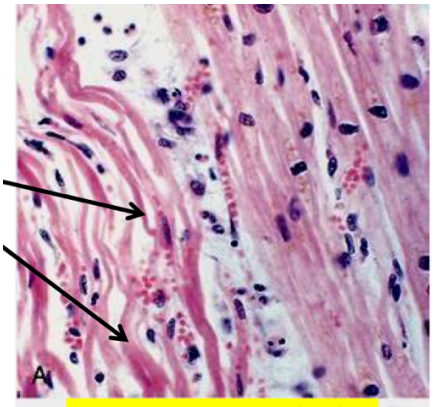

## Cardiac enzymes in MI

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.

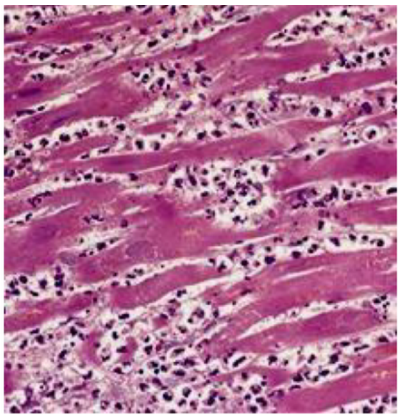
## Microscopic features of myocardial infarction and its repair.

**<24 hr:** coagulative necrosis and wavy fibers Necrotic cells are separated by edema fluid



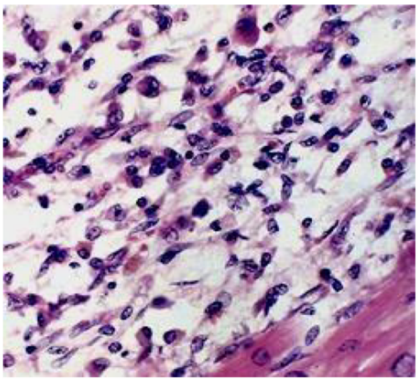
Stain: Hematoxylin & Eosin (H&E)

**2 - 3 days:** Dense neutrophil Infiltrate



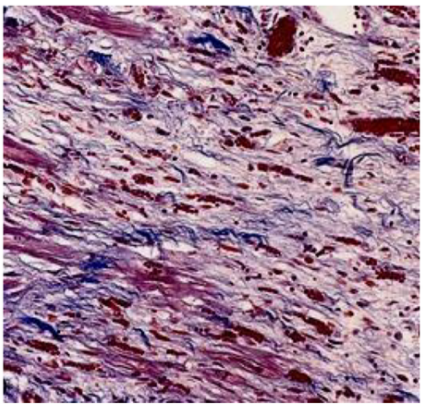
Stain: Hematoxylin & Eosin (H&E)

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



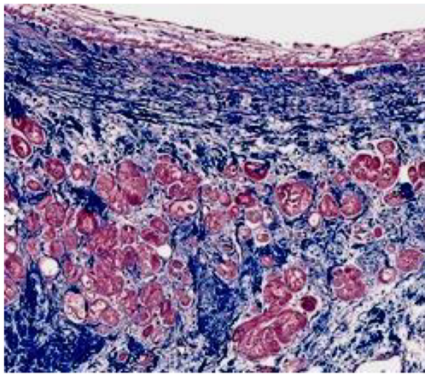
Stain: Hematoxylin & Eosin (H&E)

**up to 14 days:** Granulation tissue [loose connective tissue(blue)and abundant capillaries (red)] -weak tissue and doesn't have



Stain: Masson Trichrome (MT)

several weeks: Healed infarct consistingofa dense collagenous scar (blue)
(red) tissues is the remaining viable myocardial cells.



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)

■ 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)ruptureofthe ventricularseptum:

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 (associated with ischemia)⟶ 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

## • Complicationsof ventricular aneurysms include:

1-mural thrombus 2-arrhythmias 3-heart failure

## Long-term prognosis after MI

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

- 1st 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...