PATHOLOGY

بسم الله الرحمن الرحيم

FINAL | Lecture 7

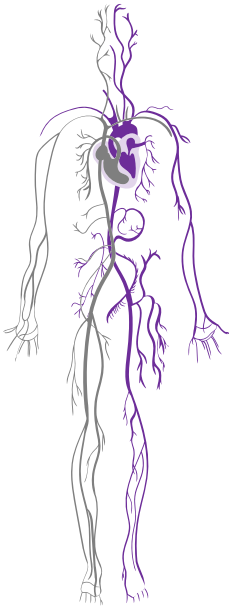
Ischemic Heart Disease

وَلَقَدْ خَلَقْنَا الْإِنسَانَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِ نَفْسُهُ ۖ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ الْوَرِيدِ

اللهم إنّا نعوذ بك من شرور أنفسنا ومن سيئات أعمالنا

Written by: Hana 'Abu-sbeih

Reviewed by: Layan Fawarseh



وَلِلَّهِ الْأَسْمَاءُ الْحُسْنَى فَادْعُوهُ بِهَا

المعنى: المبرأ من النقائص والشريك، وكل ما لا يليق بالإلهية، الذي تُسبّحه وتقدّسه الخلائق وتنزهه عن كل سوء، لكمال أسمائه وصفاته وجمالها.

الورود: لم يرد في القرآن الكريم، وورد في السنة.

الشاهد: في أذكار الركوع والسجود، (سبوح قدوس رب الملائكة والروح).

(أخرجه مسلم).

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اضغط هنا لشرحٍ أكثر تفصيلًا

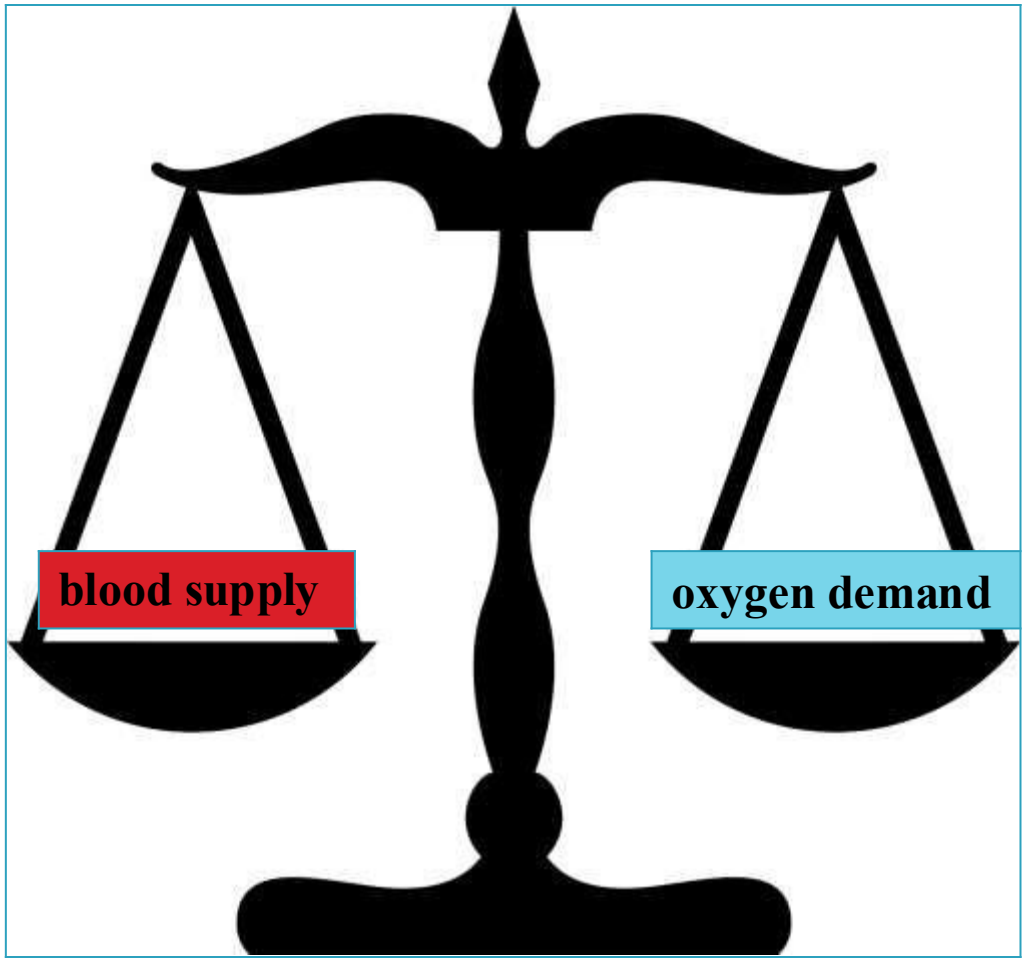
بسم الله الرحمن الرحيم

ISCHEMIC HEART DISEASE

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

Heart disease is the leading cause of morbidity and mortality worldwide

Normally …

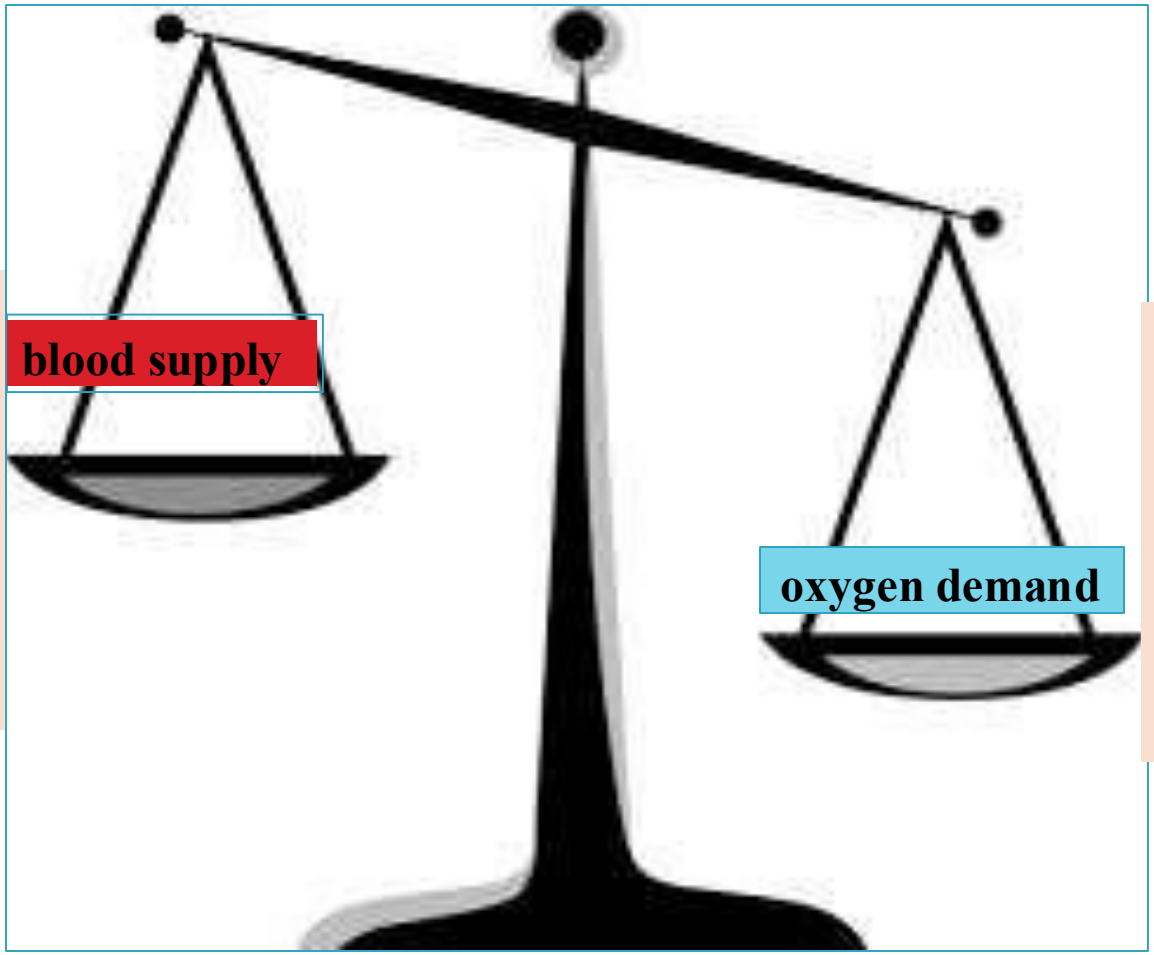


- Cardiac muscle (as any other cell) needs a **balance** between oxygen demand and blood supply.
- The **coronary arteries** provide the heart's blood supply.
- Because the heart is always working, its **oxygen demand is high**.
- If blood supply does not meet oxygen demand → **ischemia** occurs.
- Ischemia leads to **ischemic heart disease**.

myocardial *ischemia occurs when:*

things may lead to a reduction in the blood supply:

Examples:
- Atherosclerosis
- Coronary Vasospasm
- Hypovolemia
- Shock
- Thrombosis
- stenosis



Things may lead to increase the oxygen demand:

Examples (increase force or rate of contraction):
-exertion
-hypertension
-stress
- Increase emotional and physical activity
-tachycardia

Any imbalance between blood supply and oxygen demand can lead to ischemic heart disease

سبحان الله العظيم

ISCHEMIC HEART DISEASE (IHD)

- a group of related syndromes resulting from myocardial *ischemia* (**an imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand)**

- IHD ≈ coronary artery disease (CAD) (another name)

Ischemia can result from:

1 reduction in coronary blood flow, mainly due atherosclerosis (90 % of cases)

2 increased demand for oxygen (e.g., tachycardia or hypertension)

3 diminished oxygen-carrying capacity (least common cause) (e.g., severe anemia, CO poisoning)

CO will compete against oxygen for heme molecule in hemoglobin,so the blood will deliver CO instead of delivering oxygen to the cardiac muscles.

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 (necrosis).

استغفر الله العظيم
وأتوب إليه

3-Chronic IHD

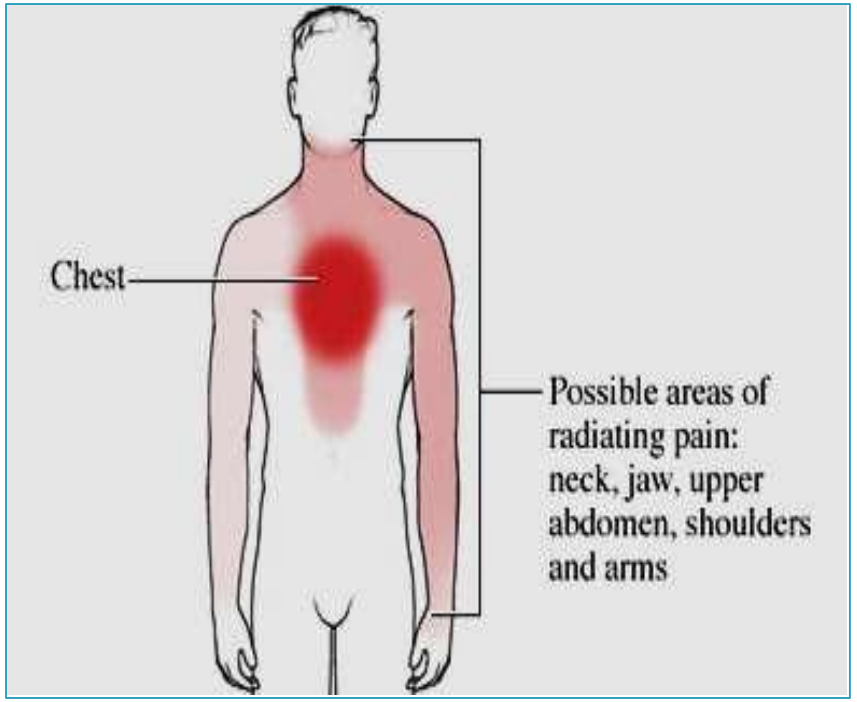
progressive cardiac decompensation (heart failure) following MI

4-Sudden cardiac death (SCD) (The worst)

can result from a lethal arrhythmia following myocardial ischemia.

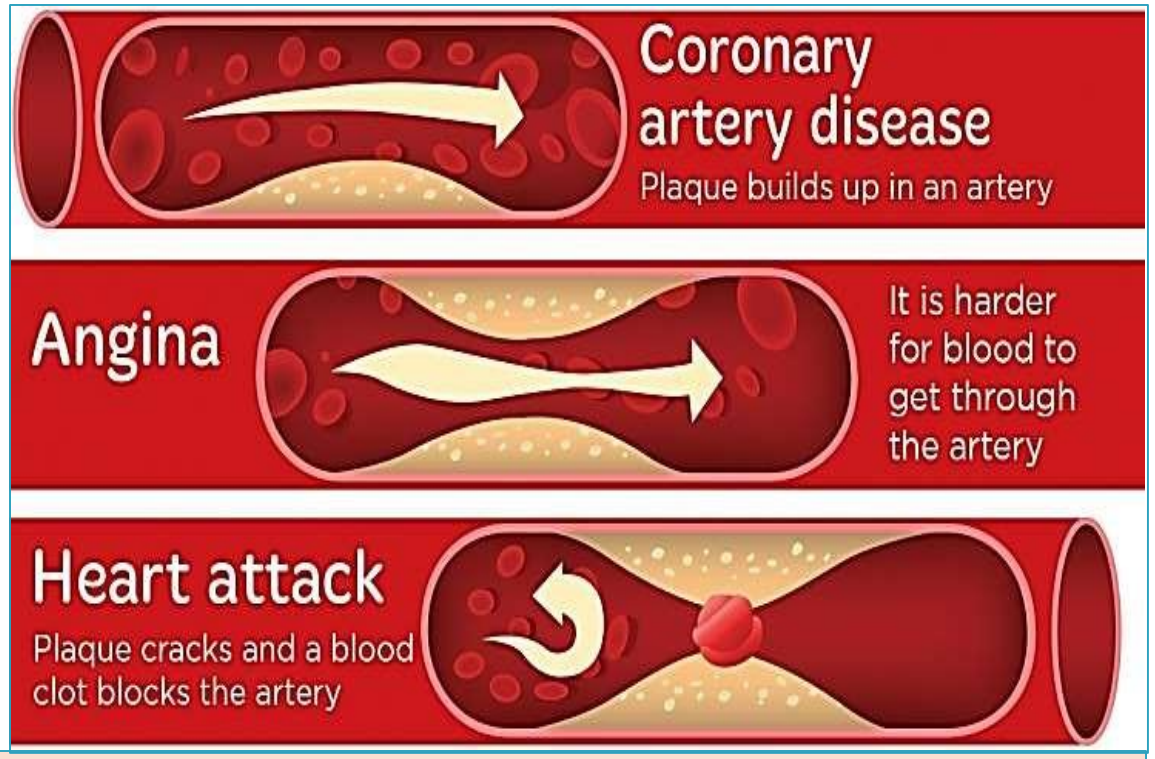
Angina pectoris الذبحة الصدرية

Clinically: Angina pain (A crushing or squeezing substernal so severe pain it may radiate also to the left side of the arm, the jaw, the neck or the upper abdomen.)



- Lose of balance btw. O2 demand and blood supply which leads to cardiac ischemia, but this cardiac ischemia happen for short duration (low intensity and severity) that is not enough to cause the necrosis of the cardiac muscle cells.

Angina pectoris vs MI



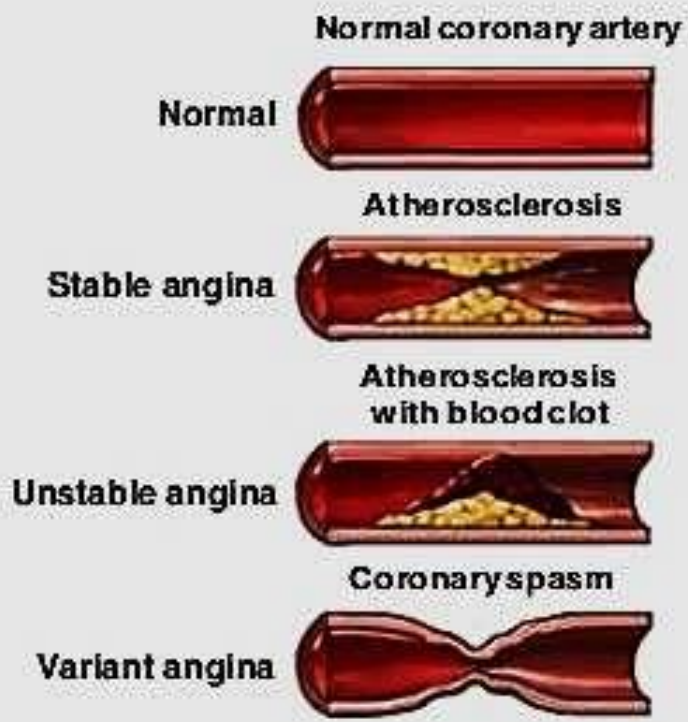
angina causes intermittent chest pain caused by transient reversible myocardial ischemia (**ischemia causes pain but is insufficient to lead to death of myocardium)**

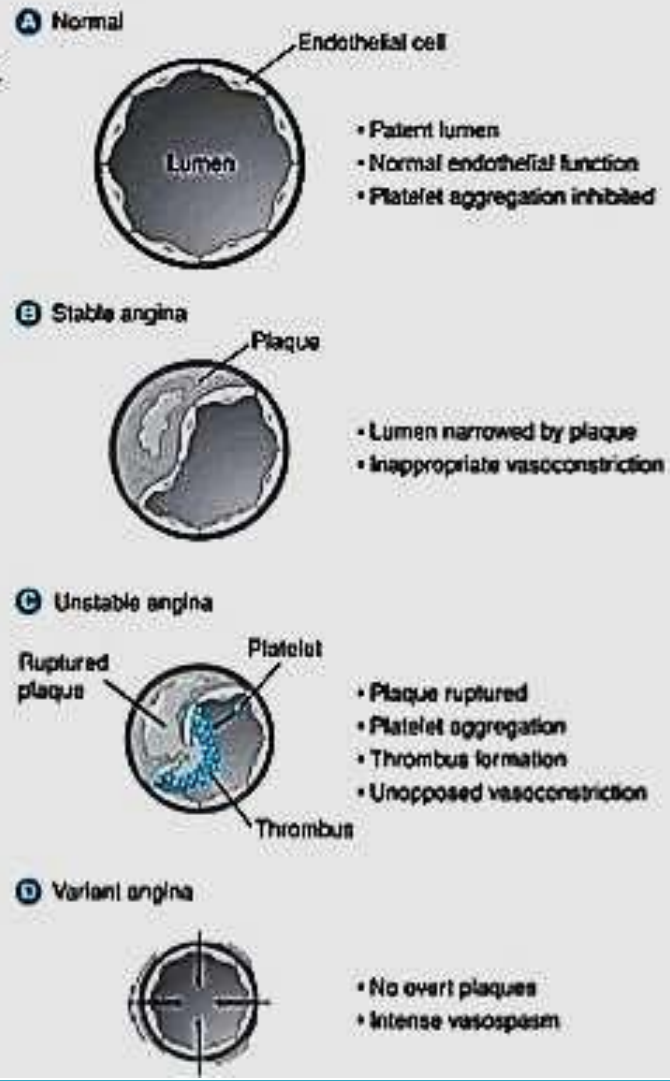
- **angina** pectoris: pain < 20 minutes and relieved by rest or nitroglycerin
- **MI**: pain lasts > 20 minutes to several hours and is not relieved by nitroglycerin or rest.

Three types of angina

according to pathogenesis (underlying mechanism)

- **Stable angina**/Classic angina/Effort angina
- **Unstable angina**/Crescendo angina تصاعدية
- **Variant angina**/Prinzmetal angina





Pathogenesis of stable angina: critical coronary stenosis

(Most frequent type of angina)

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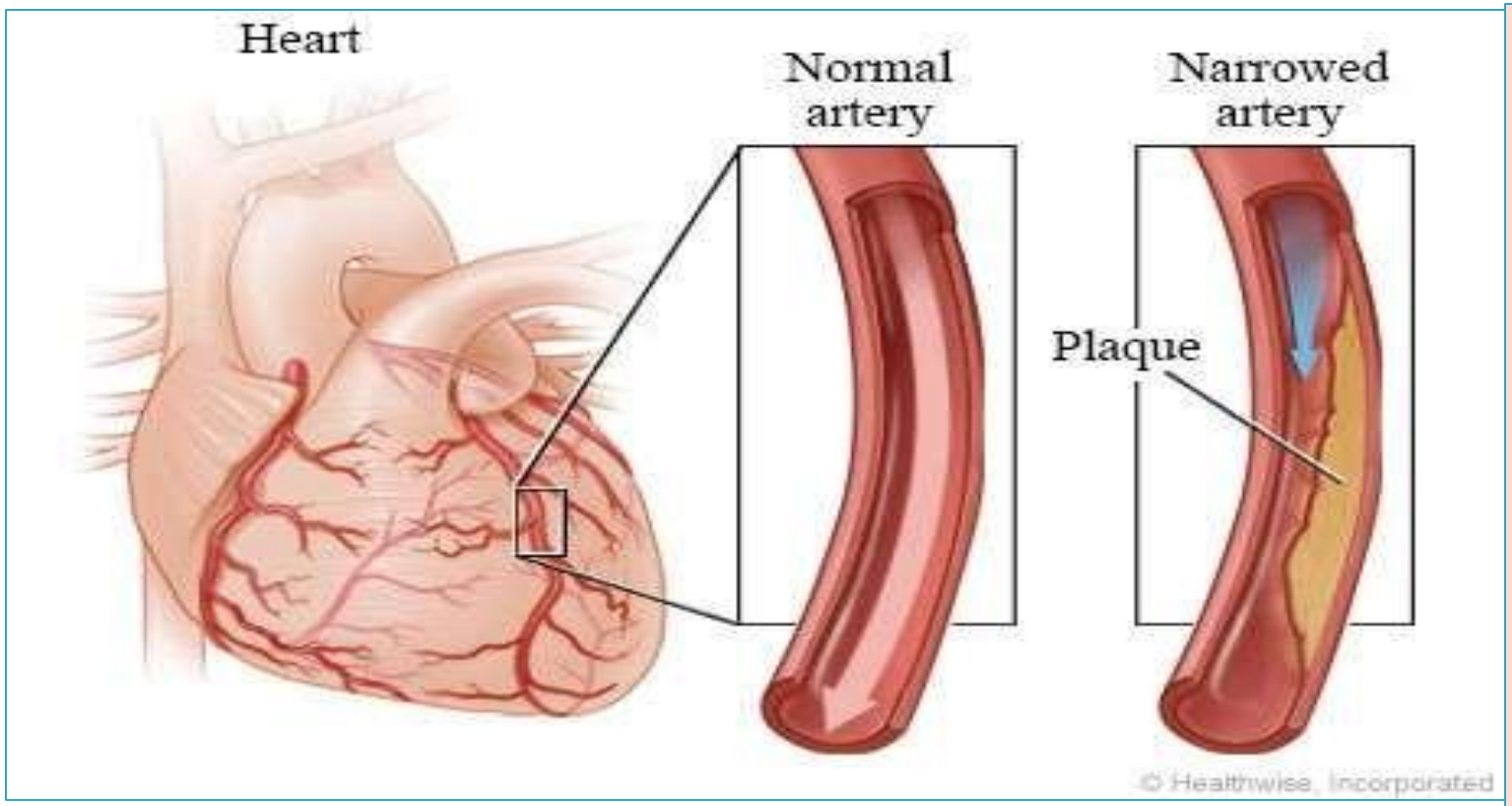
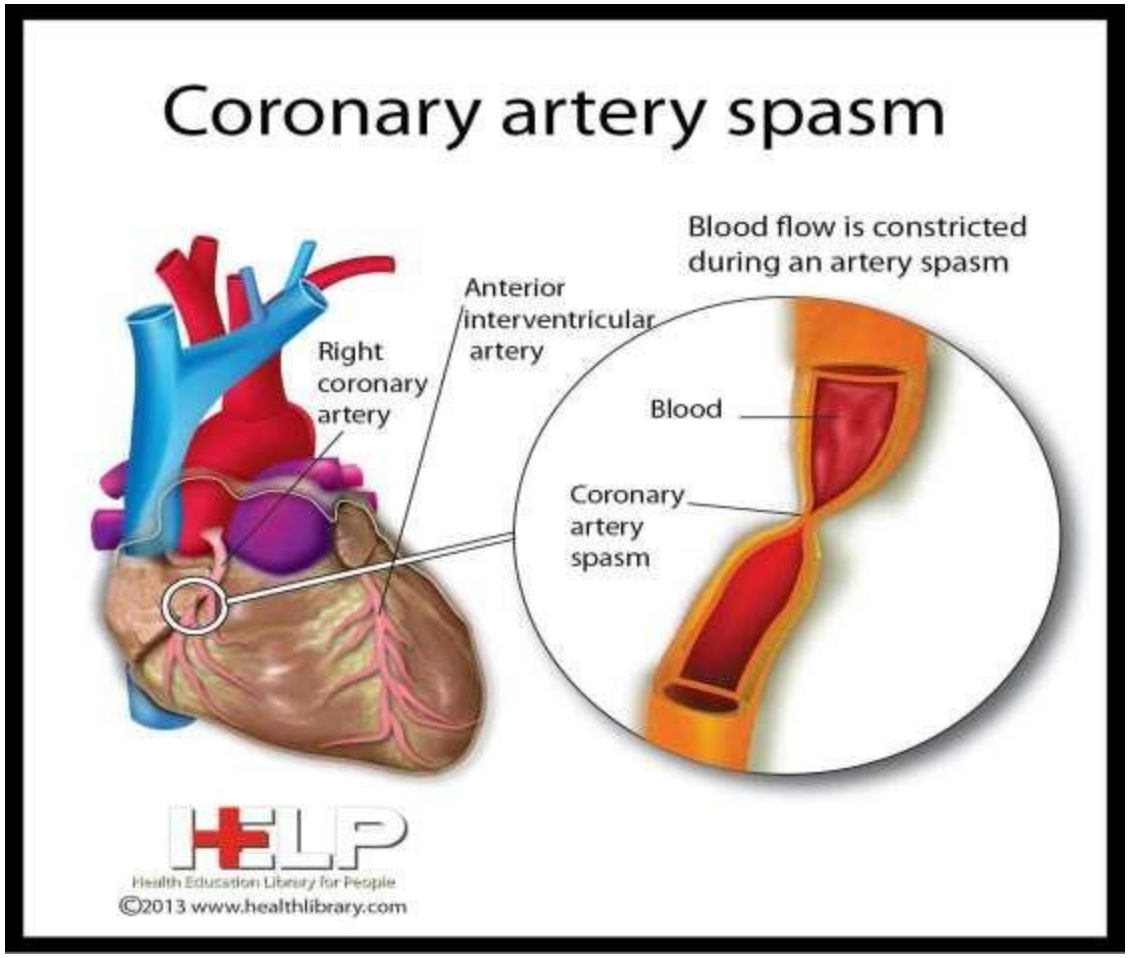


-episodic pain only <u>with increased demand</u>
-forms of ↑ myocardial oxygen demand (e.g. exertion; tachycardia; hypertension; fever; anxiety; fear)
-associated with critical atherosclerotic narrowing
-relieved by rest (reducing demand) or by drugs (e.g; sublingual nitroglycerin - vasodilators)

Critical stenosis -as a complication of atherosclerosis- means permanent narrowing in the coronary artery that has occluded a large percentage of the original lumen; for example, occlusion of >75%.
- pain occur with exertion because of increased oxygen demand.

Pathogenesis of Prinzmetal angina: severe coronary vasospasm



- **occur at rest or sleep** as ischemic chest pain (not related to exertion).
- **Vessels without atherosclerosis can be affected**
- **Etiology not clear** (Rare)
- **Treatment: vasodilators (nitroglycerin or calcium channel blockers)**

Pathogenesis of unstable angina

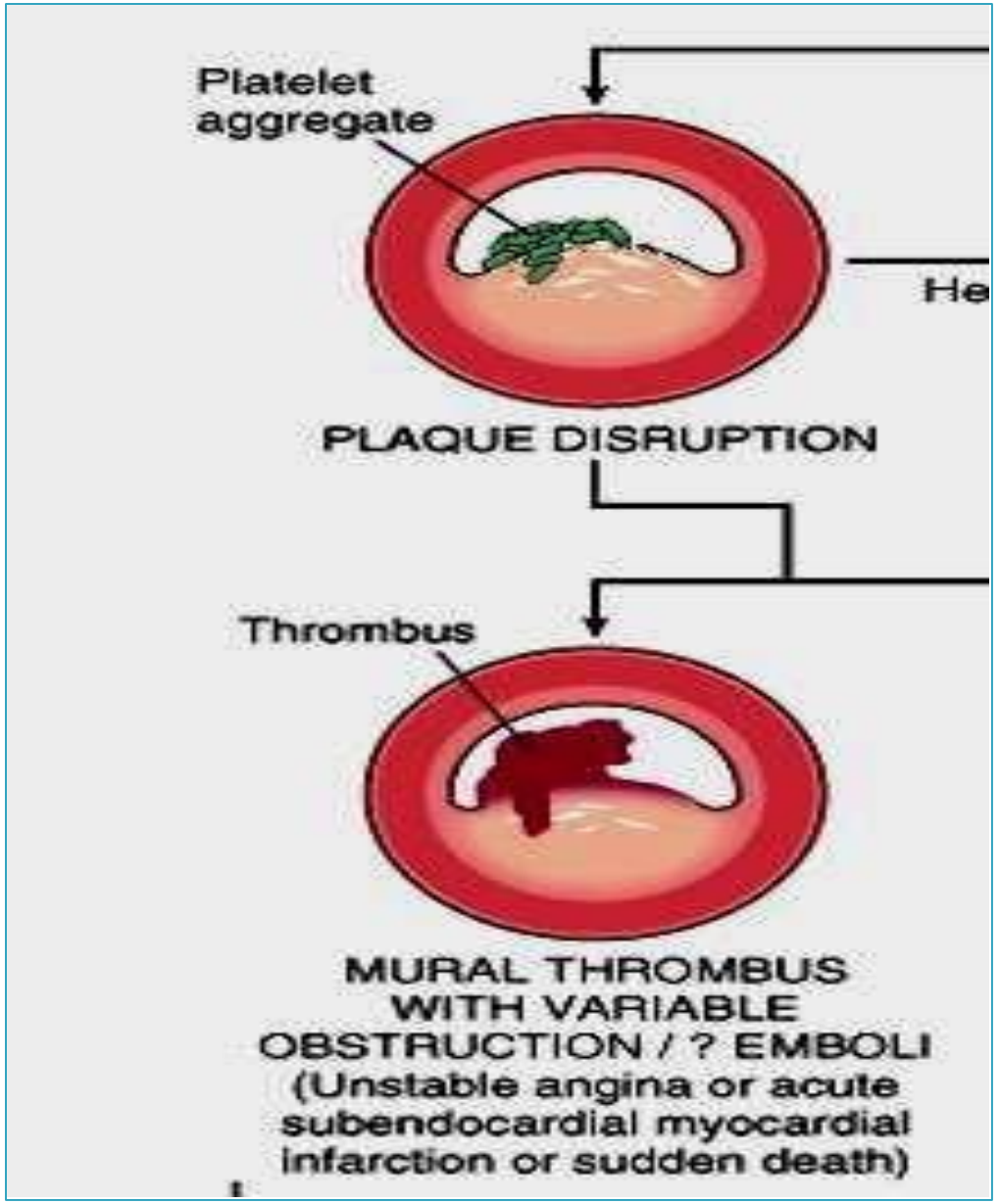
critical stenosis with superimposed Acute Plaque Change:

1-plaque disruption

2 partial thrombosis (non-occlusive)

3 distal embolization

4-vasospasm



Unstable angina (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*

It is considered a transitional stage, meaning we are just one step away from Myocardial Infarction (MI). It lies between Stable Angina and MI.

The patient already has atherosclerosis with critical stenosis. But something acute occurs on top of this chronic problem, making it worse and more dangerous.

What can this acute change be?

1. Thrombus formation overlying the critical stenosis → sudden reduction in blood supply → Unstable Angina.
2. Rupture or disruption of the atherosclerotic plaque → exposes the inner core → triggers platelet aggregation or thrombosis.
3. Superimposed vasospasm over the already narrowed artery.

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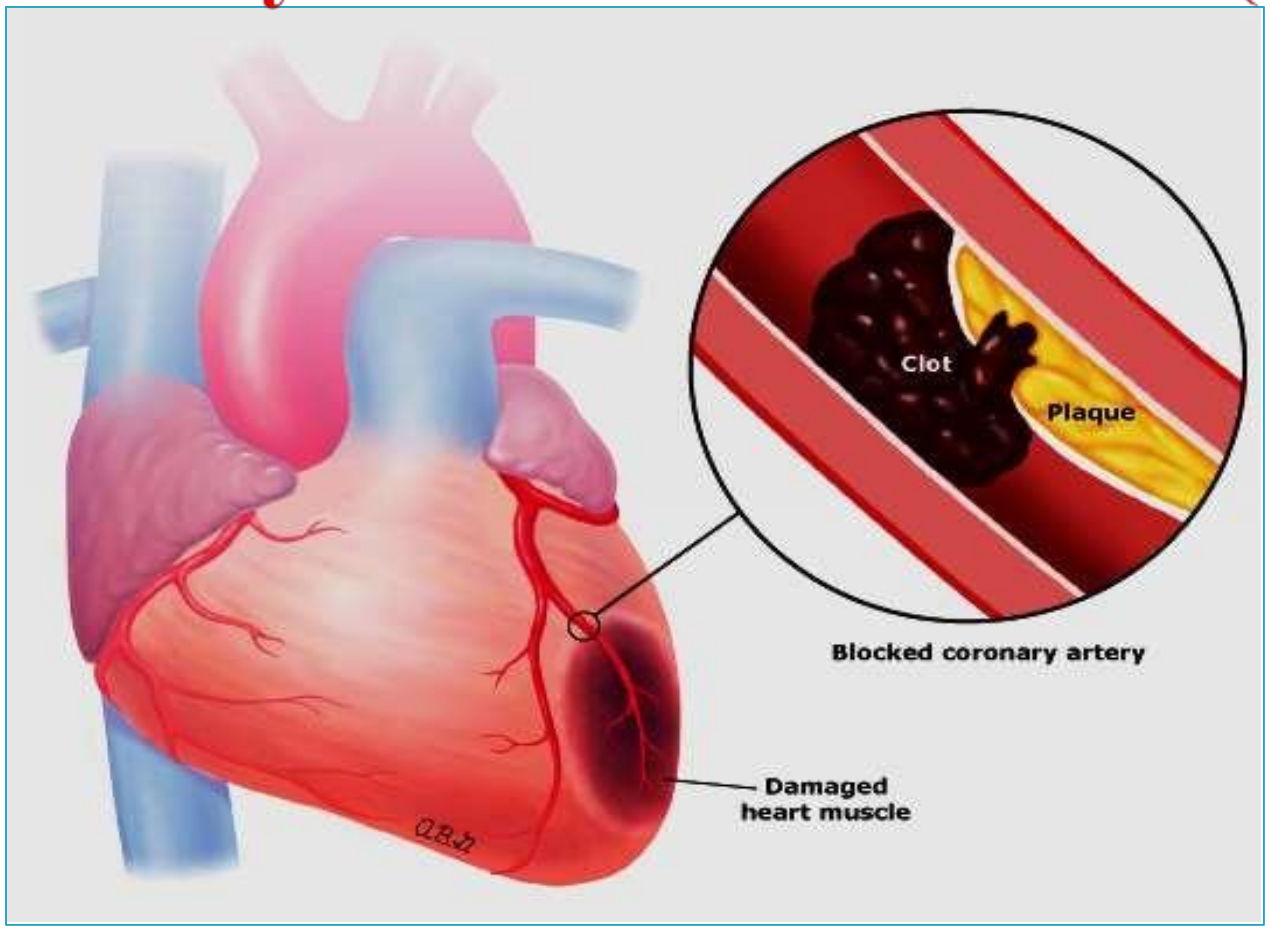
بالله العلي العظيم

Acute Myocardial Infarction

Acute Myocardial Infarction (MI)



- MI = *heart attack* (احتشاء عضلة القلب = جلطة)
- *Necrosis of heart muscle due to ischemia.*
- A significant cause of death worldwide.

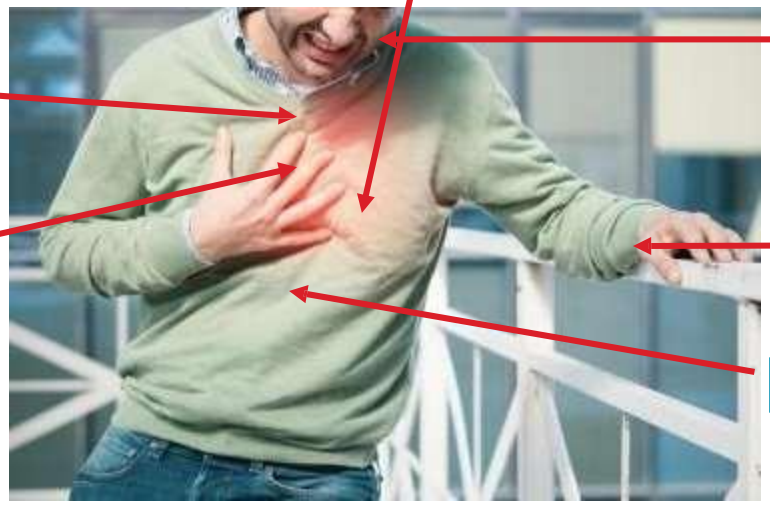
- The pain is similar to that of angina because the mechanism is the same.

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)



Dizziness; sweating

rapid and weak pulse

nausea (in posterior MI)

Sometimes: No typical symptoms (silent infarcts)

- Some patients do not show this classic picture and cannot be diagnosed clearly based on symptoms alone. This is called silent MI.

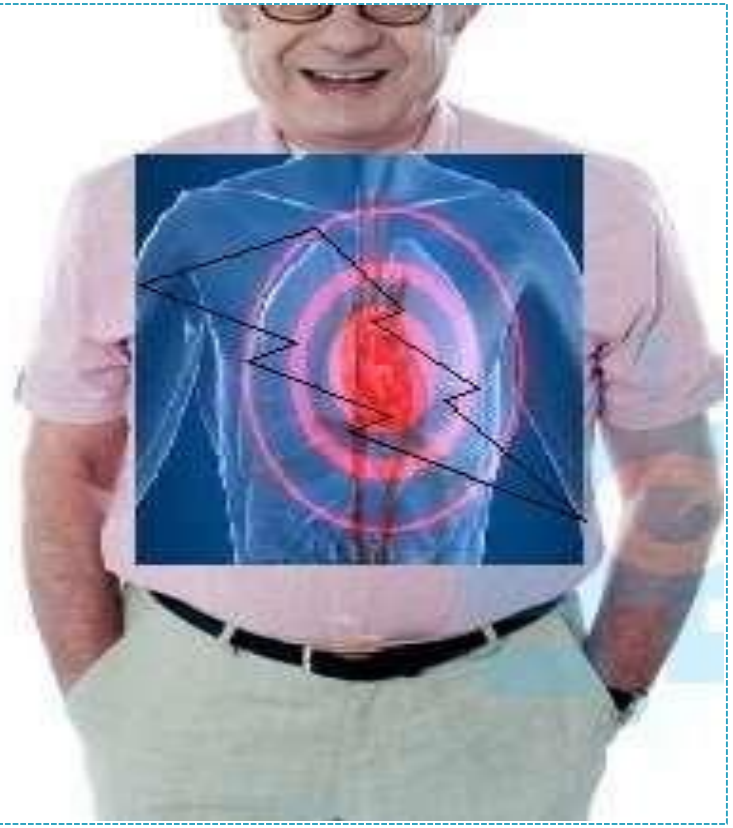
سبحان الله وبحمده عدد خلقه ورضا
نفسه وزنة عرشه ومداد كلماته

Silent infarcts:

A variable percentage of MIs are asymptomatic Confirmed only on ECG and lab workup.

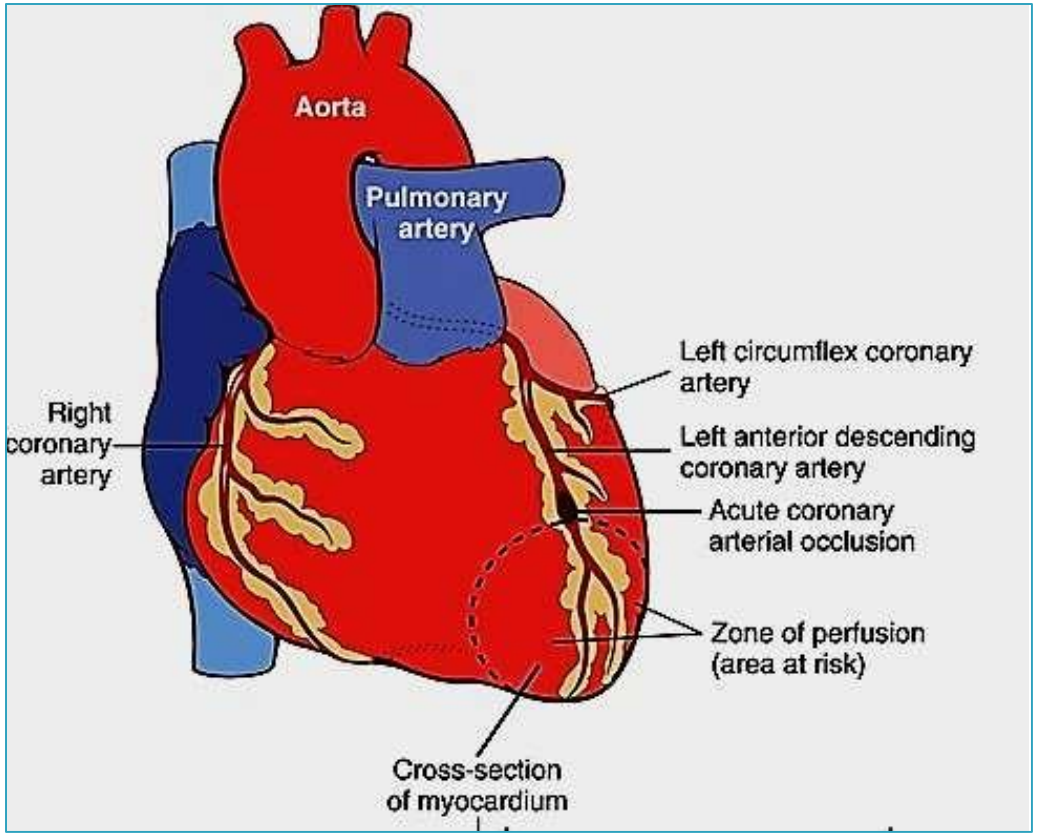
particularly in:

1. DM (peripheral neuropathies)
2. the elderly
3. Unconscious patients (e.g., in the ICU)



MI- Causes:

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



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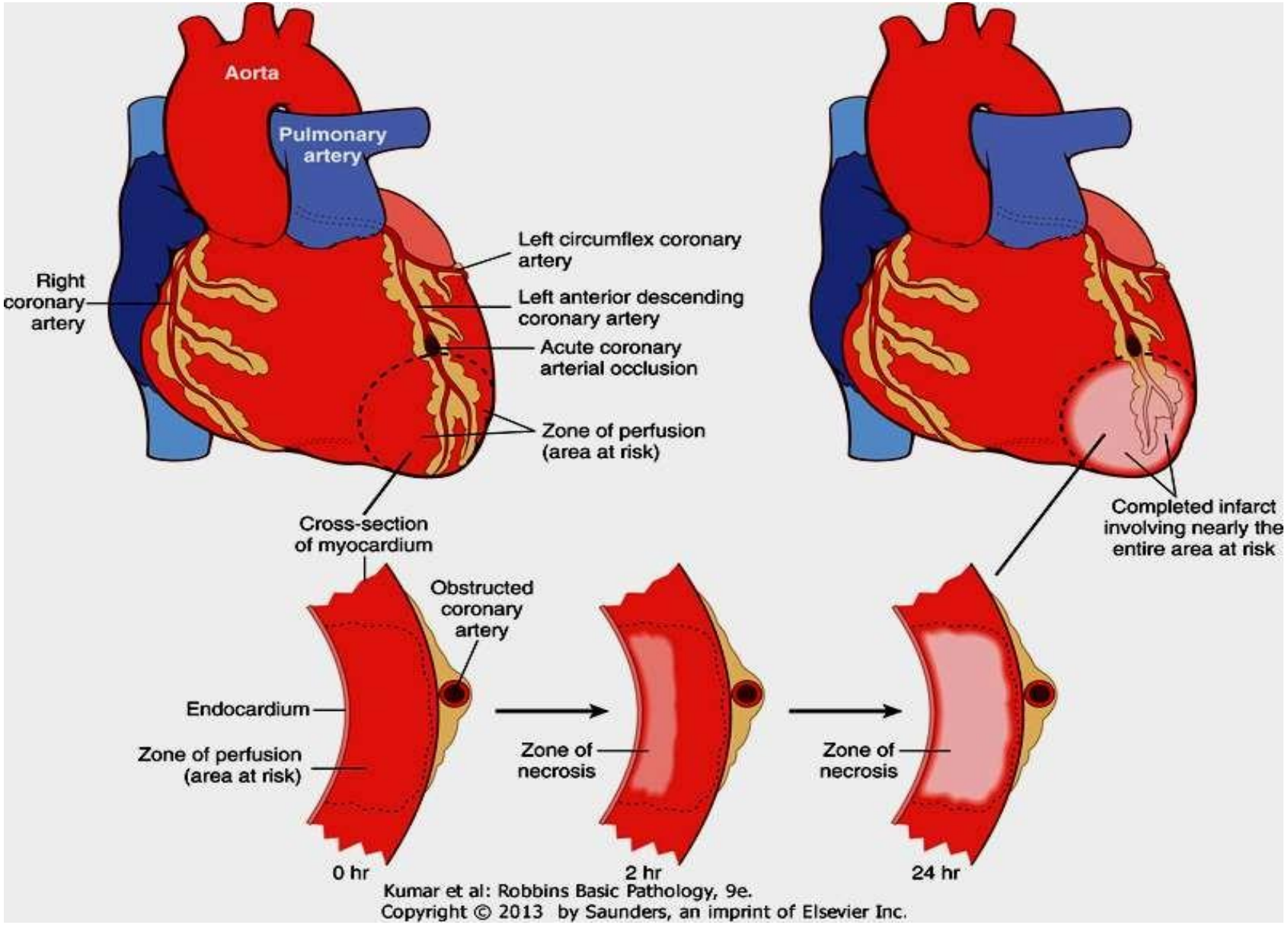
MI- Evolution



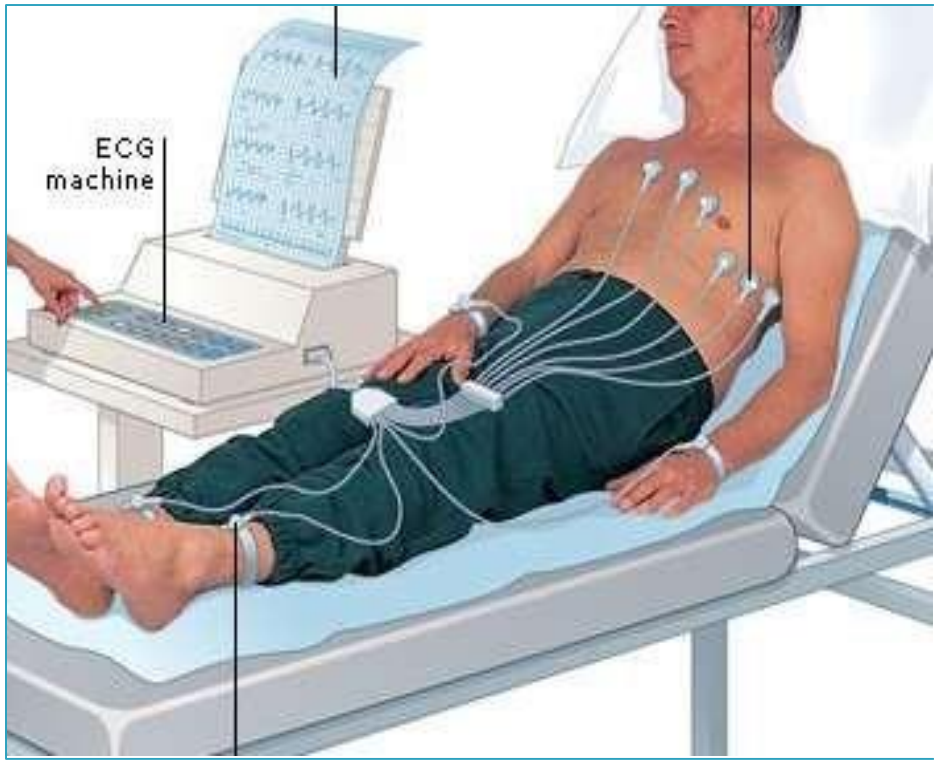
- When there is an acute coronary arterial occlusion, the area of the myocardium supplied by that segment of the artery (the zone of perfusion) will be at risk of infarction.
- The infarction will start to develop gradually, and eventually, this will result in death of the cardiac muscle covering that zone of perfusion.
- This is how the zone of perfusion transforms into a zone of necrosis

Evaluation of MI

1- Clinical signs and symptoms

*2- **Electrocardiographic (ECG)** abnormalities*

*3- **Laboratory evaluation:*** blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes.



Cardiac enzymes in MI

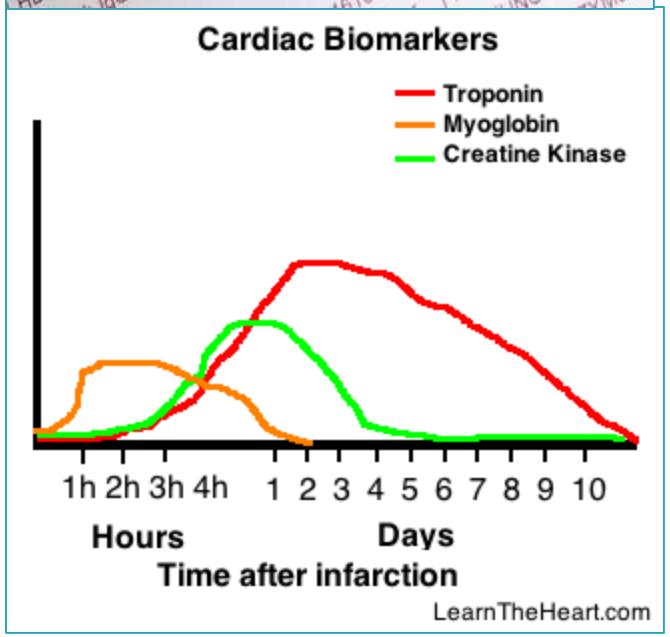
1-Myoglobin

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.



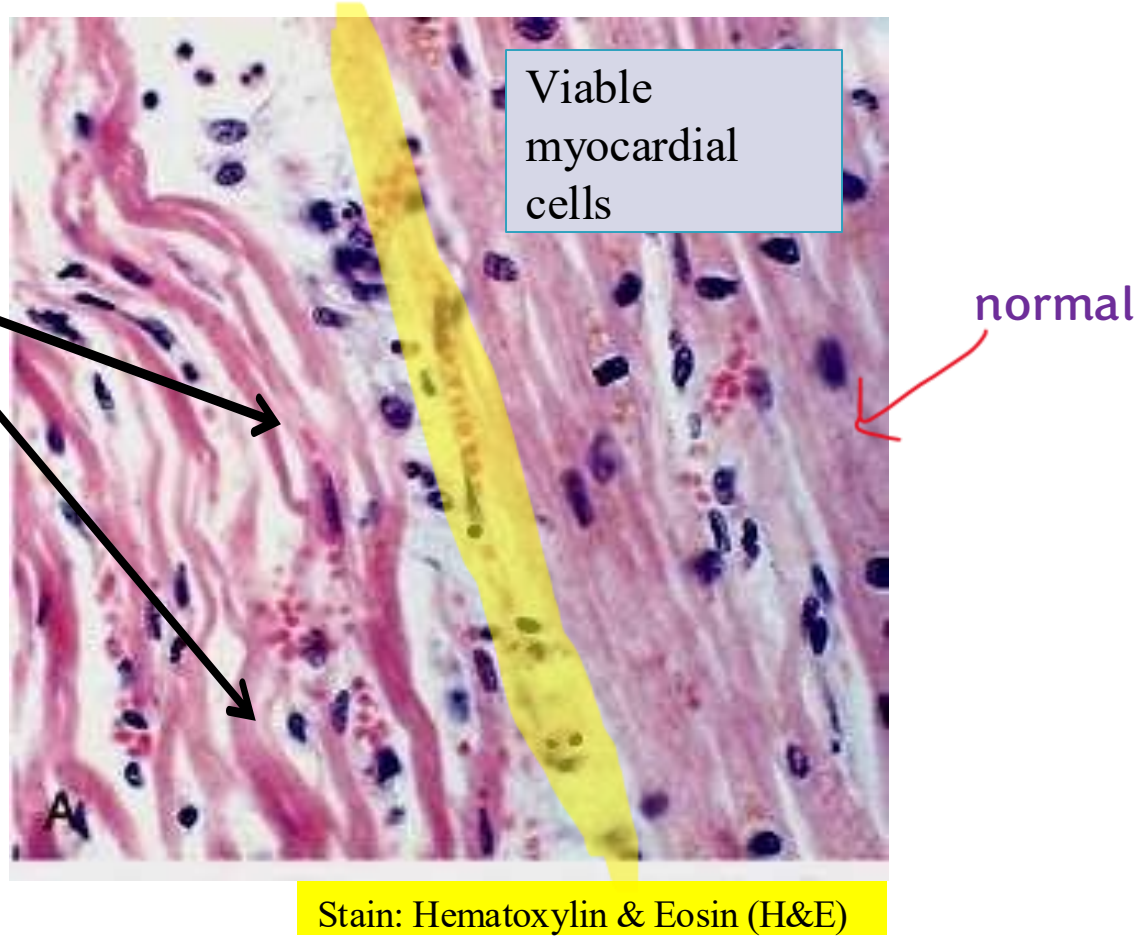
الحمد لله

Microscopic features of myocardial infarction and its repair.

<24 hr:

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

Necrotic cells (Dead cells which are characterized by loss of nuclei, shrinkage, denser and more eosinophilic cytoplasm, and may assume an abnormal position) are separated by edema fluid



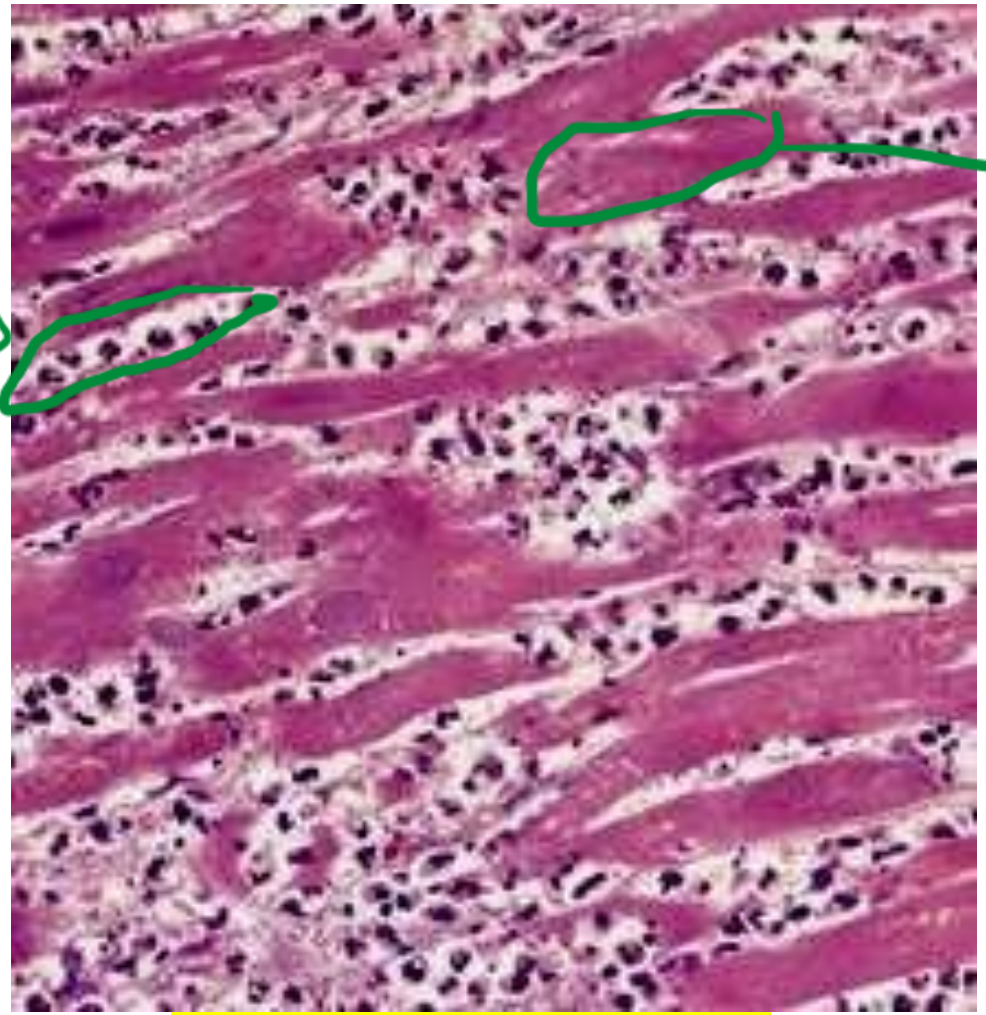
Stain: Hematoxylin & Eosin (H&E)

Microscopic features of myocardial infarction and its repair.

2 - 3 days:

Dense **neutrophil** infiltrate

Because necrosis always is followed by inflammation



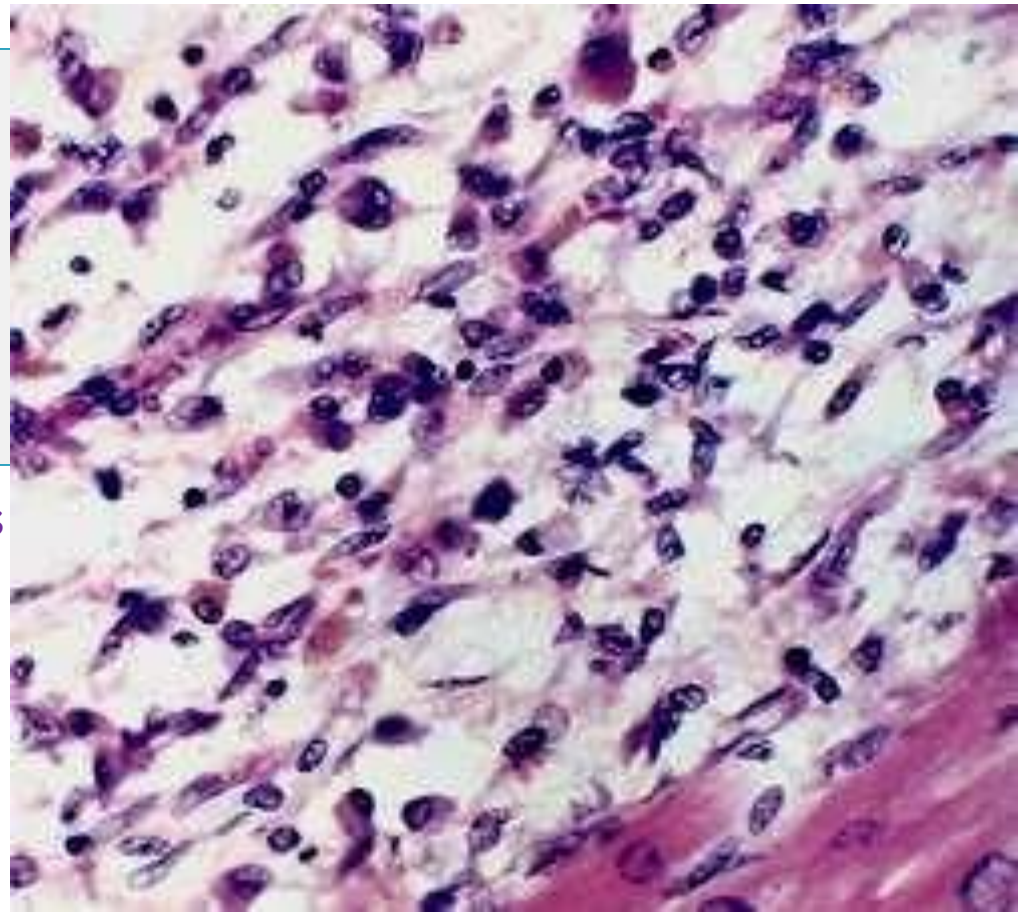
Dead cardiac cells

Stain: Hematoxylin & Eosin (H&E)

Microscopic features of myocardial infarction and its repair.

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

Different type of inflammatory cells come for cleaning of necrotic cells

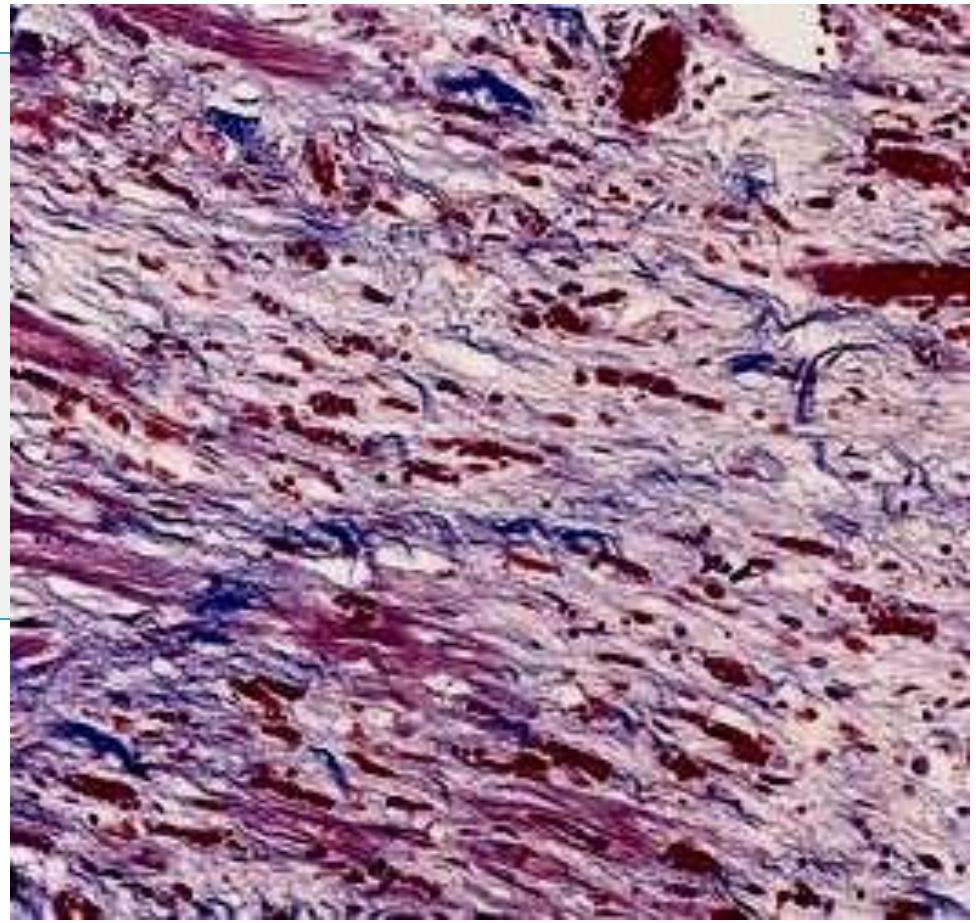


Stain: Hematoxylin & Eosin (H&E)

Microscopic features of myocardial infarction and its repair.

Tissue repair

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



Stain: Masson Trichrome (MT)

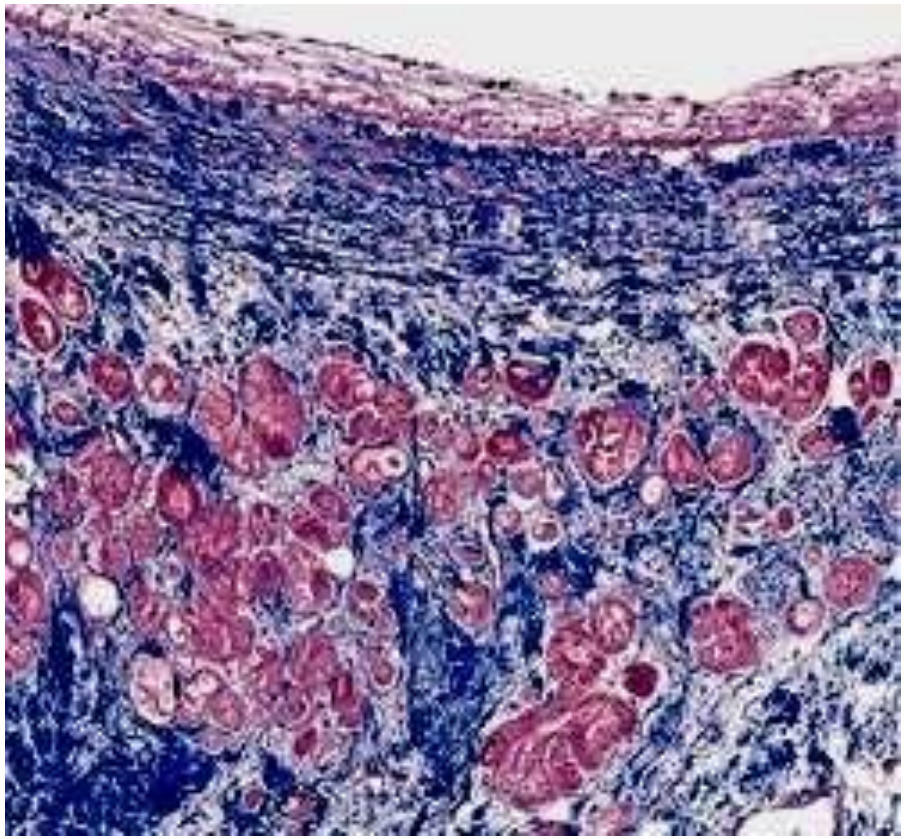
- Granulation tissue is not permanent, because it is just the base (scaffold) for repair.
- The repair will not be cardiac cells because cardiac muscle cells don't undergo mitosis.

سبحان الله

Microscopic features of myocardial infarction and its repair.

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

It may heal the defect, but it will never be as the normal myocardial in function (contractility or conductivity)



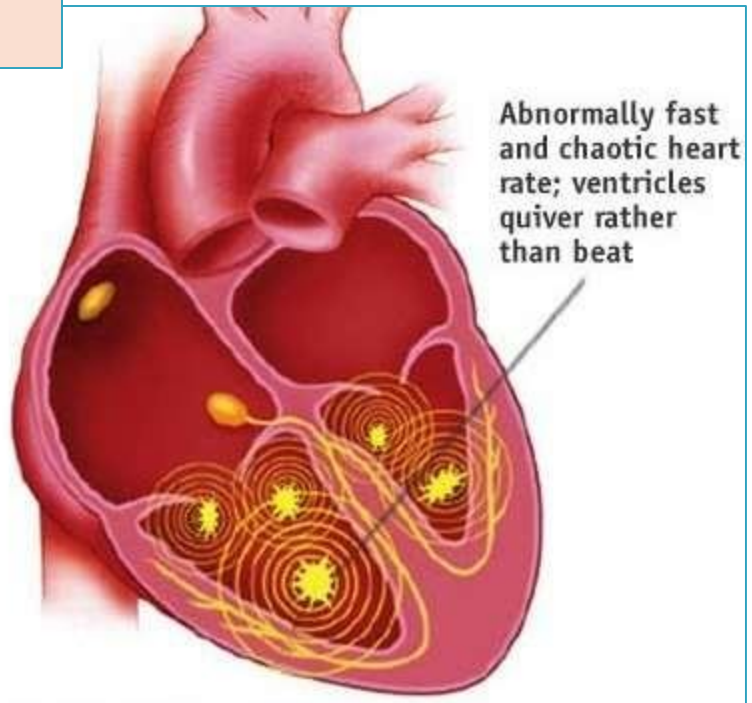
Stain: Masson Trichrome (MT)

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:

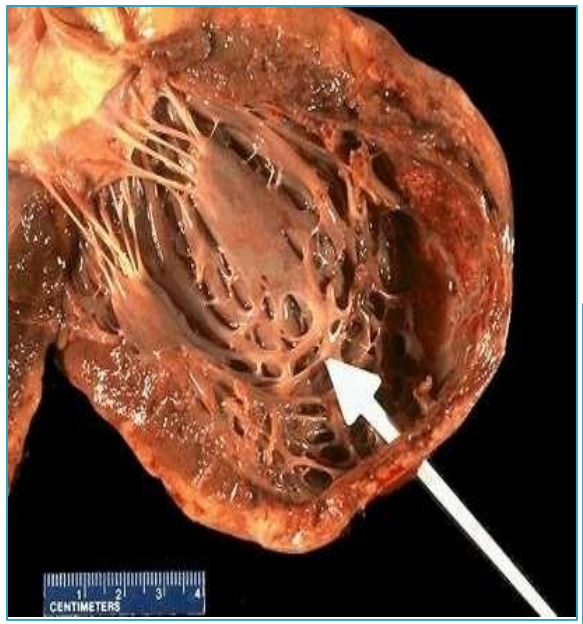
May happen before (most common) or after the scar formation

(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 or tricuspid regurgitation



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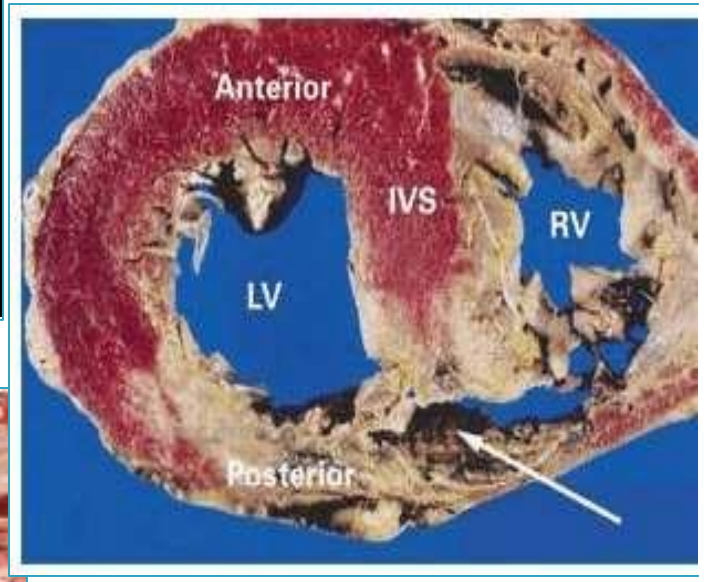

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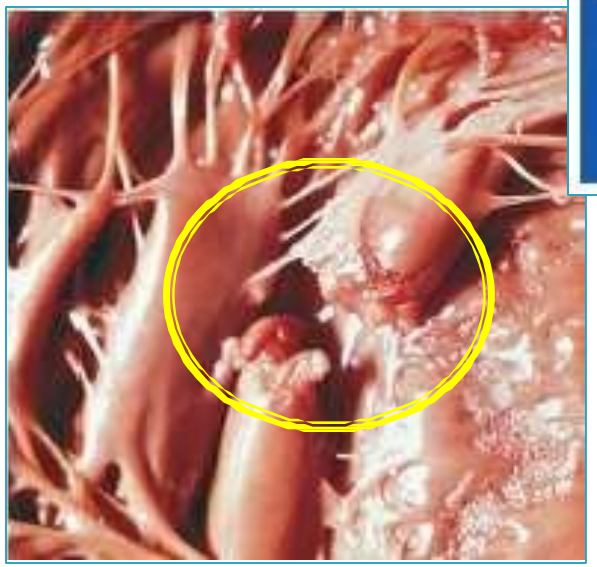

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4- Pericarditis.

- 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.

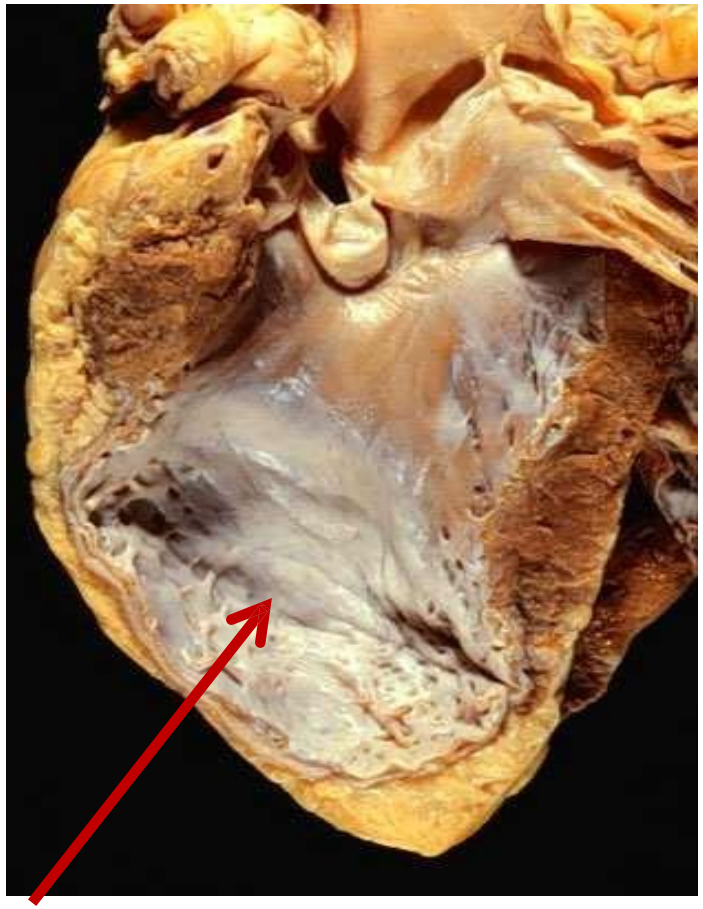
loss of contractility (causing stasis) + endocardial damage
→ ***thromboembolism***

7-Ventricular aneurysm. (True aneurysm), dilated and thinner wall

- A late complication
- most commonly result from a large transmural anteroseptal infarct that heals with the formation of thin scar tissue. So, its an old MI

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...

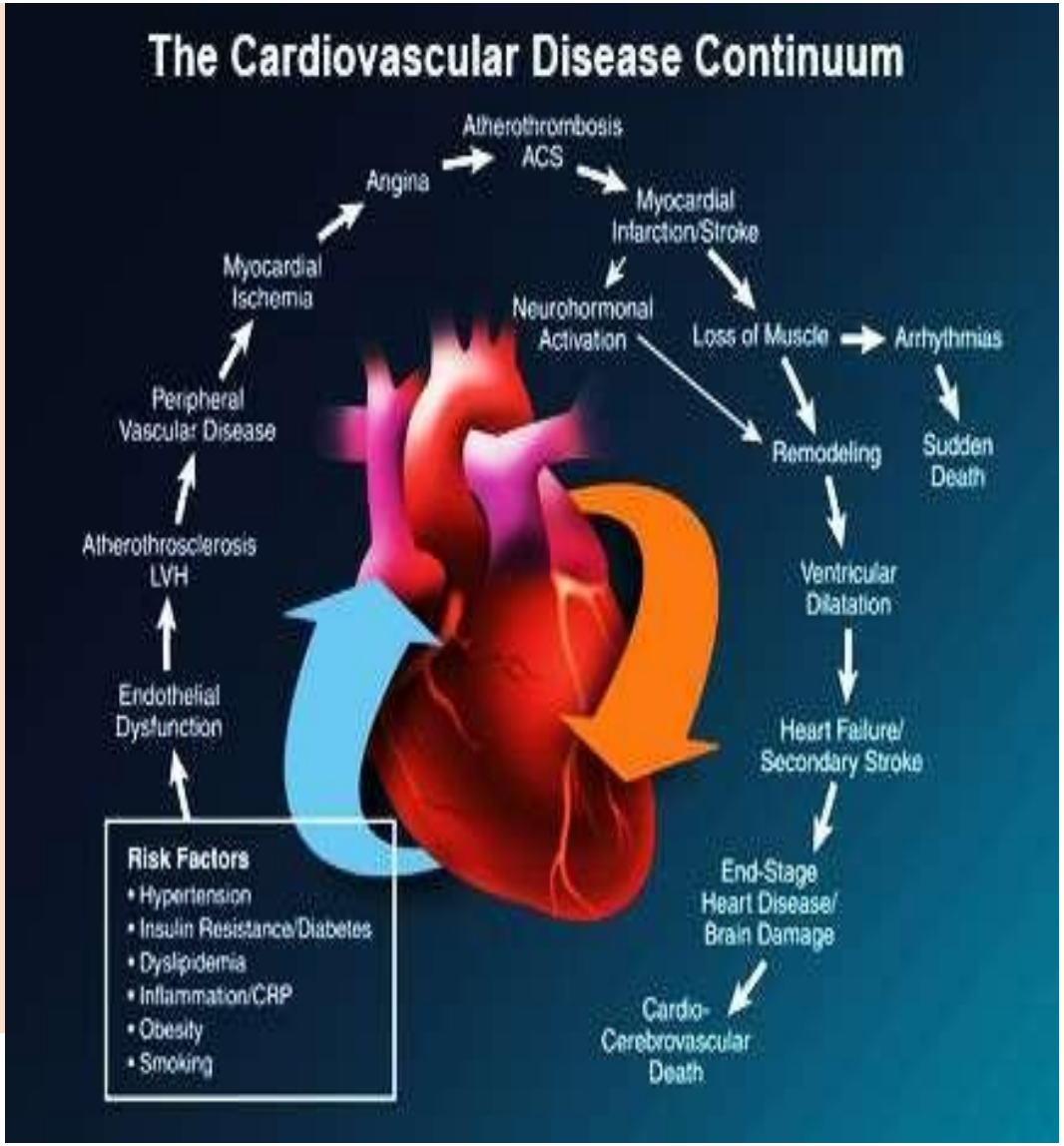
- 1st year mortality ≈ 30%.
- Thereafter, the annual mortality rate≈ 3%

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

……

- Any person even young or athletes may be susceptible to SCD

Physiology Quiz 7

الحمد لله رب العالمين

PATHOLOGY QUIZ

LECTURE 7

Scan the QR code or click it for FEEDBACK

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			