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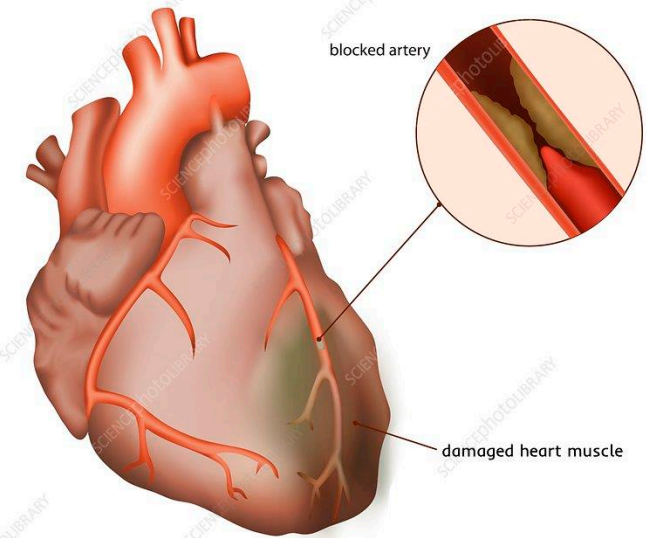
CVS module -4

ISCHEMIC HEART DISEASE

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13-11-2024

Ischemic Heart Disease

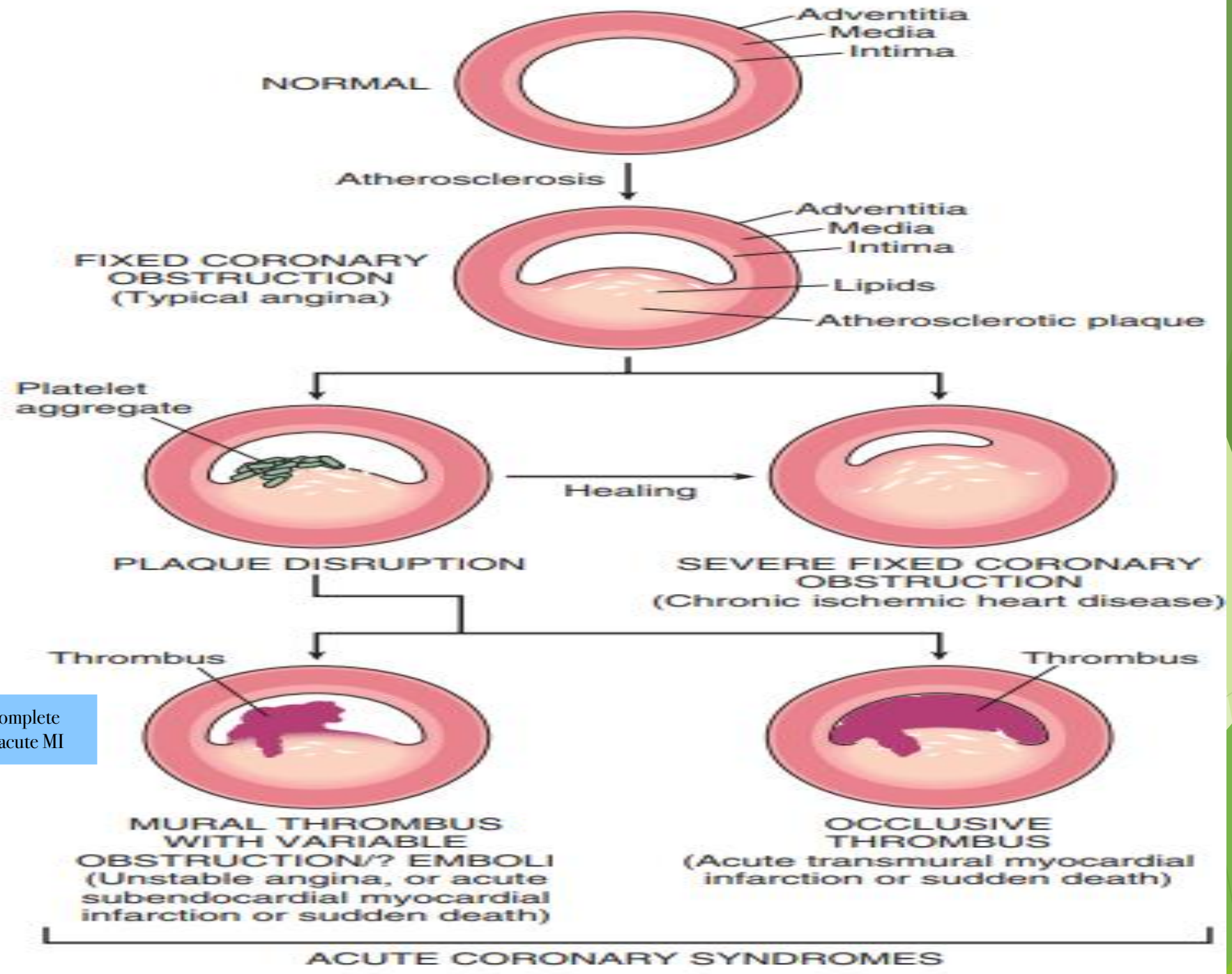


ISCHEMIC HEART DISEASE (IHD)

Due to occlusion

- ▶ A broad term including several closely related syndromes caused by myocardial ischemia (an imbalance between cardiac blood supply (perfusion) and myocardial oxygen and nutritional requirements).
- ▶ IHD usually is a consequence of reduced coronary blood flow secondary to obstructive atherosclerotic vascular disease
- ▶ In more than 90% of cases, IHD is a consequence of reduced coronary blood flow secondary to obstructive atherosclerotic vascular disease .

Thus, unless otherwise specified, **IHD usually is synonymous with coronary artery disease (CAD).**



Will get bigger then cause complete obstruction that will lead to acute MI

clinical presentation

Fast treatment : sublingual treatment

▶ Angina pectoris : Ischemia induces pain but is insufficient to cause myocyte death, can be:

- Stable (occurring predictably at certain levels of exertion). Always the same pain in the same activity
- Prinzmetal angina : caused by vessel spasm .
- unstable (occurring with progressively less exertion or even at rest).

Might differ between days some days might not have pain others may have

▶ Myocardial infarction (MI): cardiomyocyte death. Irreversible

▶ Chronic IHD with CHF.

Thrombus
Atherosclerosis plate
Now new injury
That gave time to the collaterals

Ischemia chronic in old age will make collaterals

But young age may not give them collaterals

- This progressive cardiac decompensation, which occurs after acute MI or secondary to accumulated small ischemic insults, eventually precipitates mechanical pump failure.

Even with the collaterals if there is less oxygenation
it will cause certain pathogenesis

If occlusion more than 70% open heart
surgery due to its failure

▶ Sudden cardiac death (SCD): result from tissue damage from MI, or from lethal arrhythmia .

clinical presentation

► Generally, cardiac ischemia may be the result of:

- Increased demand (e.g., with increased heart rate or hypertension).
- Diminished blood volume (e.g., with hypotension or shock).
- Diminished oxygenation (e.g., due to pneumonia or CHF) There is blood with no enough oxygen
- Diminished oxygen-carrying capacity (e.g., due to anemia or carbon monoxide poisoning) There is oxygen with low RBCs count

Pathogenesis

Increase demand should be with high perfusion

- ▶ IHD is a consequence of inadequate coronary perfusion relative to myocardial demand.
 - ▶ Due to preexisting atherosclerosis/new, superimposed thrombosis/vasospasm.

Fixed obstructions that occlude less than 70%:
Asymptomatic

Fixed lesions that occlude more than 70%:
Critical stenosis
Stable angina

Fixed stenosis that occludes 90%
:
Unstable angina

Critical
Any insult will close it and cause MI

With sublingual pills

Then any **dynamic changes** in coronary plaque morphology followed by thrombosis leading to :
ACUTE CORONARY SYNDROME

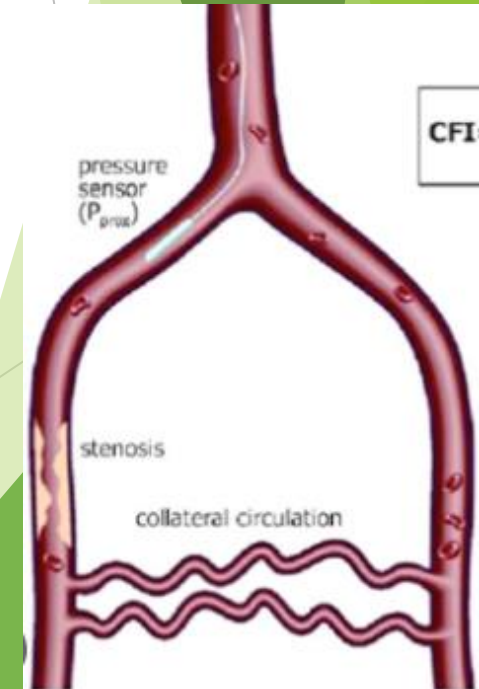
Acute coronary syndrome

- ▶ **Acute coronary syndromes result from acute obstruction of a coronary artery, and applied to any of the three catastrophic manifestations of IHD:**
 1. Unstable angina
 2. MI
 3. SCD.

collateral perfusion

Well established

- ▶ If an atherosclerotic lesion progressively occludes a coronary artery at a sufficiently slow rate over years, other coronary vessels may undergo remodeling and provide compensatory blood flow to the area at risk; such collateral perfusion can subsequently protect against MI, even if the original vessel becomes completely occluded.
- ▶ Unfortunately, with acute coronary blockage, there is no time for collateral flow to develop and infarction results.



Acute Plaque Change

- ▶ The initiating event is typically a sudden disruption (ranging from erosion to rupture) of a partially occlusive plaque.
- ▶ More than one mechanism of injury may be involved:
 - ▶ Rupture,
 - ▶ Fissuring.
 - ▶ Ulceration of plaques.
 - ▶ Hemorrhage into the core of plaques .

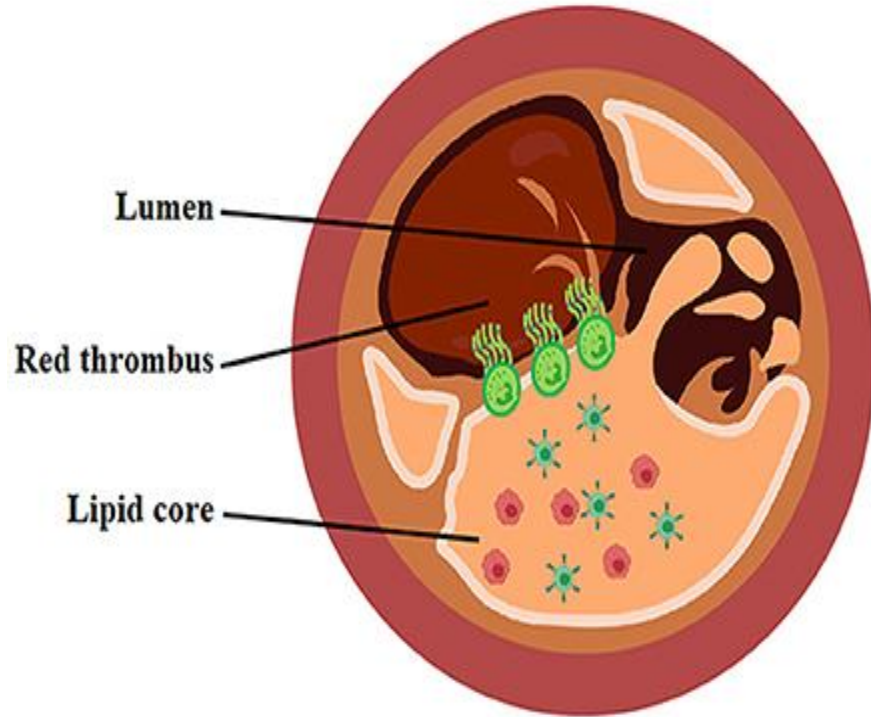
Expose highly thrombogenic constituents or underlying subendothelial basement membrane, leading to rapid thrombosis.

Due to rupture with the weak granulation tissue with any external pressure towards it

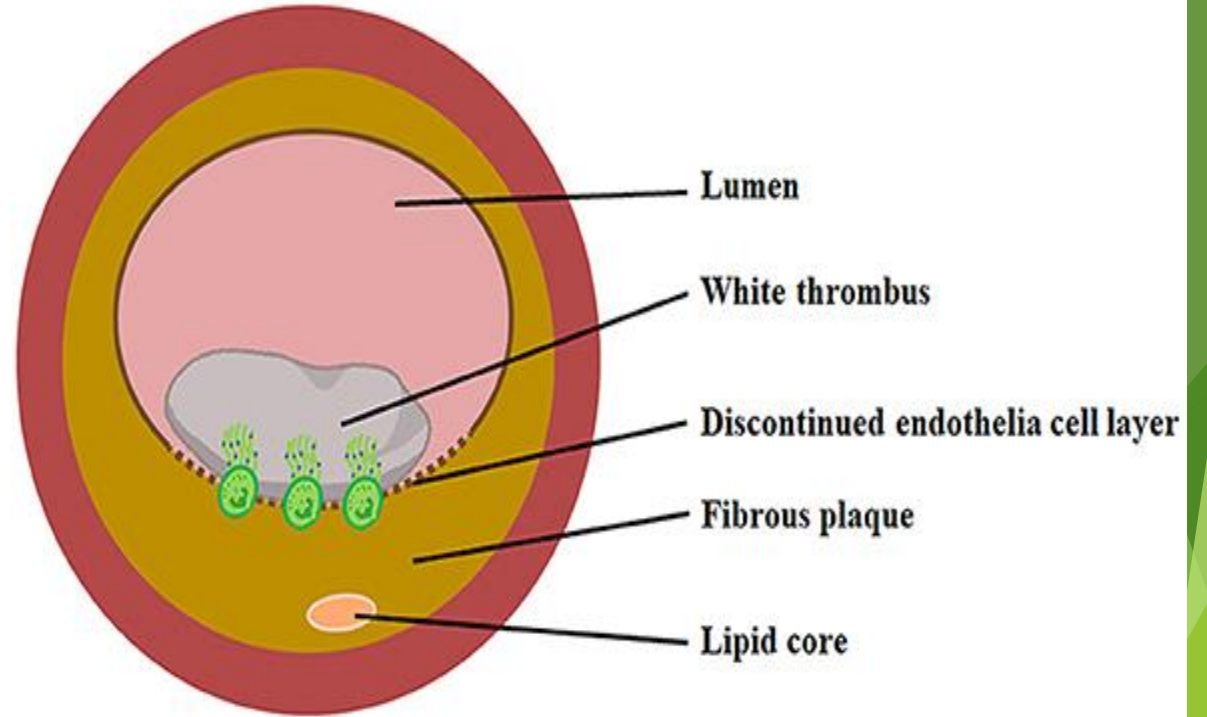
Picture for rupture, fissuring

Rupture that will invite the neutrophils ليعدلو
erusion بعدين بصير

Plaque rupture



Plaque erosion



T cell



Macrophage



Neutrophil extracellular traps

Participating factors

- ▶ Inflammation: plays an essential role from inception to plaque rupture.
- ▶ Thrombosis : eroded or ruptured plaque triggers the acute coronary syndromes.
- ▶ Vasoconstriction, mediated by:
 - Circulating adrenergic agonists.
 - Locally released platelet contents.
 - Imbalance between endothelial cell-relaxing factors (e.g., nitric oxide) and -contracting factors (e.g., endothelin) due to endothelial dysfunction.
 - Mediators released from perivascular inflammatory cells.

1. Angina Pectoris

- ▶ Angina pectoris is an intermittent chest pain caused by transient, reversible myocardial ischemia. Mediators
- ▶ The pain is a consequence of the ischemia-induced release of adenosine, bradykinin, and other molecules that stimulate autonomic nerves.

The pain is a good sign to inform the patient

▶ Subtypes:

▶ 1. Stable angina:

- episodic chest pain associated with particular levels of exertion or some other increased demand .
- The pain is described as a crushing or squeezing substernal sensation that radiates down the left arm or to the left jaw (referred pain).
- The pain usually is relieved by rest (reducing demand) or by drugs such as nitroglycerin, a vasodilator that increases coronary perfusion.

Under the sublingual is better for its fast absorption without the slow pathway that will enter the stomach

► 2• Prinzmetal angina:

- occurs at rest and is caused by coronary artery spasm.
- such spasms typically occur on or near existing atherosclerotic plaques or completely normal vessel .
- Prinzmetal angina typically responds promptly to vasodilators such as nitroglycerin and calcium channel blockers.

► 3• Unstable angina (also called crescendo angina):

- Characterized by increasingly frequent pain, precipitated by progressively less exertion or even occurring at rest.
- Associated with plaque disruption and superimposed thrombosis, distal embolization of the thrombus, and/or vasospasm; it can be a harbinger of MI, portending complete vascular occlusion.

Complete occlusion will be irreversible death of the myocyte

2. Myocardial Infarction (heart attack).

- ▶ Necrosis of the heart muscle resulting from ischemia.

Death of myocytes

- ▶ The major underlying cause is atherosclerosis.

- ▶ In general, women tend to be protected against MI during their reproductive years.

Will be equal in both genders by age

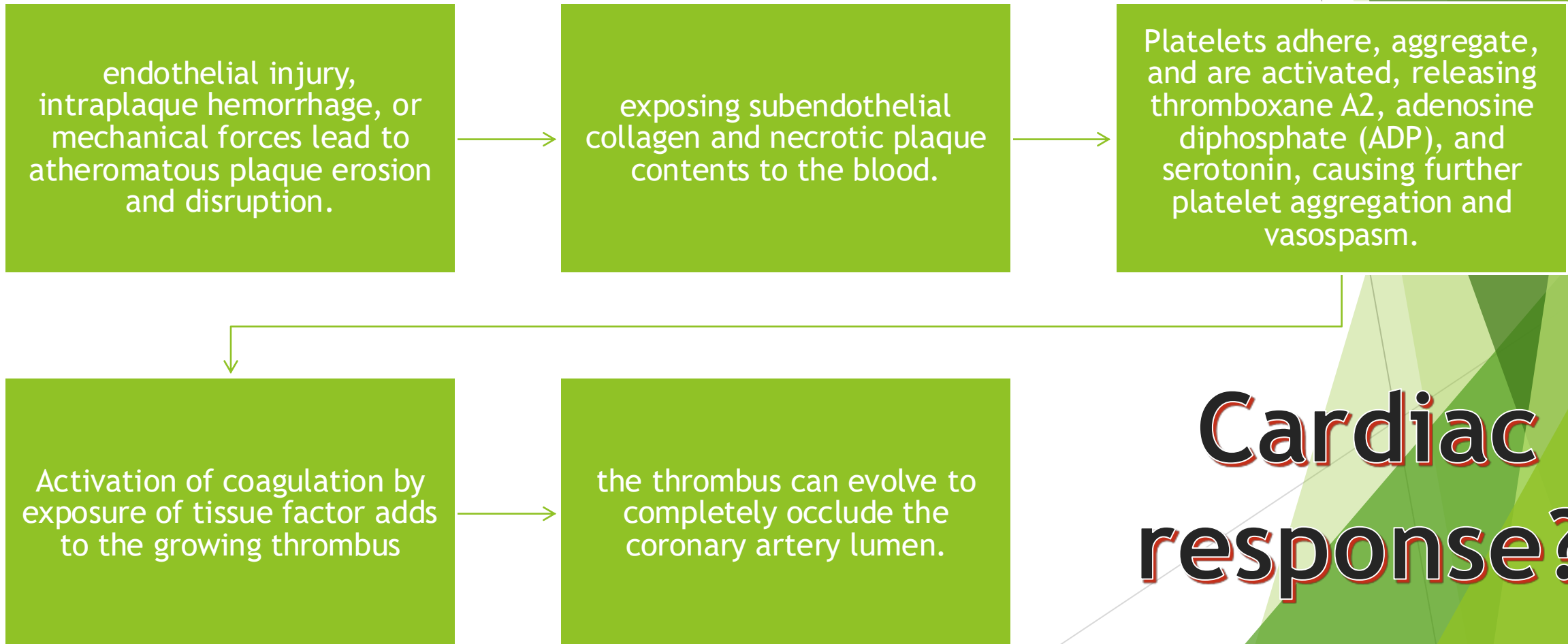
- ▶ Menopause with declining estrogen production, is associated with exacerbation of coronary artery disease, and IHD is the most common cause of death in older adult women.

Pathogenesis

- ▶ The vast majority of MIs are caused by acute thrombosis within coronary arteries, in form of:
 - disruption or erosion of preexisting atherosclerotic plaque.
 - vascular occlusion, and subsequent infarction of the perfused myocardium.
- ▶ 10% of MI occurs in the absence of occlusive atherosclerotic vascular disease; such infarcts are mostly result from :
 - coronary artery vasospasm .
 - embolization from mural thrombi (e.g., in the setting of atrial fibrillation) or from valve vegetations.

90% from occlusion
10% by the spasms

In a typical MI, the following sequence of events takes place:



**Cardiac
response??**

Myocardial Response to Ischemia

Their way in providing energy

- ▶ Since cardiac myocytes generate energy almost exclusively through mitochondrial oxidative phosphorylation, cardiac function is strictly dependent upon the continuous flow of oxygenated blood through the coronary arteries.

تتوقف تماما

- ▶ Within seconds of vascular obstruction, aerobic metabolism ceases, leading to a drop in adenosine triphosphate (ATP) and accumulation of lactic acid, leading to:

- Early (reversible) effect: rapid loss of contractility.
- prolonged ischemia lasting at least 20 to 40 minutes (irreversible):
 - coagulative necrosis of myocytes.
 - arrhythmias e.g. ventricular fibrillation.

Patterns of Infarction

Not all are fatal

- ▶ The location, size, and morphologic features of an acute myocardial infarct depend on multiple factors:
 - 1• Size and distribution of the involved vessel.
 - 2• Rate of development and duration of the occlusion. Because of the collaterals
 - 3• Metabolic demands of the myocardium (affected, for example, by blood pressure and heart rate).
 - 4• Extent of collateral supply. Depend on the duration

According to the distribution

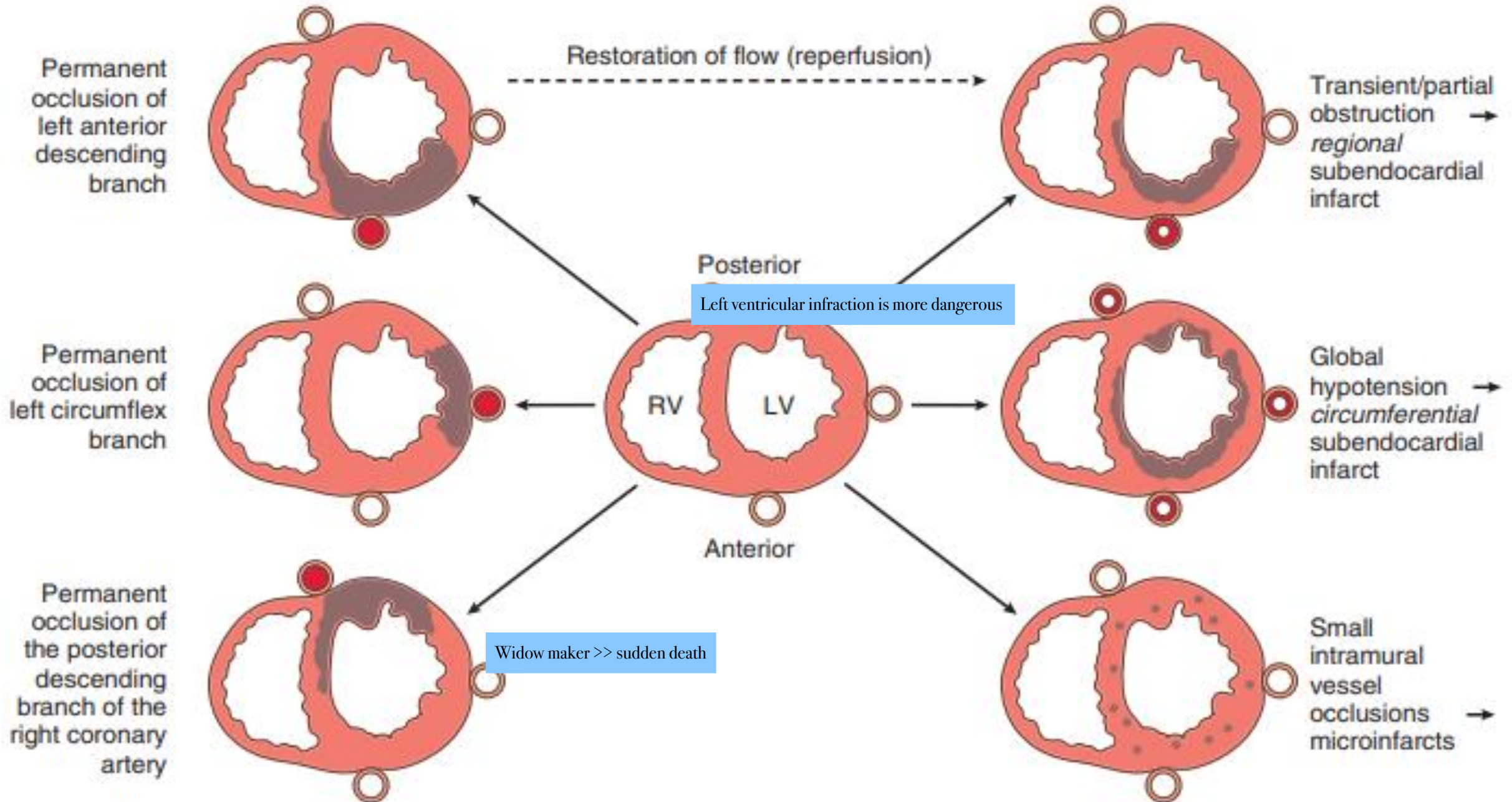
*Acute occlusion of the proximal left anterior descending (LAD) artery causes 40%-50% of all MIs & typically results in infarction of anterior wall of left ventricle, anterior two thirds of ventricular septum, & most of the heart apex.

*Proximal right coronary artery (RCA) occlusion (30% to 40% of MIs) affects much of the right ventricle.

*Acute occlusion of the proximal left circumflex (LCX) artery (15%-20% of MIs) causes necrosis of the lateral left ventricle,

TRANSMURAL INFARCTS

NON-TRANSMURAL INFARCTS



- ▶ Transmural infarctions (ST-segment elevated MIs (STEMIs).
- involve the full thickness of the ventricle & are caused by epicardial vessel occlusion
- Give rise to: ST segment elevations on (ECG) and can have negative Q waves.

- ▶ Subendocardial infarctions (non-ST-segment elevated MIs” / “NSTEMIs).

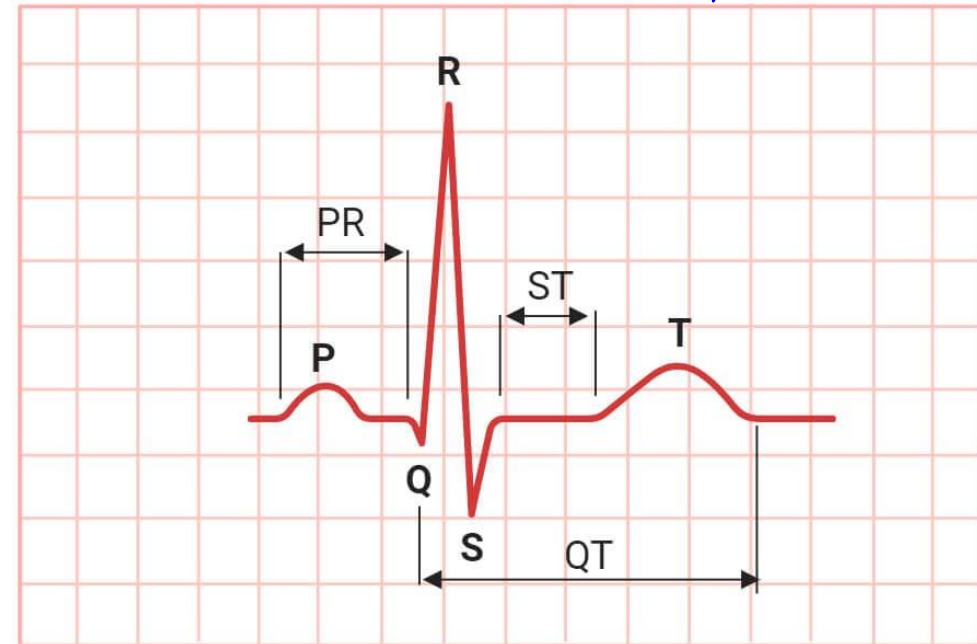
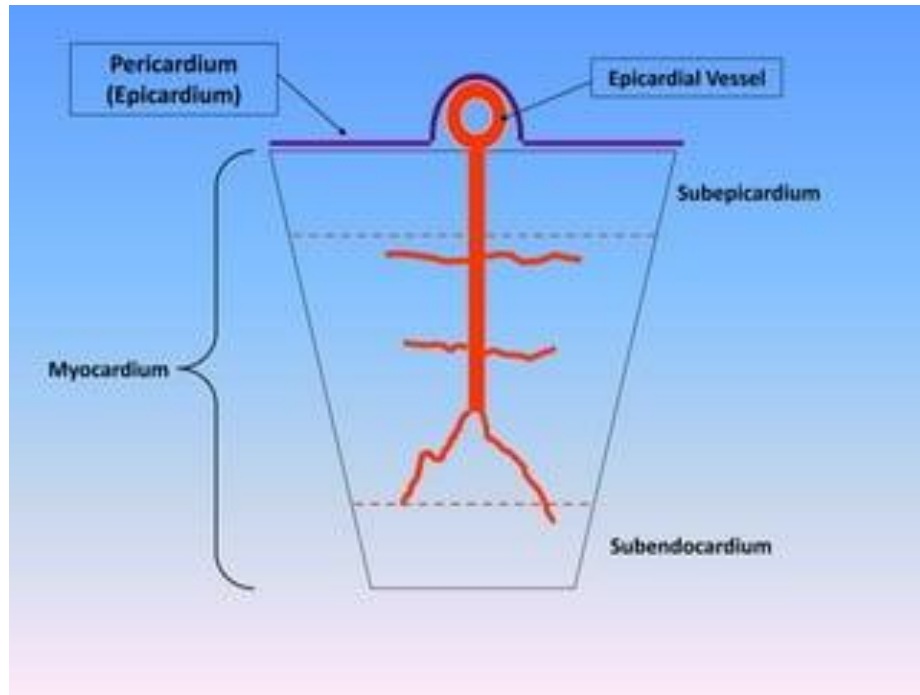
- are limited to the inner third of myocardium.

- No ST segment elevations on ECG

مش يعني لو ما في
ST segment elevation يعني ما في infraction

un-stemi

عنه بس
عنى
رج
فائل
للمعرفة



Electrocardiogram
(ECG)

Gross morphology

postmortem autopsy revealed areas of Coagulative necrosis.

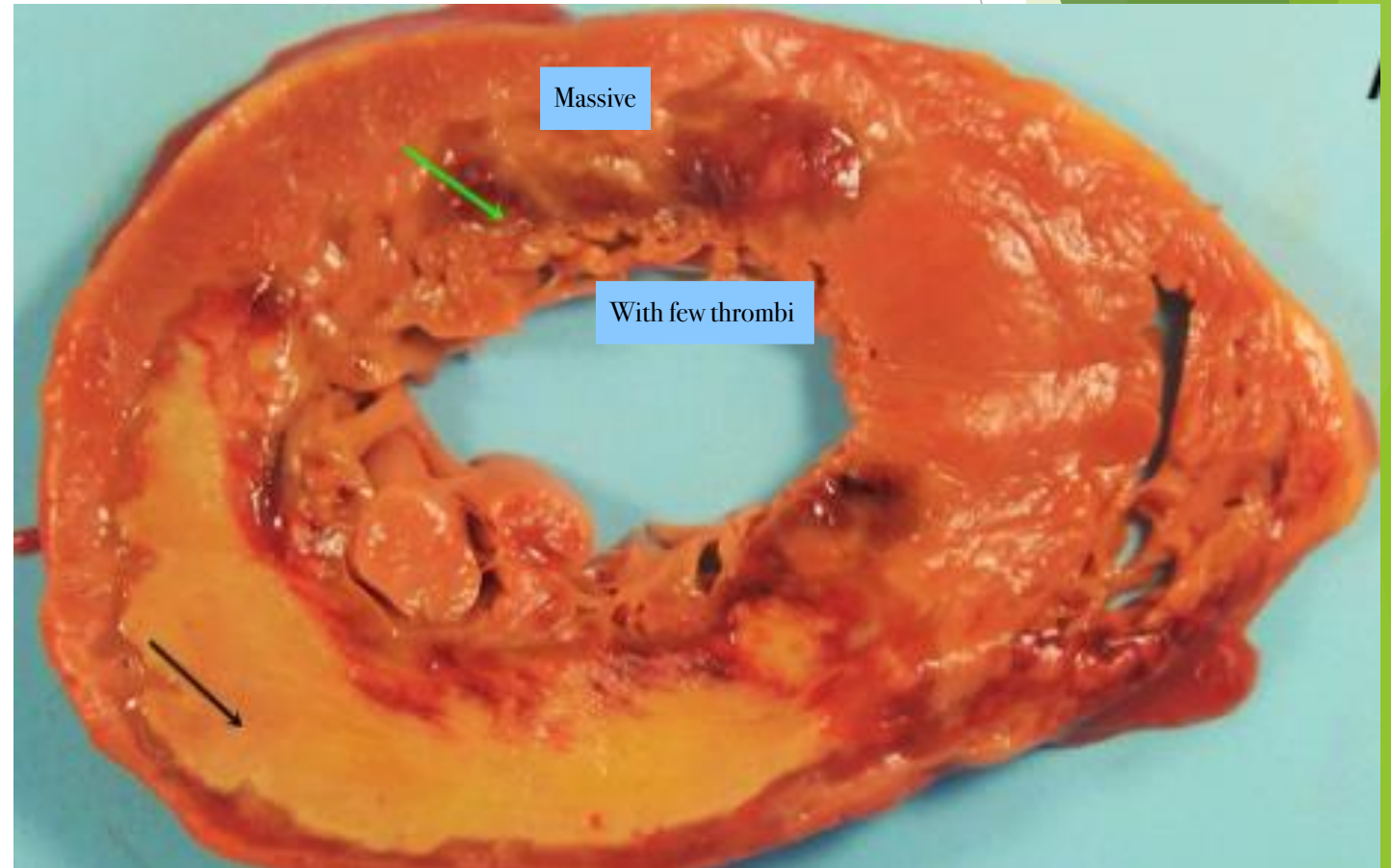


Table 11.2 Evolution of Morphologic Changes in Myocardial Infarction

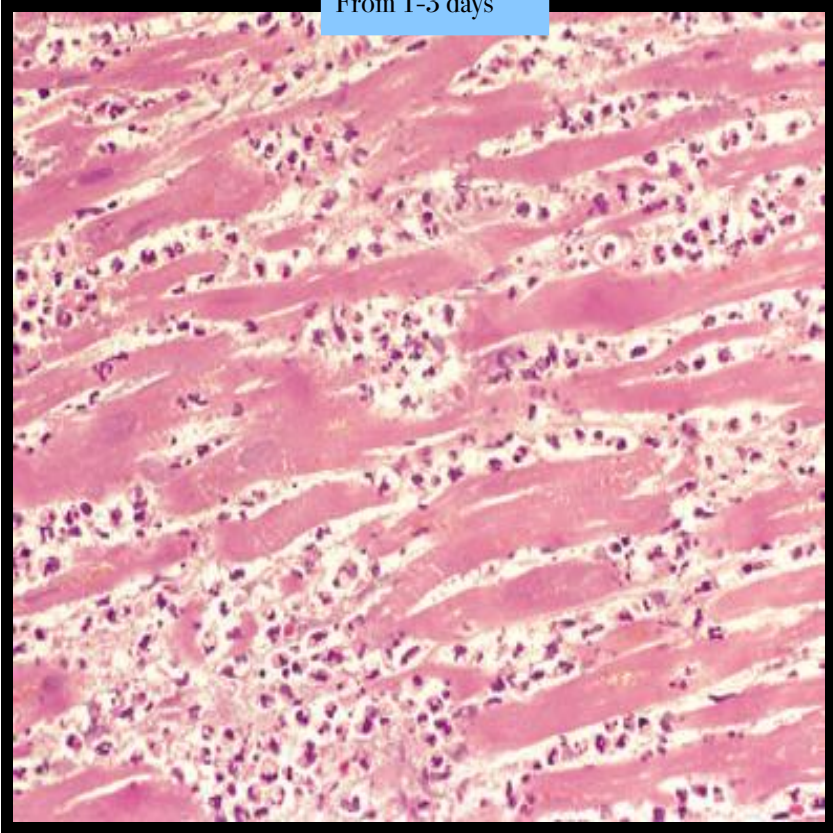
Very important



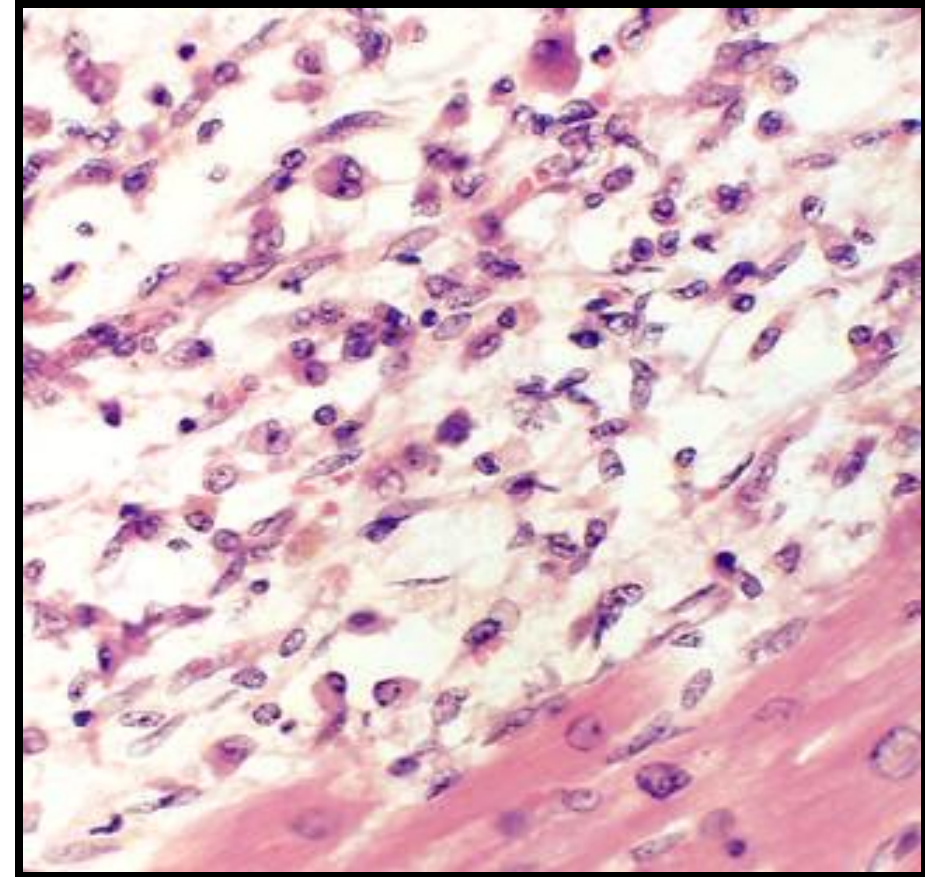
Time Frame	Gross Features	Light Microscopic Findings	Electron Microscopic Findings
Reversible Injury			
0–½ hour	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
★ Irreversible Injury			
½–4 hours	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
★ 4–12 hours	Occasionally dark mottling	Beginning coagulation necrosis; edema; hemorrhage	
12–24 hours	Dark mottling	Ongoing coagulation necrosis; pyknosis of nuclei; hypereosinophilic appearance of myocytes; marginal contraction band necrosis; beginning neutrophilic infiltrate	
1–3 days	Mottling with yellow-tan infarct center	Coagulation necrosis with loss of nuclei and striations; interstitial infiltrate of neutrophils	
★ 3–7 days	Hyperemic border; central yellow-tan softening	Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border	
★ 7–10 days	Maximally yellow-tan and soft, with depressed red-tan margins	Well-developed phagocytosis of dead cells; early formation of fibrovascular granulation tissue at margins	
★ 10–14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2–8 weeks	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
★ >2 months	Scarring complete	Dense collagenous scar	

Histopathological features

neutrophils
بداية ال
From 1-3 days



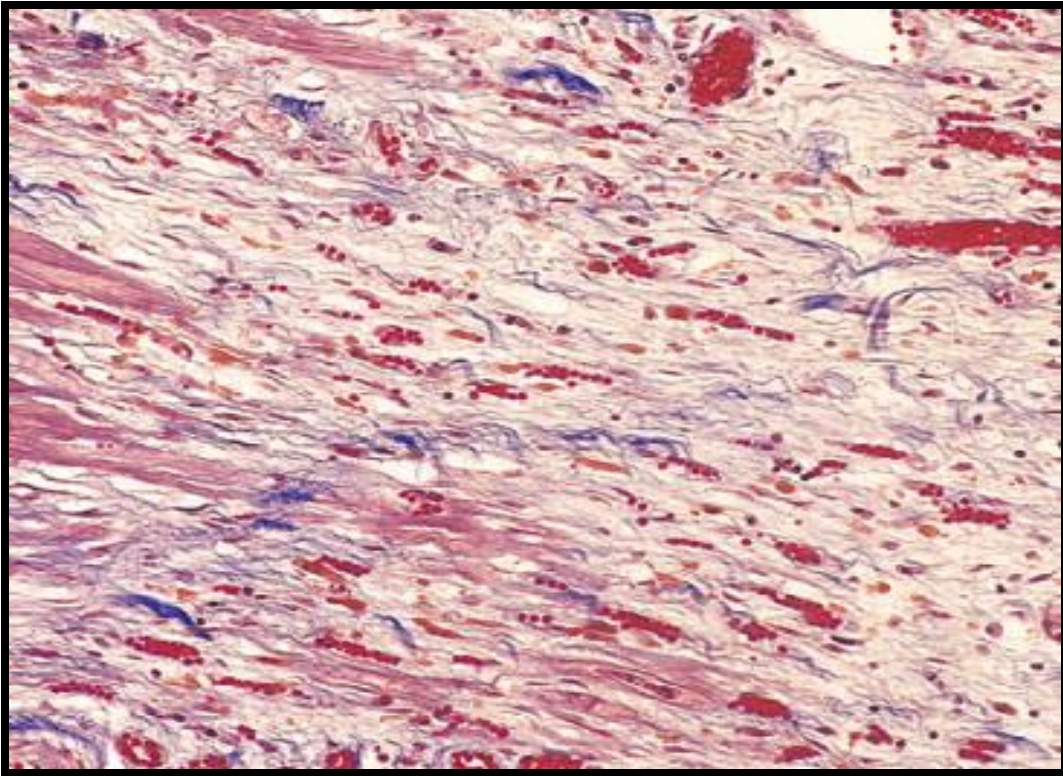
Coagulation necrosis with loss of nuclei and striations; interstitial infiltrate of neutrophils



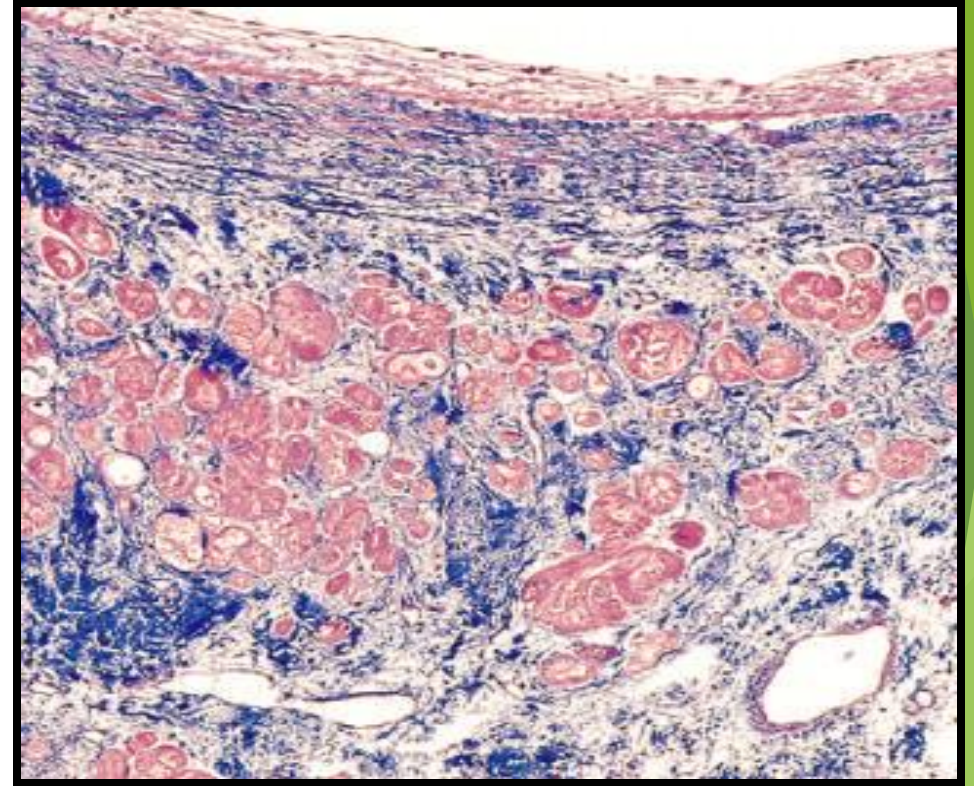
Complete removal of necrotic myocytes by phagocytic macrophages

Granulation tissue

Histopathological features cont.



well established granulation tissue
with new blood vessels & collagen
deposition.



Dense collagenous scar

Scar with more than 2 months

Clinical features

- Retrosternal pain radiate to the neck, jaw, epigastrium, or left arm, not relieved by rest or vasodilators.
- Persist for several hours (>20-30 min).
- Nausea, vomiting, sweating & weakness .
- ▶ LAB: elevated cardiac enzyme: leak out of injured myocardial cells through damaged cell membranes.
- E.g. troponins , creatine kinase (CK)
- ▶ Electrocardiographic abnormalities, include: ما حينسأل عنها
- Q waves, ST segment changes, and T wave inversions.

Complications of MI

- ▶ Contractile dysfunction in form of: who survived
 - left ventricular failure manifested as hypotension, pulmonary congestion, & pulmonary edema.
- ▶ Arrhythmias with risk of sudden cardiac death.
- ▶ Papillary muscle dysfunction or rupture.
- ▶ Myocardial rupture: during healing process, necrotic myocardium converted to soft, friable granulation tissue.
- ▶ Ventricular aneurysm.
- ▶ Mural thrombus

Chronic Ischemic Heart Disease

- Chronic IHD, also called ischemic cardiomyopathy, is a progressive heart failure secondary to ischemic myocardial damage.
- Mostly there is a known clinical history of previous MI.
- After prior infarction(s), chronic IHD appears when the compensatory mechanisms (e.g., hypertrophy) of residual myocardium begin to fail.
- severe CAD can cause diffuse myocardial dysfunction, and even micro-infarction and replacement fibrosis, without any clinically evident episode of frank infarction.

دكتور انجم
عليان مع
رعاية
وحياة مع
الاس