CVS

PATHOLOGY

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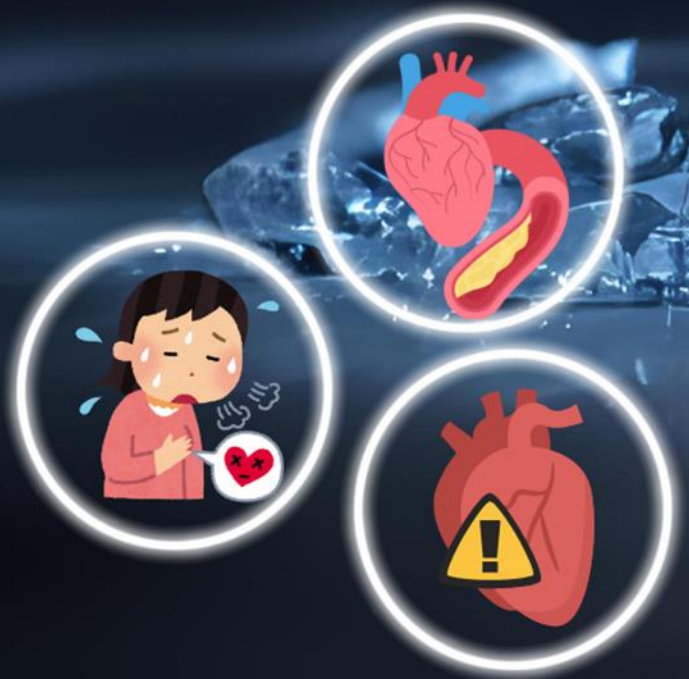

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Color code

- Slides
- Doctor
- Additional info
- Important

ISCHEMIC HEART DISEASE-2

Acute Myocardial Infarction

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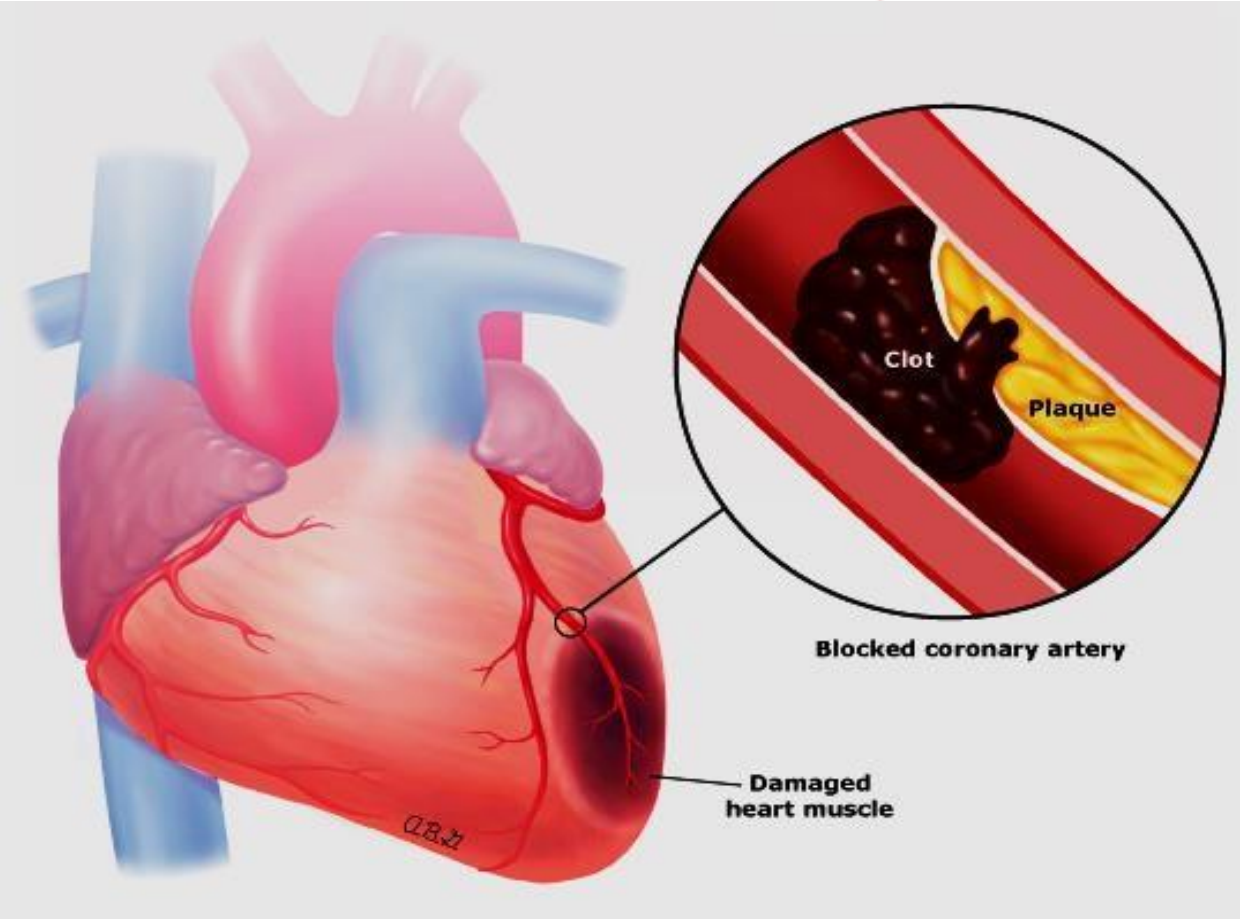
Acute Myocardial Infarction (MI)



- As you can see, there is an atherosclerotic plaque inside one of the coronary arteries and this atherosclerotic plaque is complicated by a superimposed blood clot (thrombus), this acute occlusion of the coronary artery will lead to ischemia of the downstream tissues that are supplied by that particular coronary vessel , so there will be damage and necrosis in the heart muscle that is supplied by that coronary artery, and that's **acute 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

The most significant and distinguishing one

dyspnea (shortness of breath) if pulmonary congestion and edema.

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

Dizziness; sweating

rapid and weak pulse

Related to sympathetic stimulation

nausea (in posterior MI)

because of activation of the vagus nerve.

Sometimes: No typical symptoms (silent infarcts)

occurs when there is massive MI (large portion or volume of the heart is affected), so when there is >40% of the left ventricle affected by MI, there will be loss of the pumping action of the heart leading to cardiogenic shock)

Silent infarcts (not classical symptoms):

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

1 DM (peripheral neuropathies)

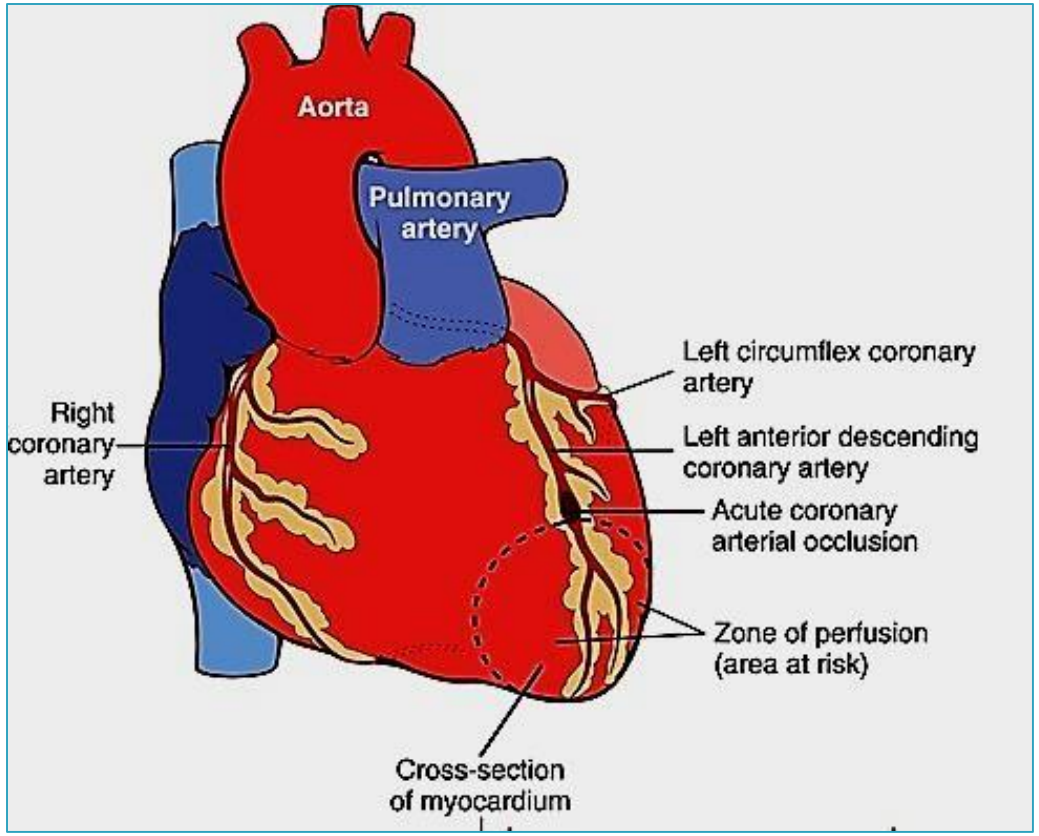
2 the elderly

Silent infarct describes a patient that is having acute MI without the classical symptoms of acute cardiac ischemia ; so, this type of MI can only be examined and confirmed by ECG and lab workup.

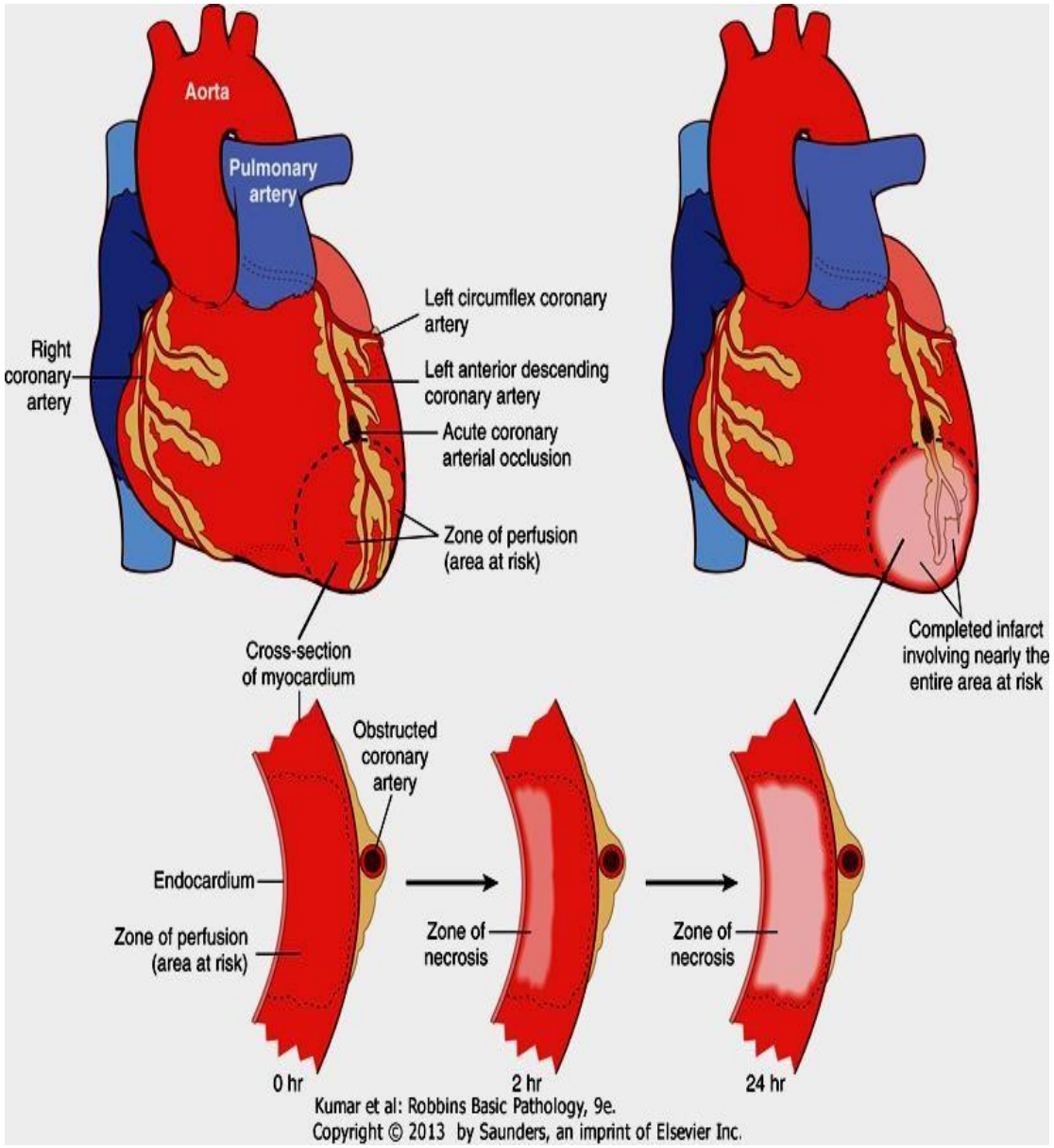
MI- Causes:

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

Remember that LAD artery branched from coronary artery.



MI- Evolution



The area that is supplied by occluded artery is at risk of ischemia and it is called **zone of perfusion**.

If you take a cross section from the myocardium of this area, you will see that the occluded coronary artery is located just beneath the pericardium overlying the myocardium, so the **endocardium is the furthest part of the cardiac tissue from the blood supply**, all this area is called zone of perfusion or area at risk of ischemia.

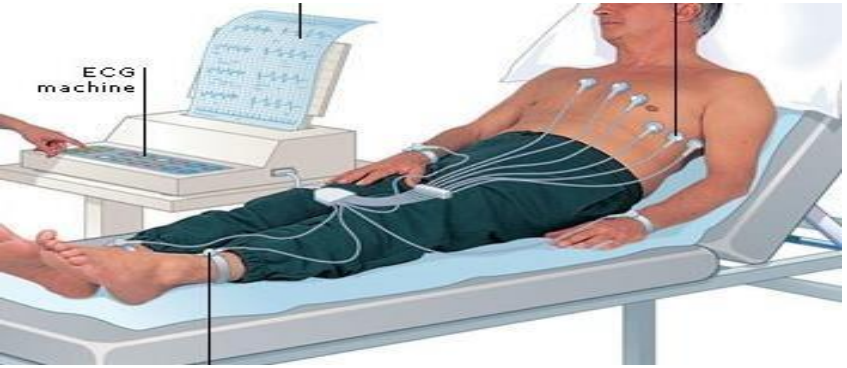
After more than 30 minutes, necrosis starts to develop and the **"zone of necrosis"** describes the area of necrotic cardiac tissue.
Note that necrosis begins just beneath the endocardium, so **the furthest part from the coronary artery blood supply is the first part of cardiac tissue to show necrosis** and these are called the **subendocardial areas of the myocardium.** However, Endocardium will be spared from the necrosis because it can get the blood supply through diffusion from cardiac chambers.

Two hours following the infarct, the zone of necrosis would be limited to the subendocardial myocardial muscle. If this area of ischemia (zone of perfusion) is not treated, zone of necrosis will expand until it covers all the area at risk (zone of perfusion).

24 hours after of the onset of occlusion, zone of perfusion is completely replaced by zone of necrosis.

Evaluation of MI

MI is the main cause of morbidity and mortality worldwide, so management is important.



- Clinical signs and symptoms
- **Electrocardiographic (ECG)** abnormalities:
  Detecting any abnormalities in the ECG could indicate MI. (usually done in emergency department).
- **Laboratory evaluation:** blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes.

Cardiac enzymes in MI

Those intracellular cardiac molecules that leak into the circulation following cardiac damage are called “Cardiac enzymes” or “Cardiac markers of MI”, these include:

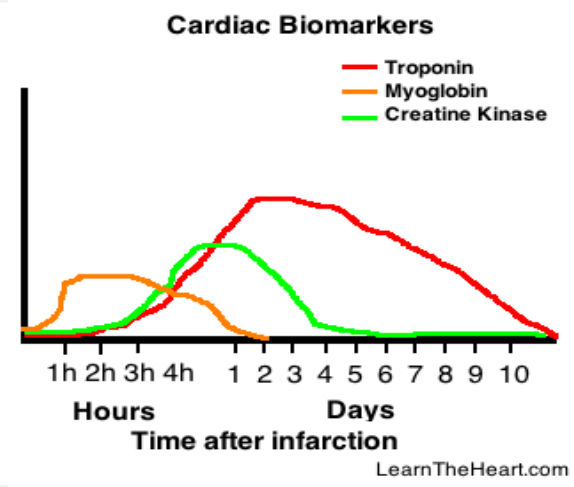
1-Myoglobin

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** because they are very specific for cardiac muscle and stay elevated for a long period following acute MI.
- Creatine kinase CK-MB isoform is **the second best marker** after the cardiac-specific troponins because it is also specific to cardiac muscles.



Microscopic features of myocardial infarction and its repair.

It is not a part of the diagnostic process of MI, you should understand that this is just an autopsy confirmation, so whenever someone dies due to cardiac causes and there is a suspicion or confirmation of acute MI; the following findings can be seen under the microscope, and they describe the chronological pattern and evolution of MI over time.

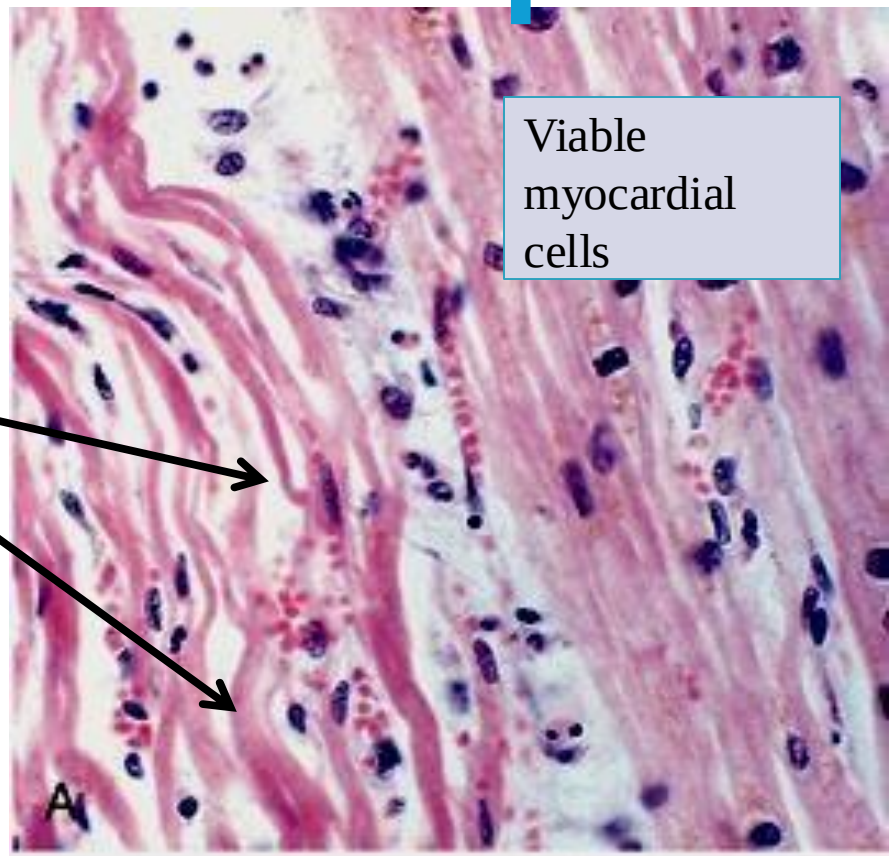
microscopic feature that occurs within the first 24 hours following the onset of coronary occlusion (<24 hrs) is **coagulative necrosis** of myocardial muscles, the necrosis will **lead to wavy fibers** which are necrotic and wavy cardiac muscle, also necrosis will be **associated with edema** which separates necrotic cells from each other.

Stain: Hematoxylin & Eosin (H&E)

<24 hr:

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

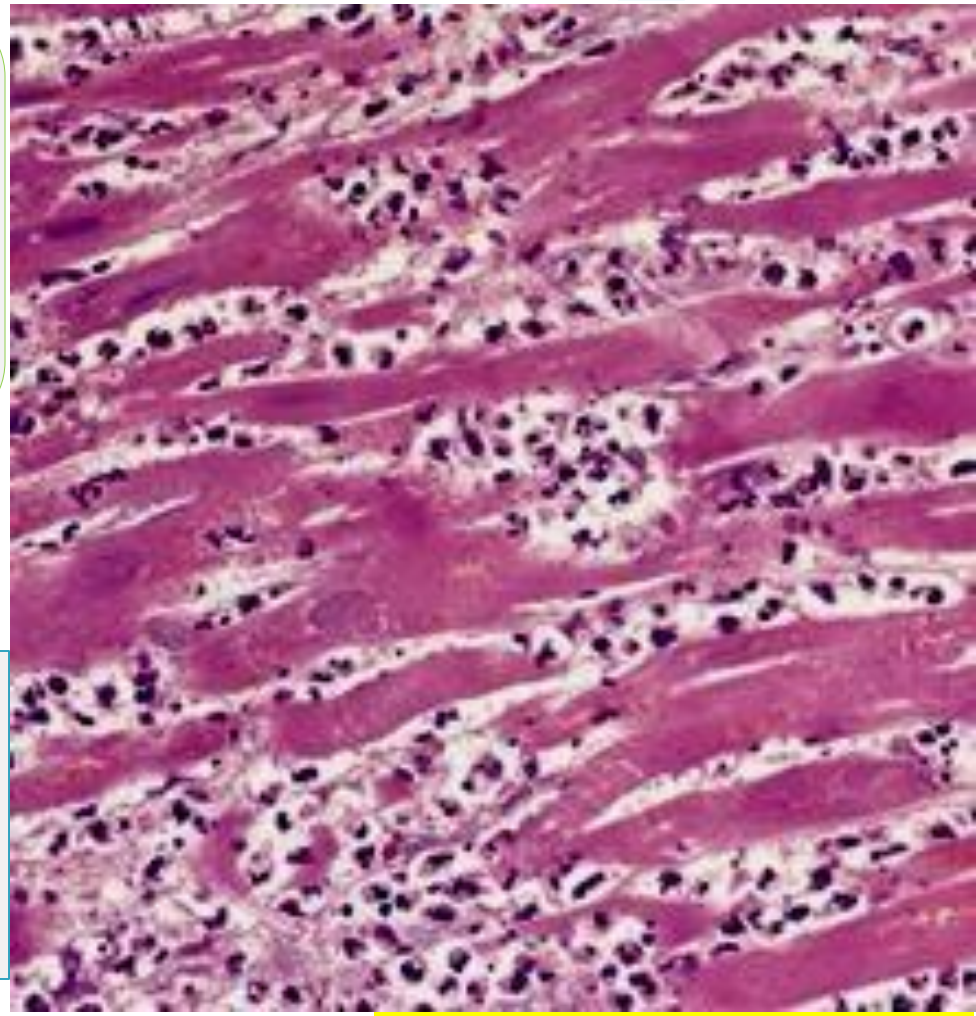
1



Microscopic features of myocardial infarction and its repair.

2

Second phase of MI evolution happens during the 2-3 days following the onset of the infarction, dense neutrophilic infiltrate would replace the edema and necrotic tissue , because necrosis is always associated with inflammation and the first part of inflammation would be acute inflammation and neutrophils would be recruited.



Stain: Hematoxylin & Eosin (H&E)

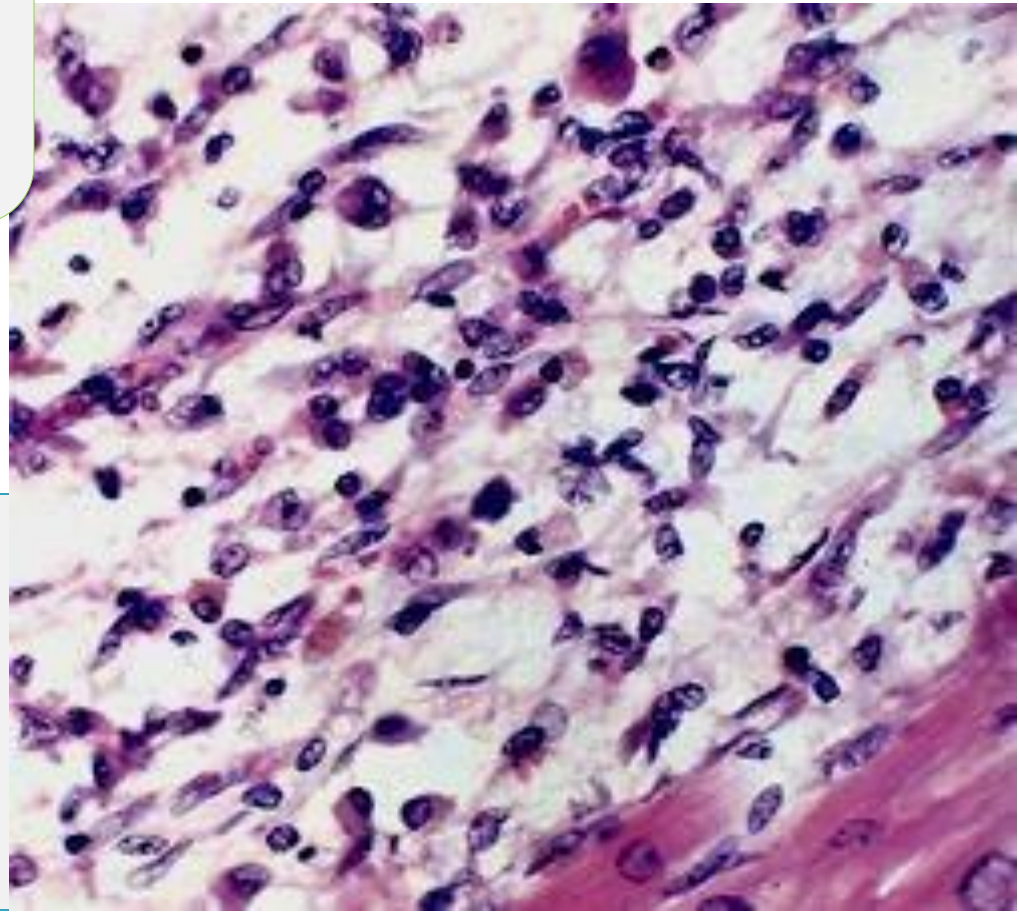
2 - 3 days:

Dense **neutrophil** infiltrate

Microscopic features of myocardial infarction and its repair.

7 to 10 days following the onset of MI , the necrotic area will be filled with another inflammatory cells which are macrophages , and during this time, there will be complete removal of necrotic tissues by those macrophages, so this is a different phase of inflammation that happens following tissue necrosis.

3



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

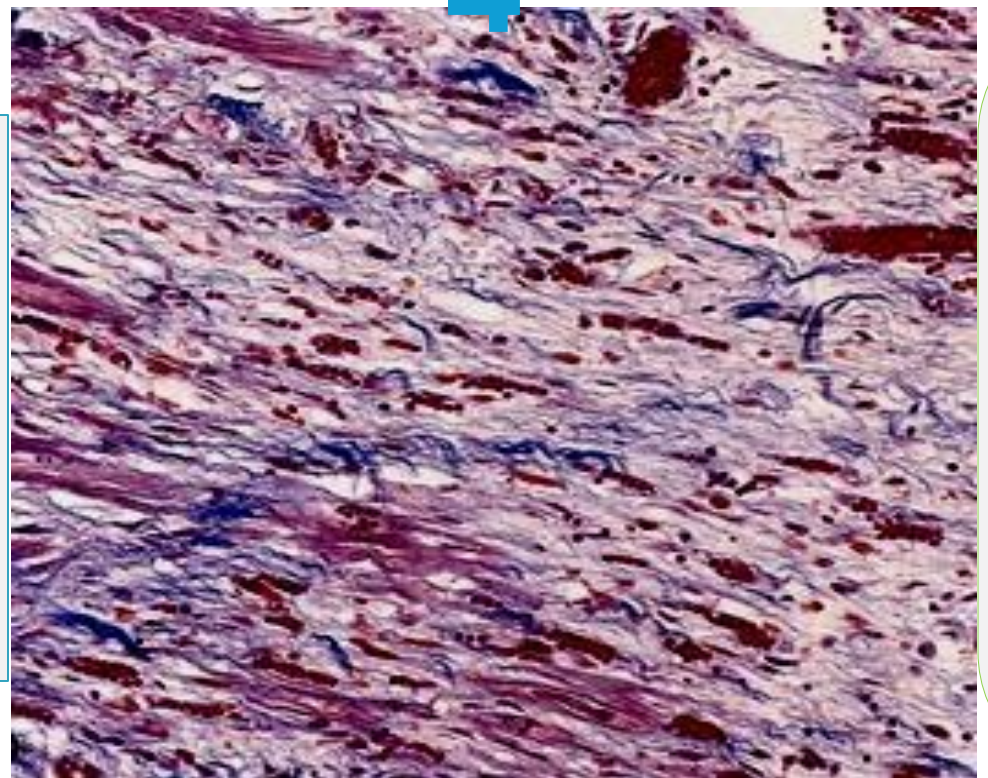
Stain: Hematoxylin & Eosin (H&E)

Microscopic features of myocardial infarction and its repair.

Repair phase: Within the **14 days following MI** and actually at day 14, there will be **Granulation tissue formation** and will peak 2 weeks after MI, this phase always end with a scar or connective tissue fibrosis
Let's sum up: We started with cell damage or necrosis then two phases of inflammation then the third important phase which is tissue repair.

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

4



The **blue color** is a granulation tissue which is a loose connective tissue or collagen fibers.

- The red color shows a lot of blood vessels.

This tissue is weak and does not have the same strength as the original myocardium.

Stain: Masson Trichrome (MT)

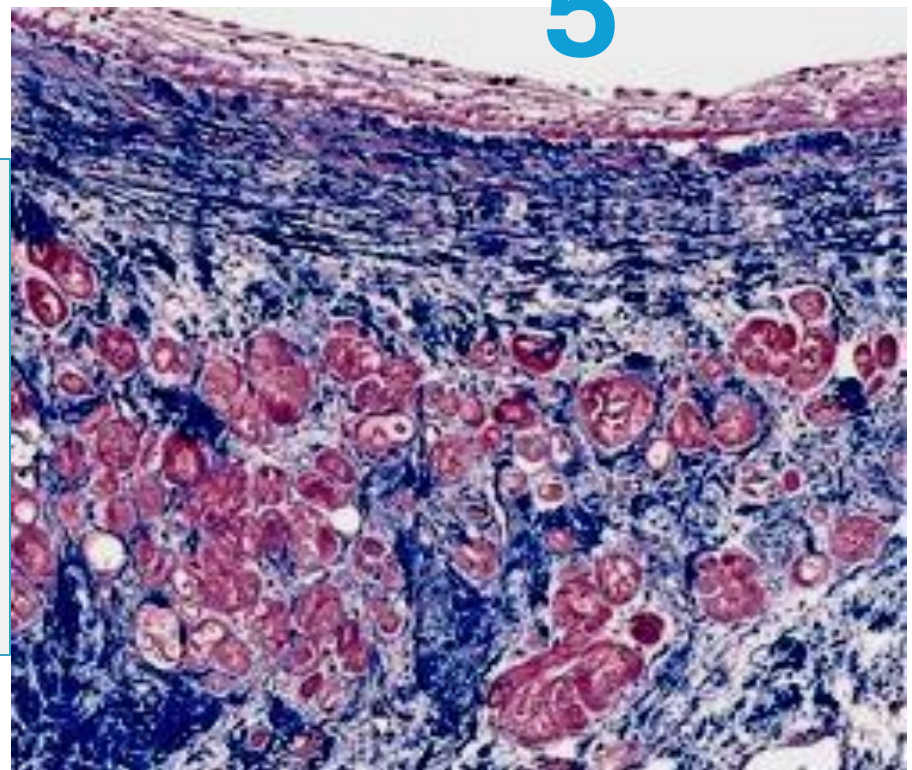
Microscopic features of myocardial infarction and its repair.

The last phase is the development of dense collagenous scar, this occurs several weeks after the onset of MI. At least 6 weeks are needed to develop a strong, dense scar that replaces the non-viable myocardium. Athough this tissue is strong, but it is not as strong as the original myocardial muscles, it doesn't have the same contractile ability as the myocardial muscles, so it will always be abnormal.

5

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

Stain: Masson Trichrome (MT)



Blue color is the scar that has developed inside the necrotic area of myocardium.

The red color is the remaining viable myocardial cells.

Stain: Masson Trichrome (MT)

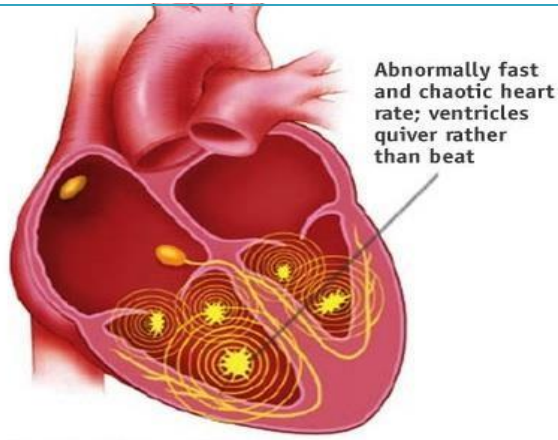
Consequences & Complications of MI

1- Death: The most important and feared consequence

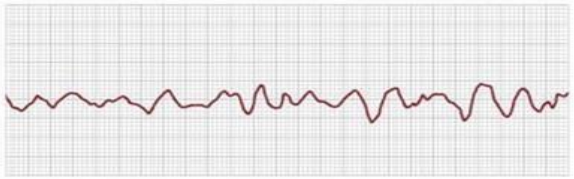
- **50%** occur before reaching hospital (within 1 hour of symptom onset-usually as a result of lethal arrhythmias (Sudden Cardiac Death)

- **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).

This is an example of lethal arrythmias that usually lead to death or acute MI, and it is called "**ventricular fibrillation**". So, there is abnormally fast and chaotic (فوضوي) heart rate and ventricles quiver (يرتعش) rather than beat appropriately.



Ventricular Fibrillation ECG



Consequences & Complications of MI

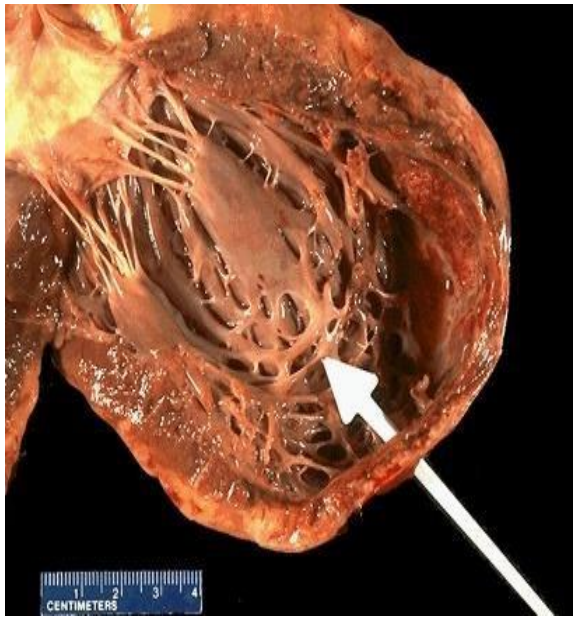
- **2- Cardiogenic shock.**
- occurs in 15% of large infarcts, involve >40% of Left ventricle and result in pump failure of the heart.
- - it is significant and dangerous due to 70% mortality rate and it is an **important cause of in hospital deaths.**
- **3-Myocardial rupture**
- **4-Pericarditis**
- **5-Infarct expansion**
- **6- Mural thrombus**
- **7-Ventricular aneurysm**
- **8-Progressive late heart failure**

Complications of Myocardial Rupture Include:

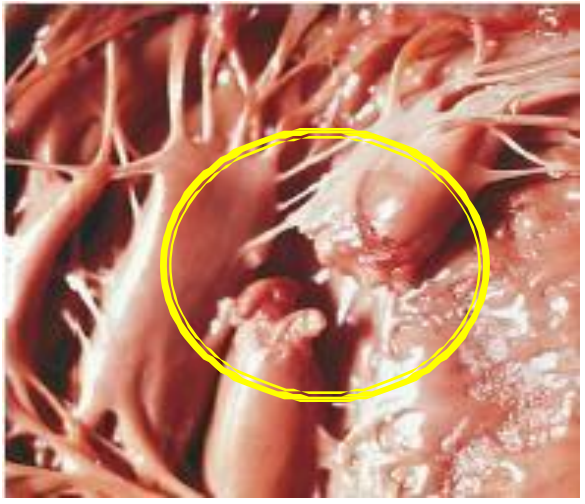
(1)rupture of the ventricular free wall leads to hemopericardium and cardiac tamponade (usually fatal)

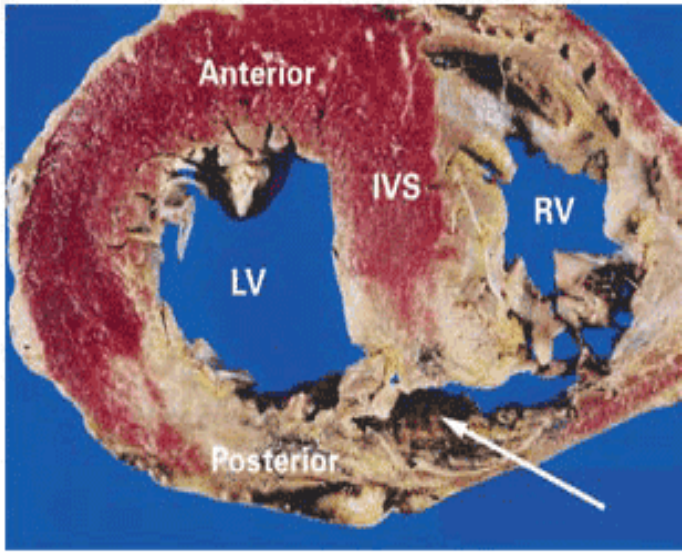
(2)rupture of the ventricular septum leads to VSD and left-to- right shunt

(3)papillary muscle rupture leads to severe mitral or tricuspid regurgitation



The consequences of myocardial rupture depends on where exactly the rupture has developed





4-Pericarditis.

- immunologic condition that develops 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 inside the damaged chamber(causing stasis which is one of the factors in Virchow's triad) + **endocardial damage** associated with ischemia **=** will together lead to **thromboembolism.**

7-Ventricular aneurysm.

- A late complication, needs several weeks to happen because it needs to have scar tissue to develop.

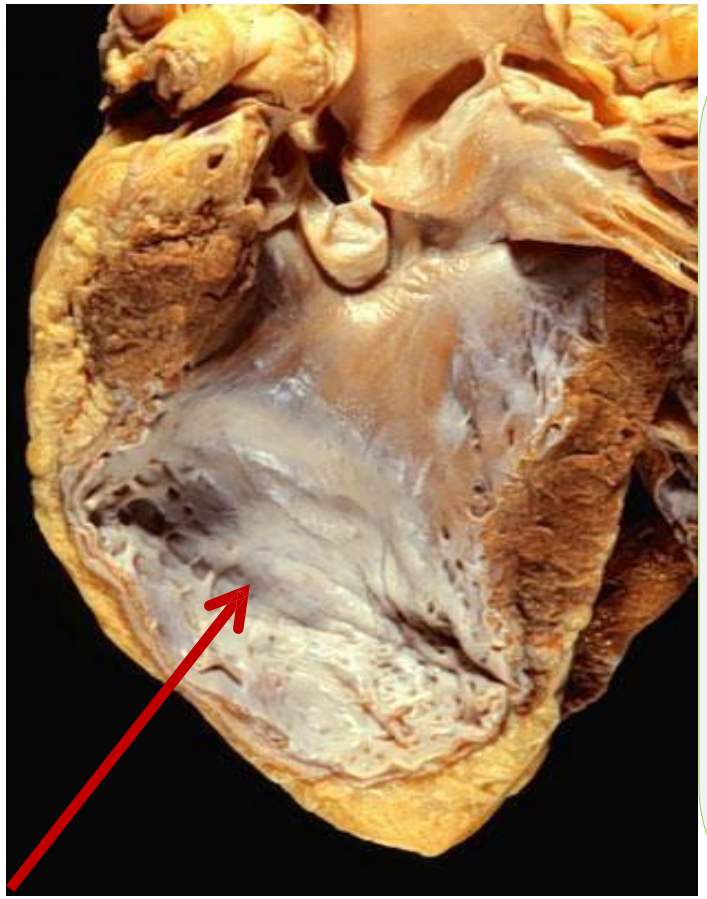
- most commonly result from a large transmural anteroseptal infarct that heals with the formation of thin scar tissue

Complications of ventricular aneurysms include:

1-mural thrombus inside the affected chamber.

2-arrhythmias because this scar has abnormal conduction process inside it.

3-heart failure



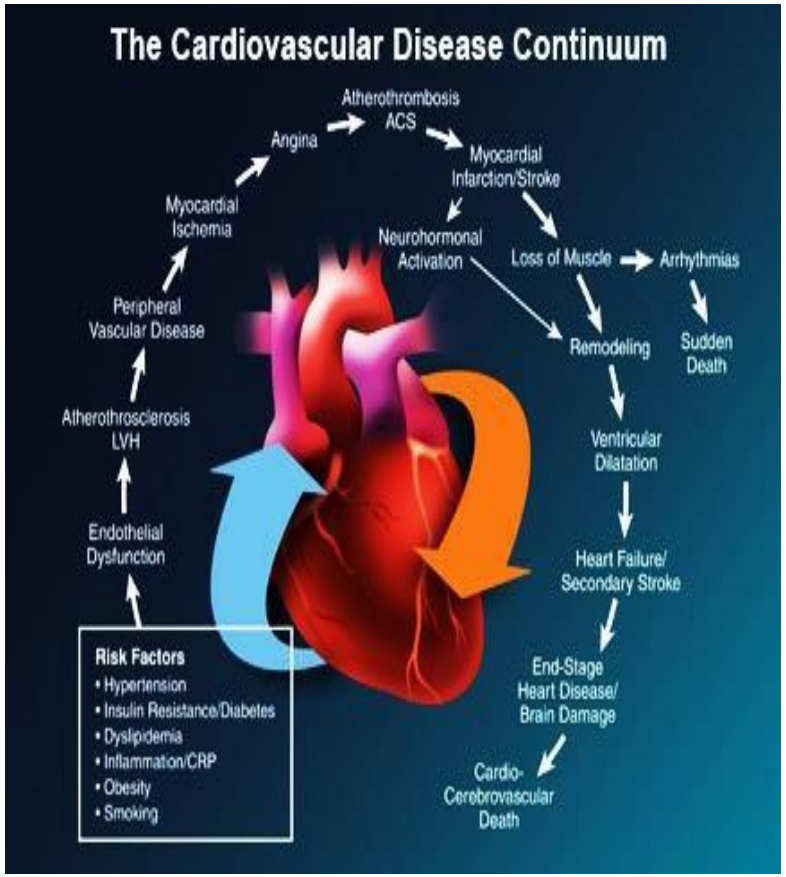
As you can see, the tissue with red arrow has a very thin scar tissue in comparison with the remaining viable myocardium in the upper part of the picture, This thin scar is quite fragile and lacks the contractile ability of the healthy myocardium, so this area is prone to develop aneurysm.

Aneurysm is an abnormal, prominent dilatation especially during cardiac contraction (during systole).

Long-term prognosis after MI

- - depends on many factors like the remaining left ventricular function and the severity of atherosclerosis in the remaining viable myocardium, etc .
- -The highest mortality rate following acute MI is during the first year =30%.
- Thereafter, the annual mortality rate is always constant and much less than before =3%

Chronic Ischemic Heart Disease

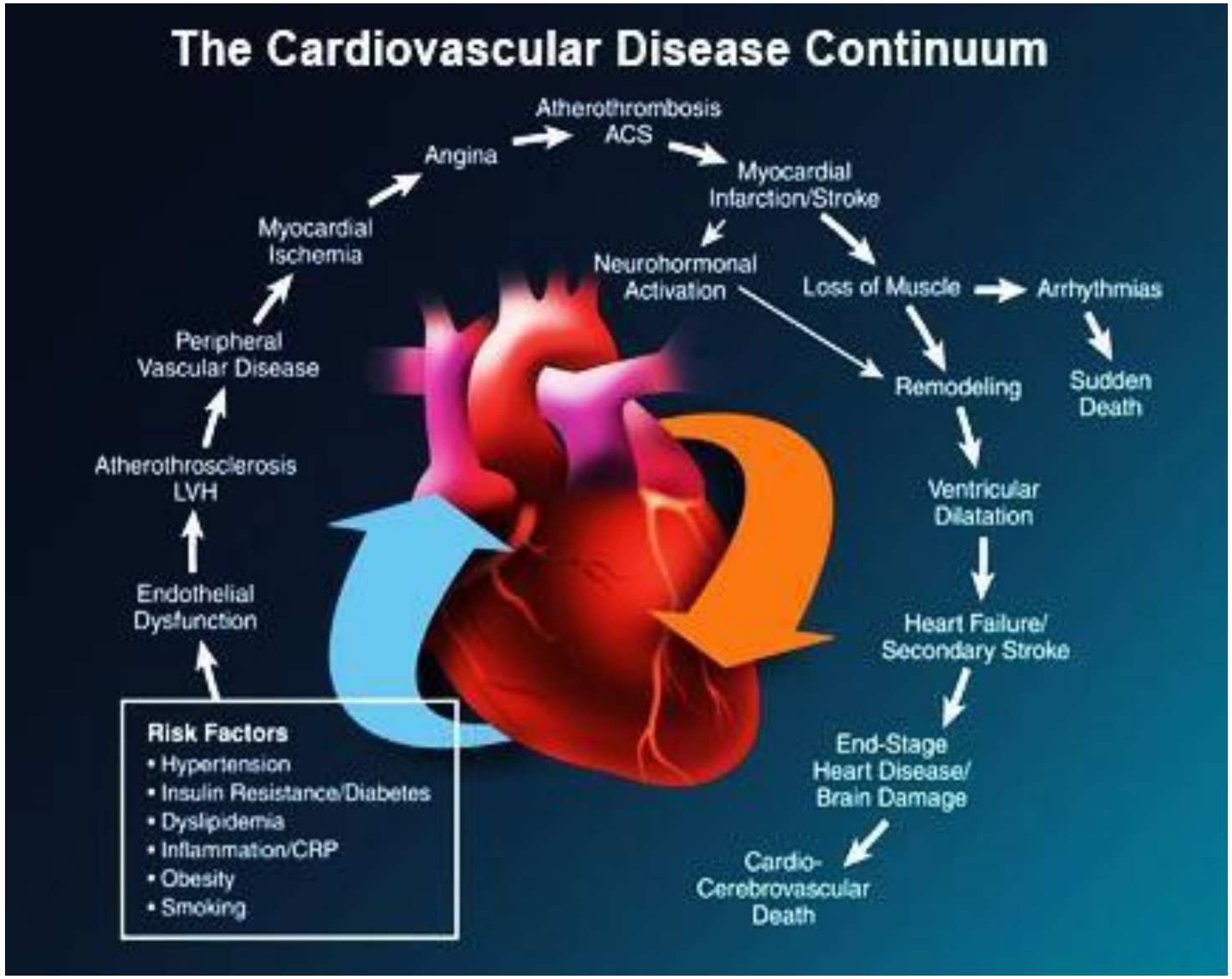


- results from **post- infarction** cardiac decompensation that follows exhaustion of hypertrophic viable myocardium.
- **progressive heart failure that is** sometimes punctuated by episodes of angina or MI
- Arrhythmias are common

As you can see here, cardiovascular disease has a continuum, it starts with endothelial dysfunction, atherosclerosis, hypertension or peripheral vascular disease, all of these can lead to myocardial ischemia.

Remember :**the first clinical syndrome of myocardial ischemia is angina** and with more and more ischemia there will be **MI** ,then following acute MI , in a patient who survives there will be some kind of remodelling and ventricular dilatation that will lead to progressive heart failure that is called "**Chronic Ischemic Heart Disease**"

Chronic Ischemic Heart Disease



Sudden Cardiac Death (SCD)

- **Unexpected death from cardiac causes either without symptoms or** within a short period after symptoms onset (**< 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 ……

These can be seen in young patients, athletes or people who have certain hereditary or acquired abnormalities in cardiac conduction system or cardiac muscle.

Occlusion of the Right circumflex coronary artery is responsible for the majority of acute myocardial infarctions.

- ○ True
- ⊙ False ✔

You got 1 of 1 points

1/1

▶ Continue

The best cardiac enzymes in the evaluation of acute myocardial infarction are:

- ✔ Troponins
- Creatine Kinases
- LDH
- Myoglobins

ONE match is FALSE regarding the histological findings and the corresponding time frame following acute myocardial infarction:

- ✔ Wavy fibers: 6 weeks
- Macrophages: 7-10 days
- Granulation tissue: 2 weeks
- Neutrophils: 2-3 days

Additional sources

اللهُمّ هدأت أنفاس النّاس وأظلَم اللّيل، وهناك قلوب لا تنام! فاكتب النَّصر والثّبات والتّمكين يا ربّ، لإخواننا في غزّة وسوريّا والسّودان ولبنان، وكلّ مكانٍ يُجاهِد صدقًا في سبيلك، اكتب لهم نصرًا وأجرًا، وثباتًا في كُلّ ميدان، يا ربَّنا.

VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2			
V2→V3			

امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!