

Acute Myocardial Infarction (MI)

Clinical Features of acute MI

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graph TD; A[Clinical Features of acute MI] --- B[Dyspnea (if pulmonary congestion and edema)]; A --- C[Cardiogenic shock (in massive MI >40% of left ventricle)]; A --- D[Sometimes no typical symptoms (silent infarcts)]; A --- E[Severe, crushing substernal chest pain that radiates to neck, jaw, epigastrium, or left arm]; A --- F[Dizziness; sweating]; A --- G[Rapid and weak pulse]; A --- H[Nausea (in posterior MI)]; D --> I[Silent infarct: <br/> * Asymtomatic pateints <br/> * Confirmed only on ECG and lab <br/> * Particularly in: DM and erderly];
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Dyspnea (if pulmonary congestion and edema)

Cardiogenic shock (in massive MI >40% of left ventricle)

Sometimes no typical symptoms (silent infarcts)

Silent infarct:

- * Asymtomatic pateints
- * Confirmed only on ECG and lab
- * Particularly in: DM and erderly

Severe, crushing substernal chest pain that radiates to neck, jaw, epigastrium, or left arm

Dizziness; sweating

Rapid and weak pulse

Nausea (in posterior MI)

Evaluation of MI

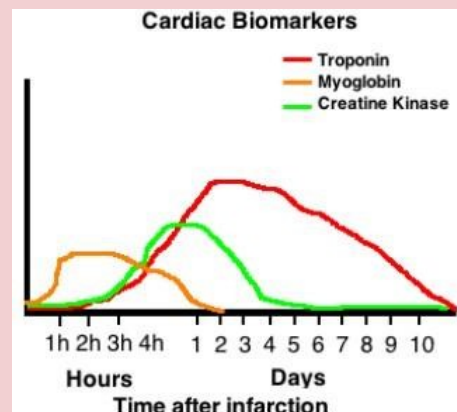
Clinical signs & symptoms

ECG abnormalities

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

Such as search about cardiac enzyme in MI

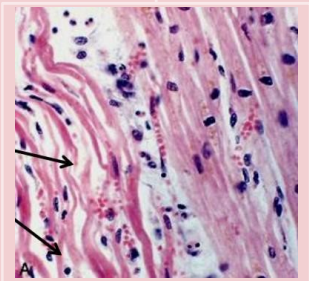
1. Myoglobin
2. Cardiac Troponins T and I (TnT, TnI) --- the best marker
3. Creatine kinase (CK); specifically the myocardial-specific isoform (CK-MB) --- the second best marker
4. Lactate dehydrogenase



Microscopic features of myocardial infarction and its repair

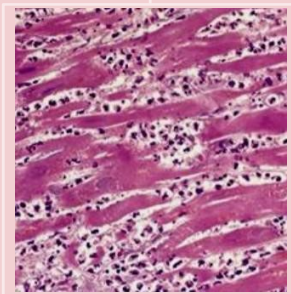
<24 hr:

coagulative necrosis and wavy fibers
Necrotic cells are separated by edema fluid.
Stain: H&E



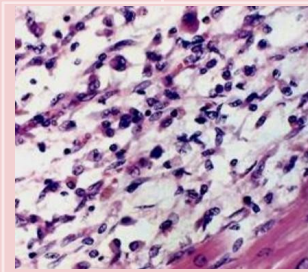
2 - 3 days:

Dense neutrophil infiltrate.
Stain: H&E



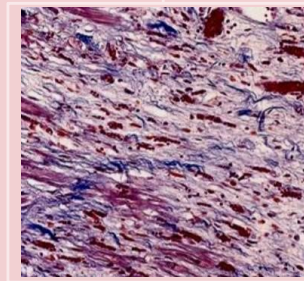
7 to 10 days:

complete removal of necrotic myocytes by macrophages.
Stain: H&E



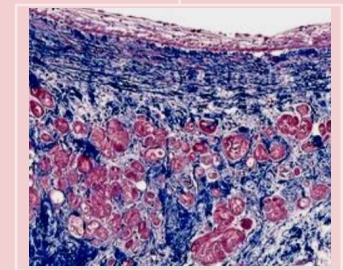
up to 14 days:

Granulation tissue [loose connective tissue (blue) and abundant capillaries (red)].
Stain: Masson Trichrome (MT)



several weeks:

Healed infarct consisting of a dense collagenous scar (blue).
Stain: Masson Trichrome (MT)



Consequences & Complications of MI

Myocardial Rupture, it's complication:

rupture of the ventricular free wall:
Hemopericardium and cardiac tamponade
(usually fatal)

rupture of the ventricular septum:
VSD and left-to-right shunt

papillary muscle rupture:
severe mitral regurgitation

Pericarditis

2-3 days after transmural MI, spontaneous healing

Mural thrombus

loss of contractility (causing stasis) +
endocardial damage = thromboembolism
It's complication:

mural thrombus

arrhythmias

heart failure

Death

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

Cardiogenic shock:

- In large infarcts (>40% of Left ventricle)
- 70% mortality rate (important cause of in hospital deaths).

Progressive late heart failure

Infarct expansion

disproportionate stretching, thinning, and dilation of the infarct region (especially with anteroseptal infarcts)

Ventricular aneurysm

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

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

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 arrhythmias (v. fibrillation) is the most common direct mechanism of death

NOTE:

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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