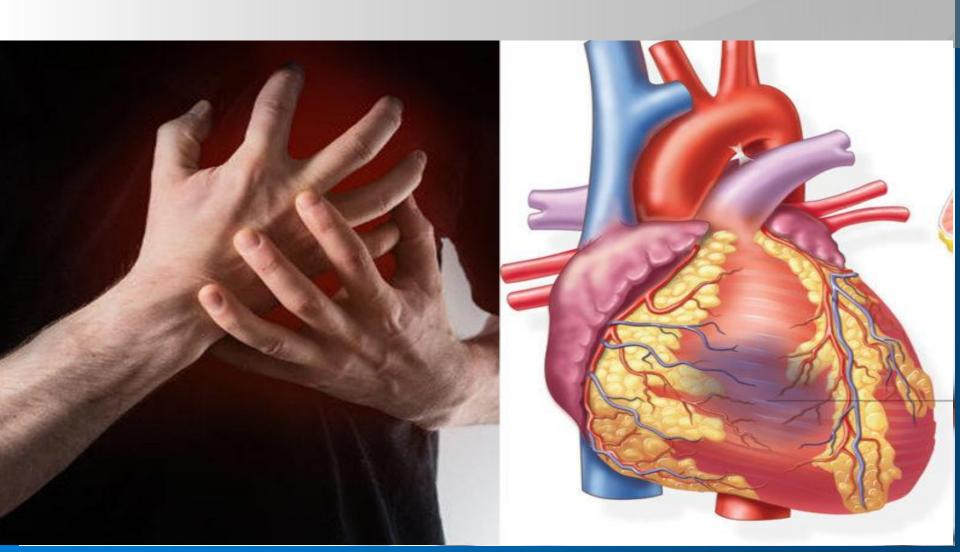
Myocardial Infarction

BY Dr.Maha Alsadik



Definition

 Ischemic necrosis of part of the cardiac muscle due to sudden, persistent & complete cessation of its blood supply.



Etiology:

1. Thrombosis on top of atherosclerosis.

(The most common cause)

2. Non-atherosclerotic causes of myocardial infarction:

Coronary artery diseases: congenital anomalies, spasm, dissection, PAN, Takayasu's disease.

Aortic stenosis, regurge.

Embolism: IE, Artificial valve, myxoma.

Risk factors for Atherosclerosis

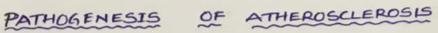
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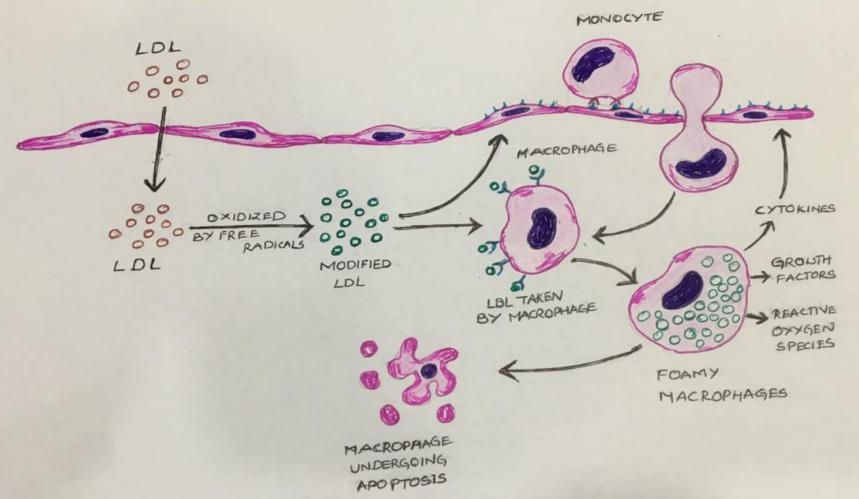
- Age.
- Sex: male> female.
- +ve family history.

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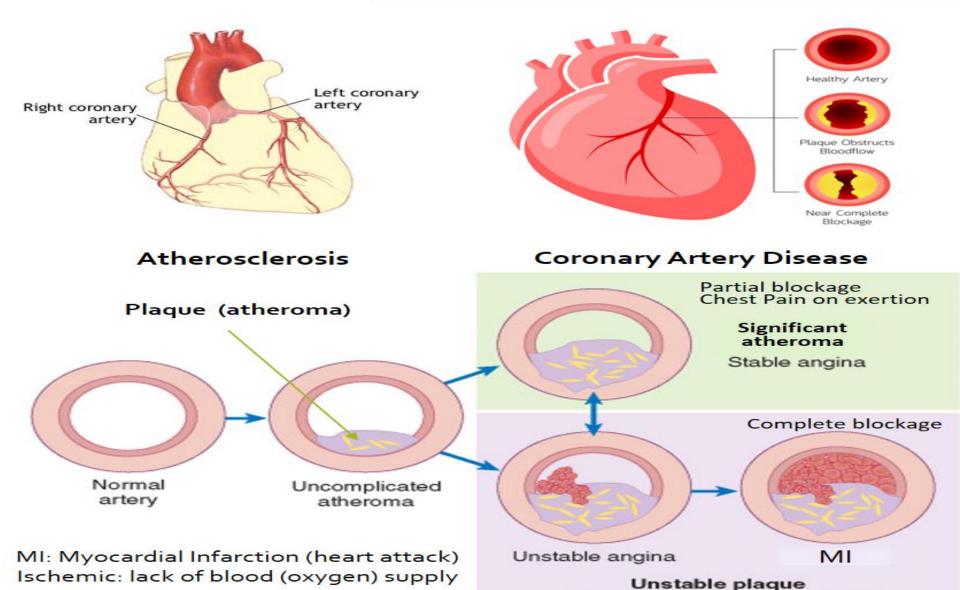
- Hypertension: cause endothelial damage.
- Hyperglycemia.
- Hyperlipidemia especially LDL .
- Hyperuricemia .
- Sedentary life style.
- Smoking .
- Stress.

pathogenesis





pathogenesis



Acute coronary syndrome (ACS)

Classification

- Site:
- 1- Occlusion of the left anterior descending artery
- \rightarrow (anterior infarction).
- 2- Occlusion of the circumflex artery→ (lateral infarction).
- 3- Occlusion of the right coronary artery→ (inferior infarction).
- Types:
- Transmural infarction (ST elevation myocardial infarction - STEMI): infarction of full thickness of the ventricular wall.
- Subendocardial infarction (Non ST elevation myocardial infarction -NSTEMI): Transient or incomplete vessel occlusion.

clinical picture

Pain and/or complications

- I. Chest pain: Similar to angina but:
- More severe, it may be severe enough to be described as the worst pain the patient has ever felt
- Radiates more: may below epigastric area but never below umbilicus.
- More prolonged : up to several hours.
- Unrelated to precipitating factors: may at rest.
- Not relieved by rest or sublingual nitrate.
- Associations: sweating, dizziness, dyspnea, fear of death (angor animi) & may also associated with complications.

- Painless infarction:
- ✓ Elderly.
- ✓ Diabetic neuropathy.
- ✓ Patient under anesthesia.
- √ Transplanted heart (denervated).

II. Complications

early complications:

- 1- Shock:
- cardiogenic shock :Caused by massive infarction (> 40% of the cardiac muscle) leading to severe pump failure& high jugular venous pressure.

C/P: Hypotension, tachycardia ,pulmonary edema.

Prognosis: very bad.

> Neurogenic shock

Caused by severe pain (vagal stimulation).

- C/P: Hypotension, bradycardia.
- Prognosis: good.
- 2- Acute heart failure: with normal heart size (within 24hs).
- 3. Arrhythmia(within 24hs) :All types may occur.

The most serious are: VT, CHB.

4- Myocardial rupture:

- ➤ Rupture of the septum →acquired VSD.
- ➤ Rupture of papillary muscles →acute MR → acute heart failure.
- ➤ Rupture of the ventricular free wall →blood fills the pericardium→ cardiac tamponade.
- 5- Dry pericarditis: (within 1-3 days)

Hemorrhagic pericardial effusion may develop especially with thrombolytic therapy.



6- Sudden death:

- ➤ Arrhythmia (VT, VF): most deaths occur during few hours after MI.
- > Acute heart failure.
- Cardiogenic shock.
- Cardiac rupture.



Late complications:

 1- Post infarction syndrome: (Dresslers syndrome) within 4 weeks or more

Autoimmune phenomenon in response to necrotic cardiac tissue characterized by:

Pericarditis - Pleurisy - Pneumonitis -fever.

- 2- Post infarction angina :Due to affection of other diseased coronaries.
- 3- Myocardial aneurysm: (dilatation of the scar tissue of MI)
- ➤ On examination: double apex.
- ECG: persistent ST segment elevation.

4- Thrombo-embolism :

Mural thrombosis :(infarction→ rough surface→thrombosis→systemic emboli)

DVT : due to prolonged recumbency →pulmonary embolism .

 5- Complications of treatment: anticoagulant, prolonged bed rest,

signs

(not specific) nothing or anything

- The physical examination may be entirely normal.
- Pallor, sweating, nausea, vomiting & fever.
- Pulse:
- ➤ Tachycardia: sympathetic stimulation, cardiogenic shock.
- > Bradycardia: neurogenic shock, HB, inferior MI.
- > Irregular : arrhythmias.
- Blood pressure :
- > Hypertension : sympathetic stimulation .
- > Hypotension : LVF, shock.

Cardiac auscultation :

- ➤S1: weak.
- >S2: reversed splitting.
- >S3: due to LVF.
- ➤ S4 : due to decreased myocardial compliance.
- ➤ Murmur : of MR, VSD .
- > Pericardial rub : Dry pericarditis.

Differential Diagnosis

causes of acute chest pain:

- Stable angina.
- ➤ Unstable angina.
- > MI.
- > Pulmonary embolism.
- > Aortic dissection.
- > Pneumothorax.
- >Acute dry pericarditis.

Diagnosis of MI

At least 2 of the following 3 criteria:

- 1. Classic chest pain.
- 2. ECG changes.
- 3. Positive biomarkers (cardiac enzymes)

Investigations

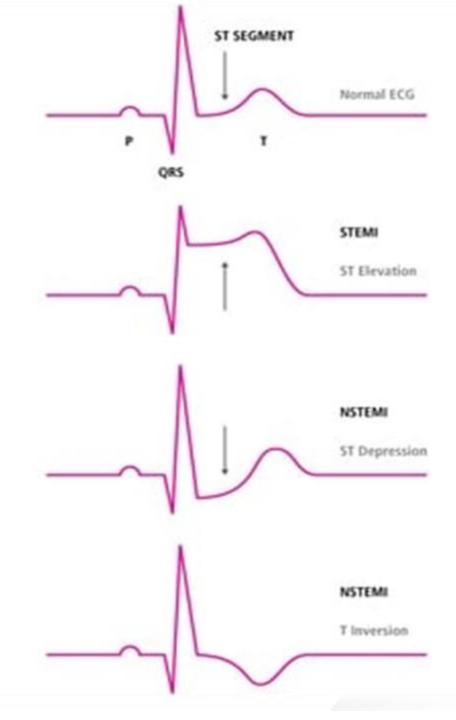
1- ECG:

- > In transmural infarction (ST Elevation MI):
- 1. Convex elevation of ST segment.
- 2. Twave: Tall (hyperacute) in the first few minutes after vessel occlusion (the earliest change)
- <u>later on</u>: Inverted T wave (representing sever ischemia)
- 3. <u>Finally</u>, pathological Q waves occur, representing significant myocardial necrosis -& replacement by scar tissue.
- (Pathologic Q waves are usually defined as duration ≥ 0.04 s of >25% of R-wave amplitude)

- In subendocardial infarction (Non ST Elevation MI):
- 1. ST segment: normal or depressed.
- 2. No pathological Q waves (non Q wave MI)
- 3. T wave: inverted.

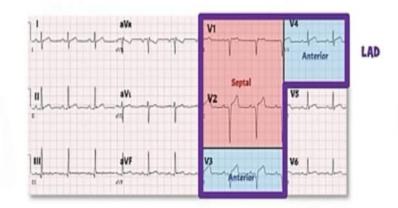
In old MI: The only residual change is the pathological Q wave.

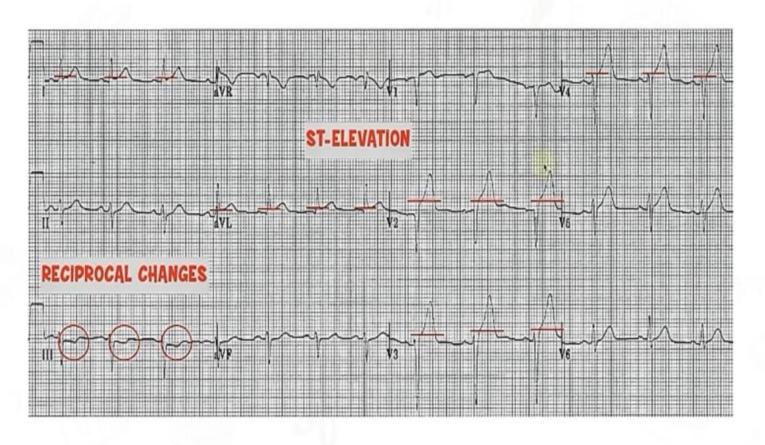


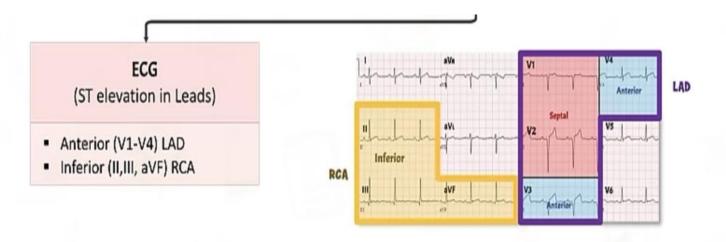


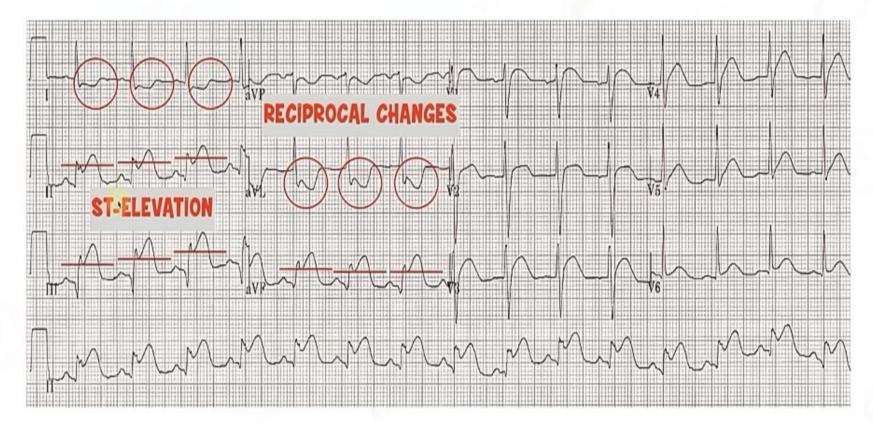
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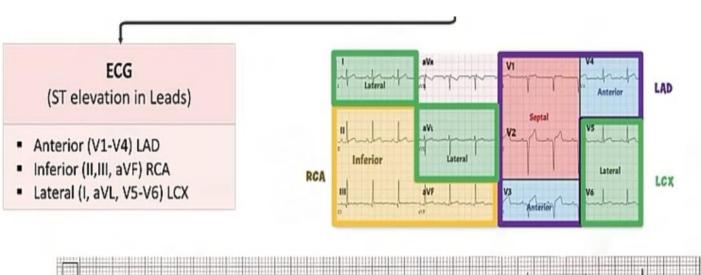


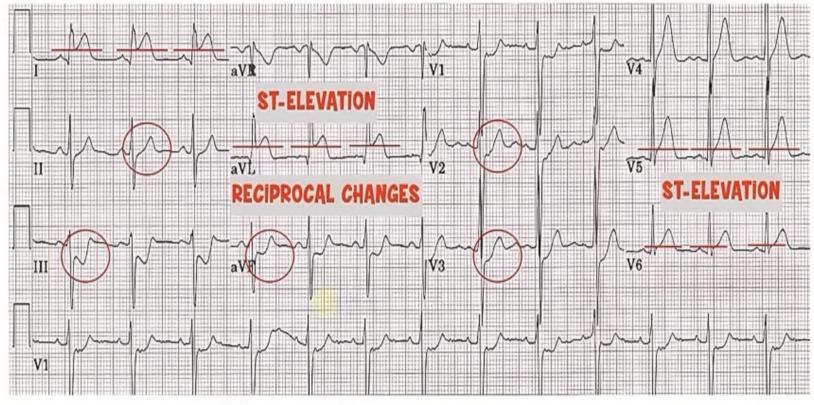












2. Cardiac enzymes are released into blood from necrotic heart muscle after an acute MI.

Marker	Initial rise	Return to normal	Notes
Creatine phosphokinase (CPK)	4-8 h	2-4 days	Non specific because it may rise in damaged skeletal muscles or brain.
CPK-MB	4-8 h	2-4 days	It's isoenzyme of CPK, specific to cardiac muscle
Lactic dehydrogenase (LDH)	10 h	1-2 weeks	Not specific .
Troponin (cTnT,cTnl)	3-12 h	1 week	Most sensitive & specific markers of myocardial damage .
Myoglobin	1-4 h	24 h	

3- Echocardiography :

Ventricular wall motion abnormalities.

Complications: MR, myocardial aneurysm.

- 4- Cardiac scan: (Radioactive Thallium 201)
 Thallium 201: is taken up by healthy myocardium & not by ischemic myocardium (cold spot)
- 5- Coronary angiography (coronary catheter): reveals which vessels have been affected and the extent of damage.

 6- Leukocytosis , ↑ ESR: as there is tissue damage.

Treatment

- Rapid transfer to hospital is a must (Time lost is lives lost).
- > Hospital care:
- 1- General:
- a. Admission to CCU (coronary care unit) with hemodynamic monitoring & continuous ECG
- b. Oxygen inhalation.
- c. Complete rest.
- d. Diet: Light frequent meals & avoid constipation

e. Sedative: Diazepam.

f. Aspirin: is now considered an essential element (325 mg initial dose then 75 mg daily-oral)

g. ACE Inhibitor: Oral therapy e.g. Lisinopril 5mg on dayl & 2, then 10 mg daily.

- ACE Inhibitors are vasodilator that reduce cardiac work & decrease myocardial energy requirement.
- ACE Inhibitors also have inhibitory effect on the cardiac remodeling

2- Relieving of chest pain:

- a. Morphine (4 mg IV every 5 to 10 minutes as needed)
- b. Nitroglycerine.
- c. B blockers.

3- Thrombolvtic therapy:

 The earlier that thrombolytic therapy is given after the onset of chest pain, the greater the benefit (thrombolytic therapy is beneficial up to 6 hours but may be given for up to 12 hours)

Drugs:

- Streptokinase: 1.5 million units IV 9ver 60 min. may cause allergy.
- Alteplase, tenecteplase (tissue plasminogen activator - tPA)



- Anticoagulant (heparin) & antiplatetelet (aspirin) are given with & after thrombolytic therapy to reduce the risk of reocclusion.
- Contraindications: the major risk is Bleeding
- > Bleeding disorders.
- Major surgery within past 2 weeks.
- > Recent cerebral hemorrhage within past 12 months.
- > Active internal bleeding e.g. peptic ulcer.
- > Severe hypertension.
- > Diabetic retinopathy with recent bleed.
- > Aortic dissection.

4- Angioplasty: Percutaneous Transluminal Coronary Angioplasty (PTCA)

 Introduction of balloon or stent to dilate the stenotic artery (balloon-tipped catheter)

More effective than thrombolytic therapy (fewer complication, shorter hospitalization).

5. CABG (Coronary Artery Bypass Graft):

Grafting a piece of saphenous vein or internal mammary artery between the aorta & the coronary artery distal to any obstruction.

Indication of CABG:

- Stenosis of 3 or more vessels.
- Stenosis of left main coronary artery.
- ► For diabetic patients with 2 or 3 vessel disease.



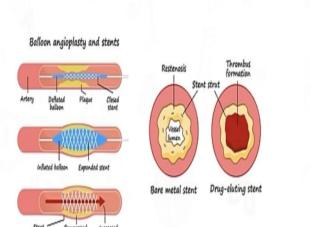
6- Treatment of early complications: e.g.

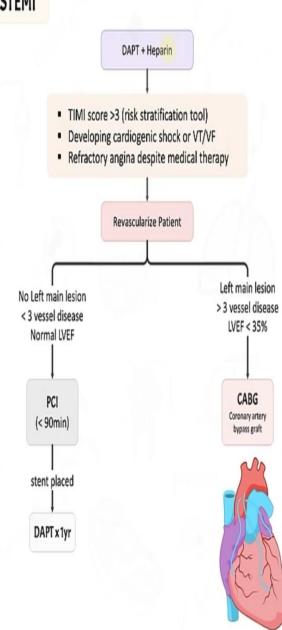
- Acute heart failure.
- Arrhythmia.
- cardiogenic shock



Treatment of Unstable Angina/NSTEMI

Туре	Purpose of Treatment	Treatment
Unstable Angina & NSTEMI	Prevent thrombus propagation	- ASA PLUS - Clopidogrel PLUS - Heparin
	Reduction of Anginal chest pain	- Nitroglycerine - Beta Blocker - Morphine
	Revascularization of coronary vessel	- PCI or CABG
	Prevent stent thrombosis	- DAPT x 1 year
	Prevent coronary plaque progression	- Statins





TIMI Risk Score for NSTEMI/UA

Age ≥ 65 years

≥ 3 Traditional CAD risk factors

CAD with ≥ 50% diameter stenosis

≥ 2 Anginal episodes in last 24 hours

ECG ST-segment deviation of ≥ 0.5 mr

Aspirin use in the past 7 days Elevated cardiac biomarkers

Score Risk of death/MI/Revasc

0-1 points - 5% 2 points - 8%

3 points - 13%

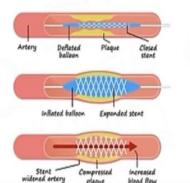
4 points - 20%

5 points - 26% 6 points - 41%

Treatment of **STEMI**

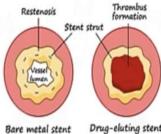
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	Revascularization of coronary vessel	- PCI or CABG or tP
	Prevent stent thrombosis	- DAPT x 1 year
	Prevent ventricular remodeling	- Ace-Inhibitors
	Prevent coronary plaque progression	- Statins

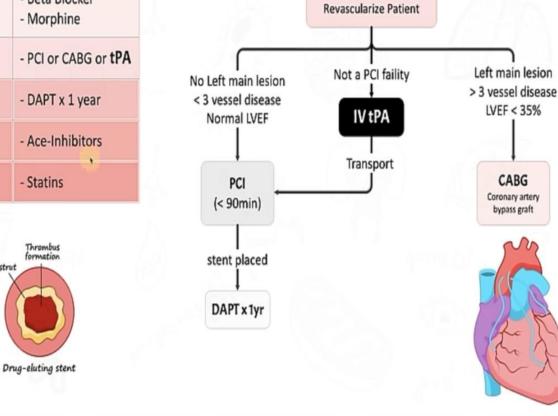
Balloon angioplasty and stents



plaque

blood flow





DAPT + Heparin

All STEMIs require revascularization

DATE OF THE PARTY OF THE PARTY

.After discharge :(ABCDE)

A: Aspirin. ACEIs.

B: B blockers., BP control.

C: Cholesterol control.

D: Diabetes control. diet.

E: Education., reassurance & rehabilitation



