Myocardial Infarction

BY

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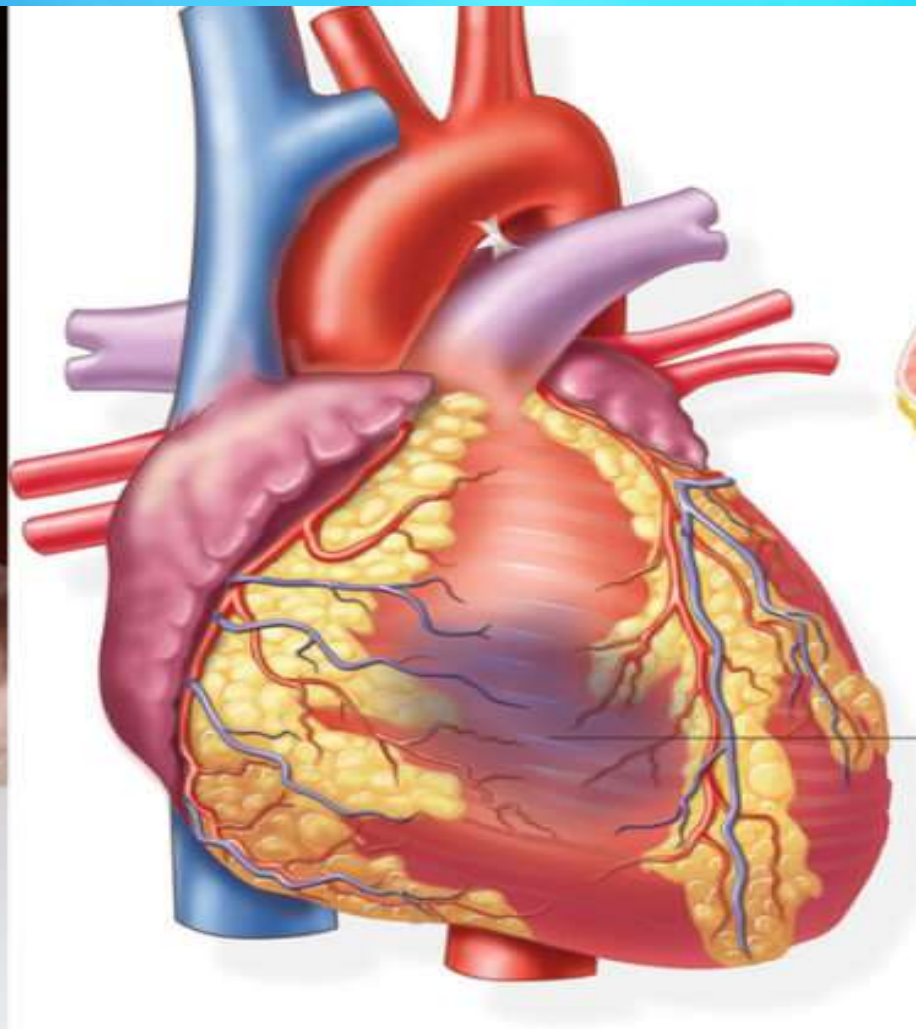


Definition

- 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

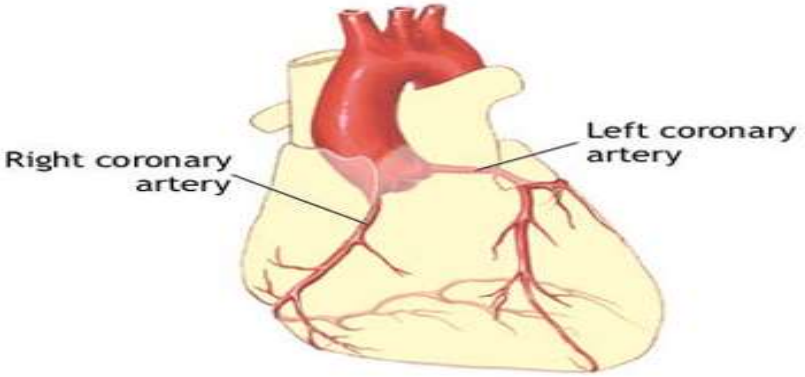
Non modifiable:

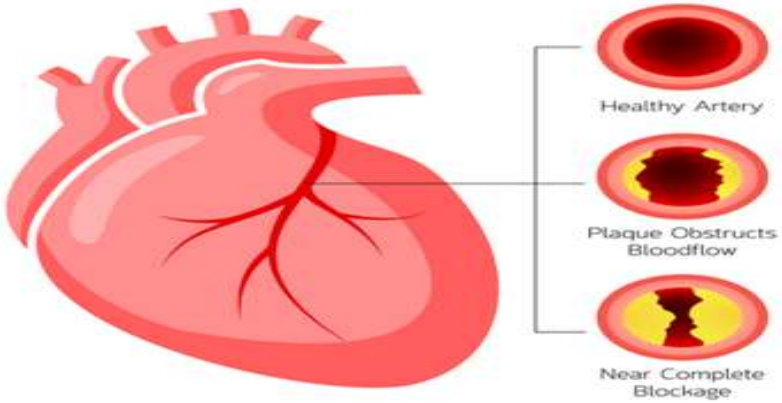
- Age.
- Sex: male> female.
- +ve family history.

Modifiable :

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

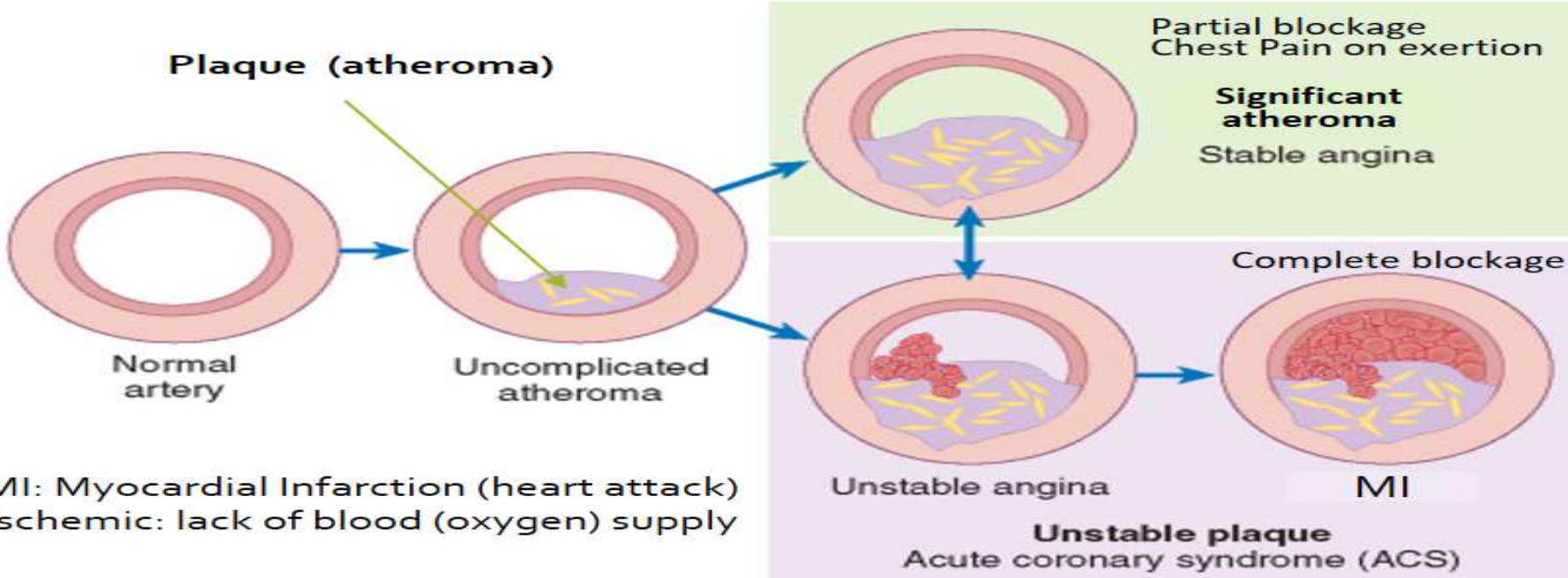
pathogenesis





Atherosclerosis

Coronary Artery Disease



MI: Myocardial Infarction (heart attack)
Ischemic: lack of blood (oxygen) supply

Classification

- Site:

1- Occlusion of the left anterior descending artery →(anterior infarction).

2- Occlusion of the circumflex artery→ (lateral infarction).

3- Occlusion of the right coronary artery→ (inferior infarction).

- Types:
- Transmural infarction (**ST e**levation **m**yocardial **i**nfarction - **STEMI**) : infarction of full thickness of the ventricular wall.
- Subendocardial infarction(**N**on **ST e**levation **m**yocardial **i**nfarction **-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 Ml.
- 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 Ml)

➢ 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 Ml.

➢ 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.
- Ml.
- 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)

later on : Inverted T wave (representing sever ischemia)

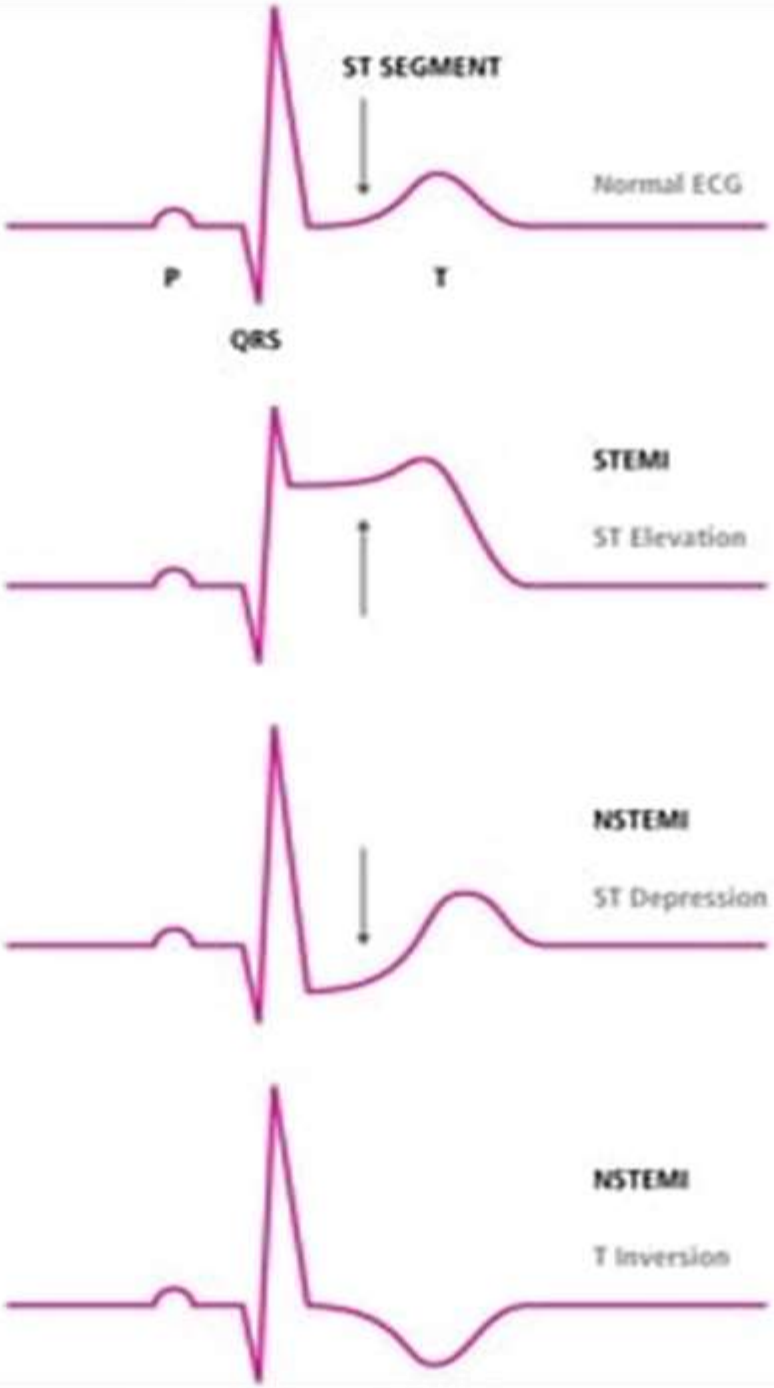
- 3. Finally, pathological Q waves occur, representing significant myocardial necrosis -& replacement by scar tissue.

(Pathologic Q waves are usually defined as duration ≥ 0.04 s or >25% of R-wave amplitude)

- In subendocardial infarction (Non ST Elevation Ml) :

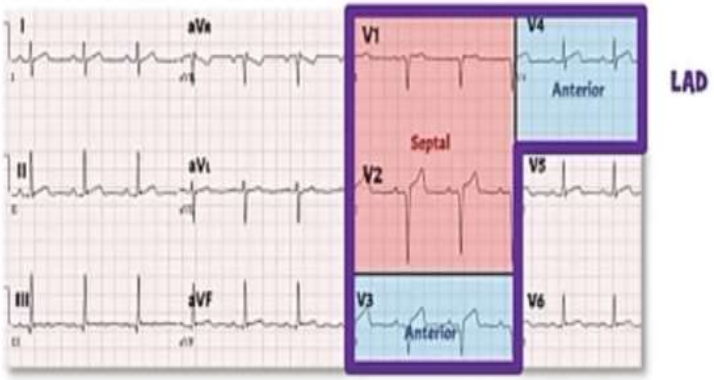
- 1. ST segment : normal or depressed.
- 2. No pathological Q waves (non Q wave Ml)
- 3. T wave : inverted.

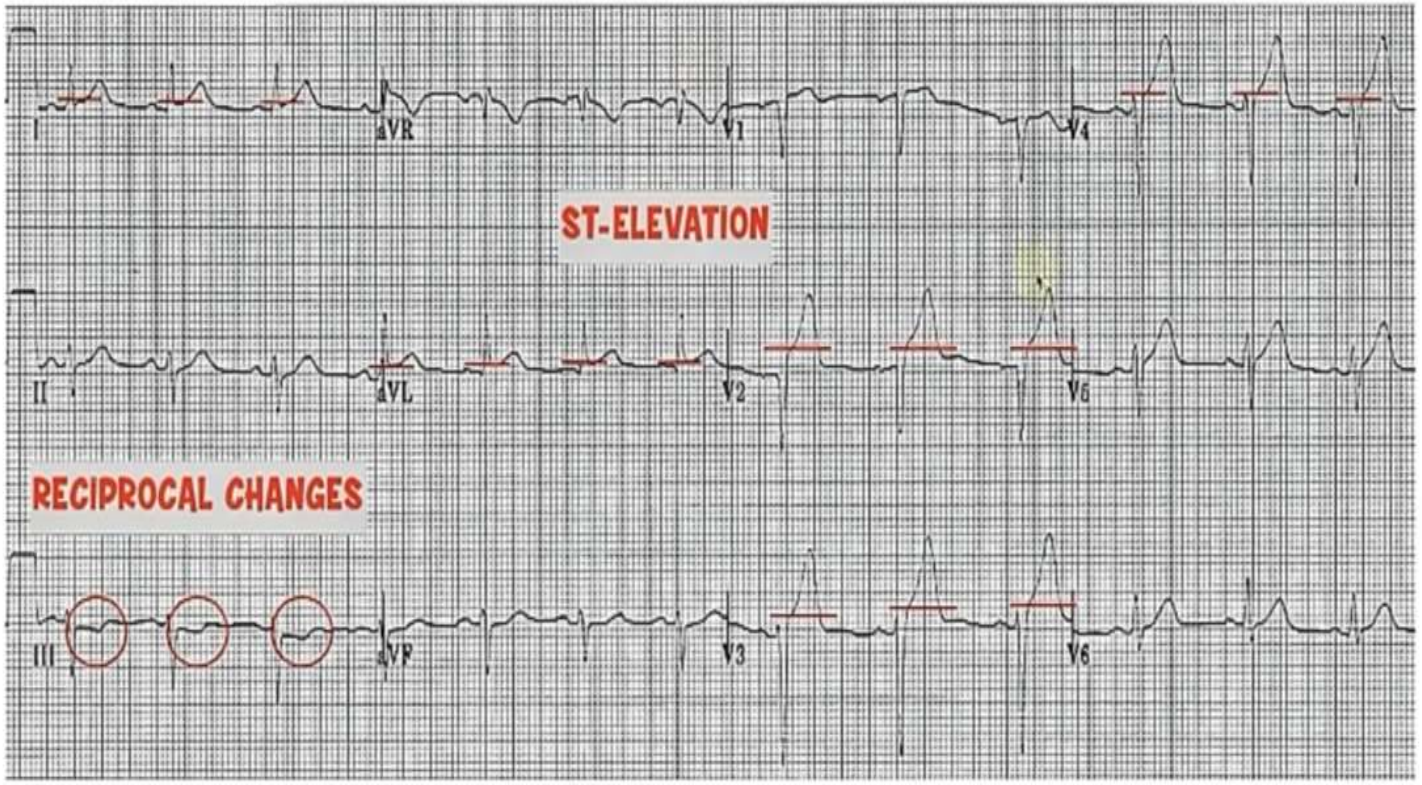
In old Ml : The only residual change is the pathological Q wave.



ECG
(ST elevation in Leads)

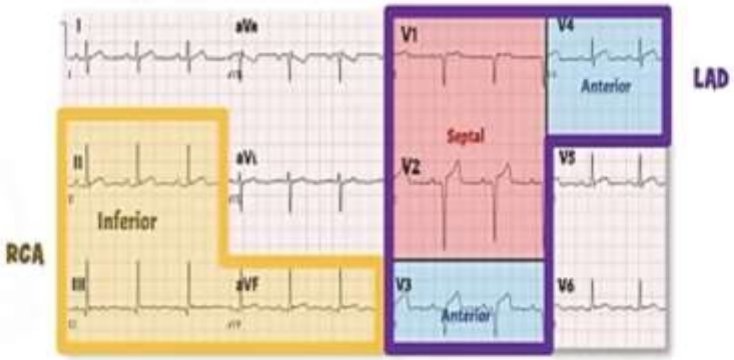
- Anterior (V1-V4) LAD

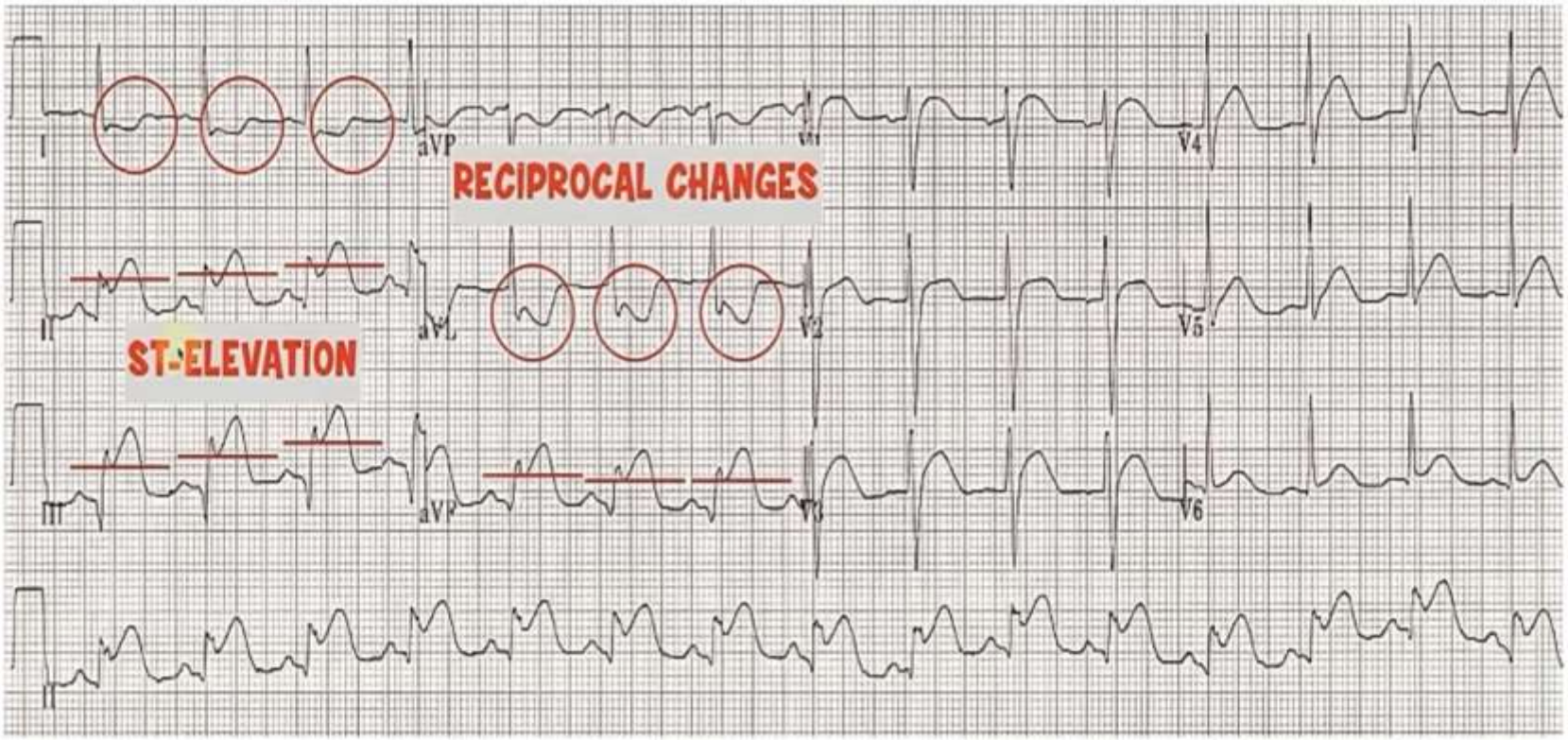




ECG
(ST elevation in Leads)

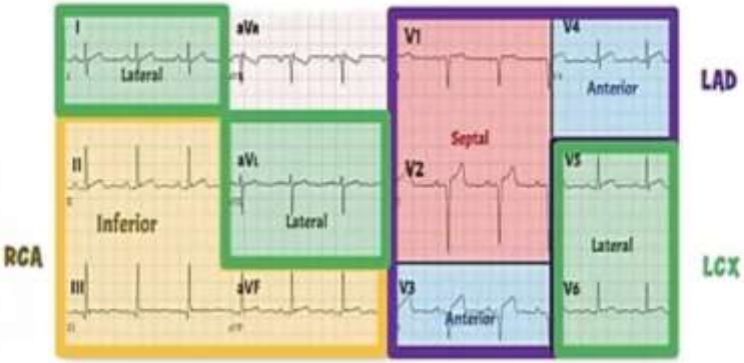
- Anterior (V1-V4) LAD
- Inferior (II,III, aVF) RCA

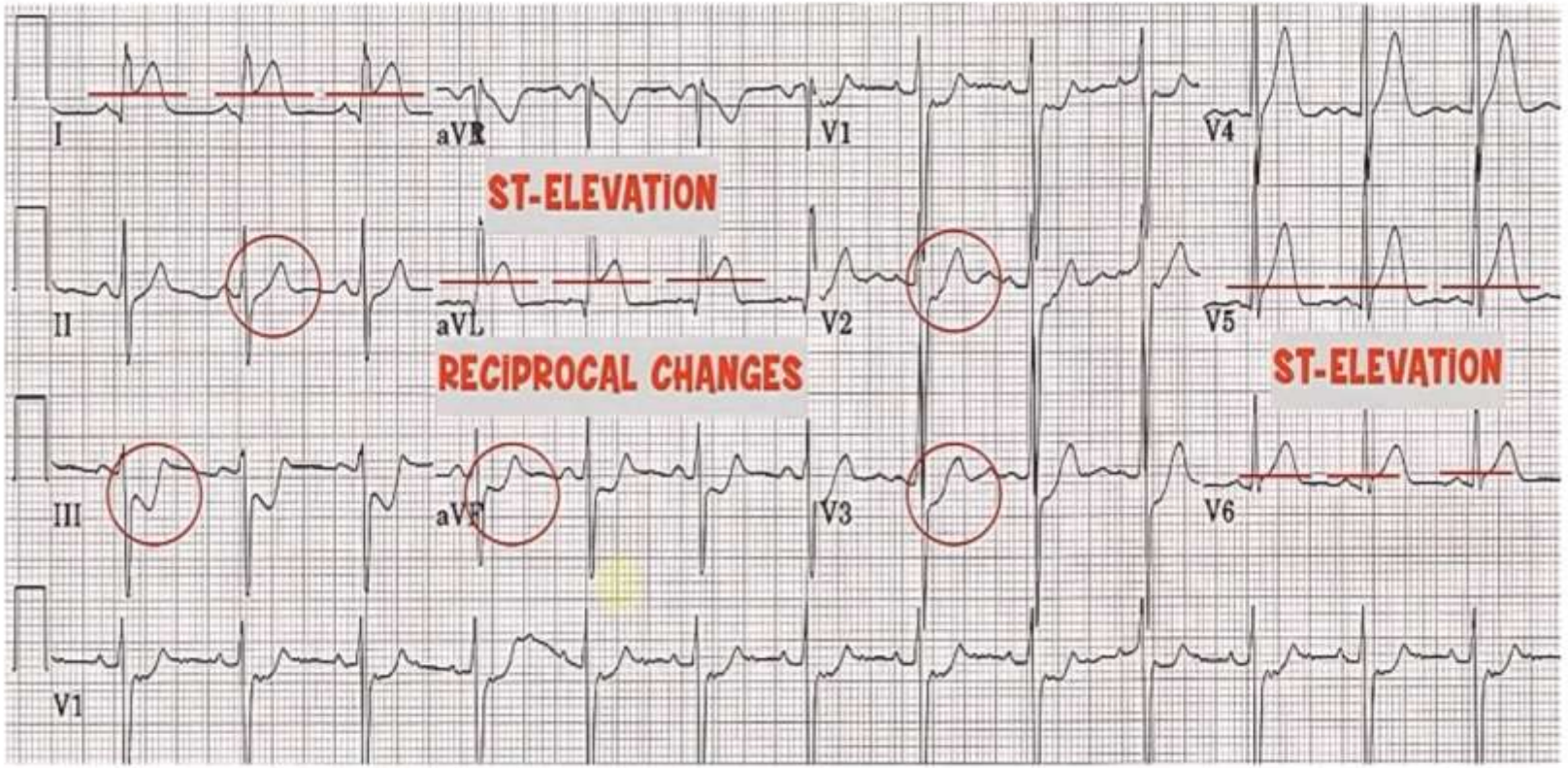




ECG
(ST elevation in Leads)

- Anterior (V1-V4) LAD
- Inferior (II,III, aVF) RCA
- Lateral (I, aVL, V5-V6) LCX





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 , cTnI)	3-12 h	1 week	Most sensitive & specific markers of myocardial damage .
Myoglobin	1-4 h	24 h	

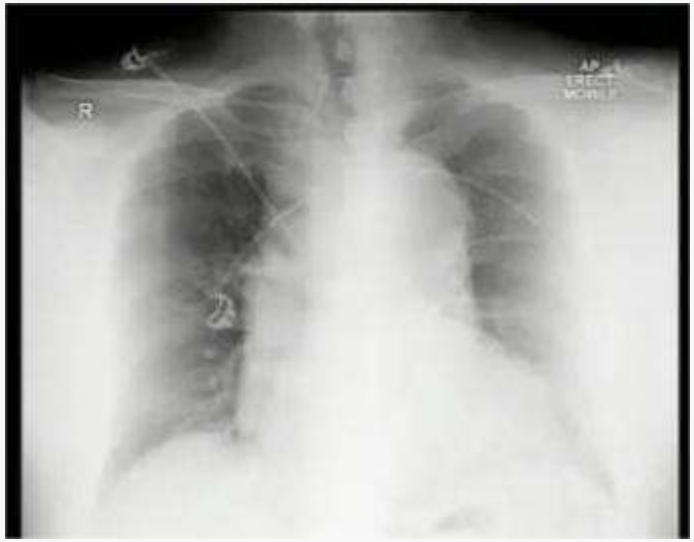
	EXTENT OF OCCLUSION	ECG	TROPONIN
STEMI	Total occlusion	ST elevation New LBBB	RAISED
NSTEMI	Incomplete occlusion	Other ischaemic changes / none	RAISED
UNSTABLE ANGINA	Incomplete occlusion	Other ischaemic changes / none	NORMAL

- **3- Echocardiography** :

Ventricular wall motion abnormalities.

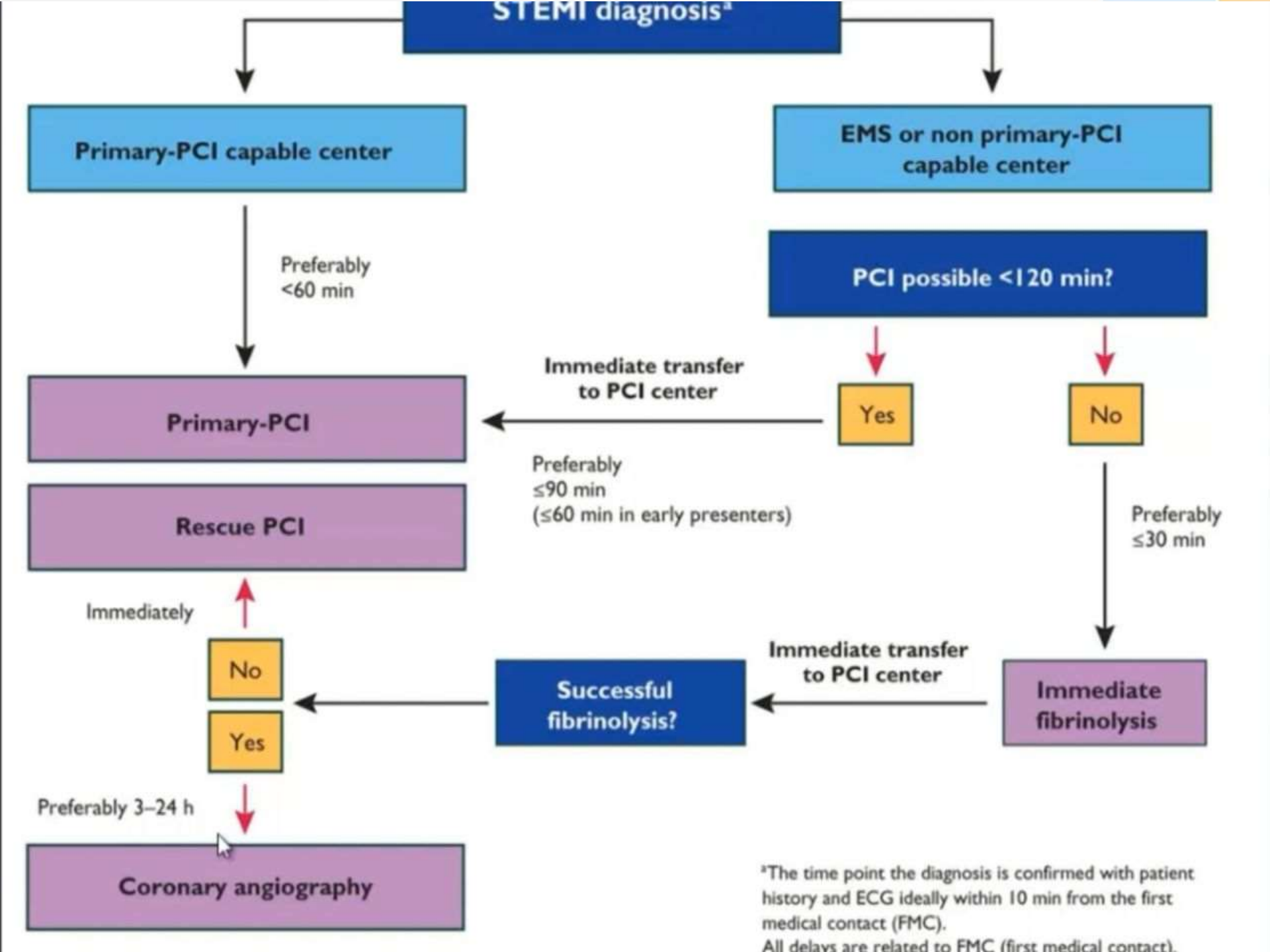
Complications: MR, myocardial aneurysm.

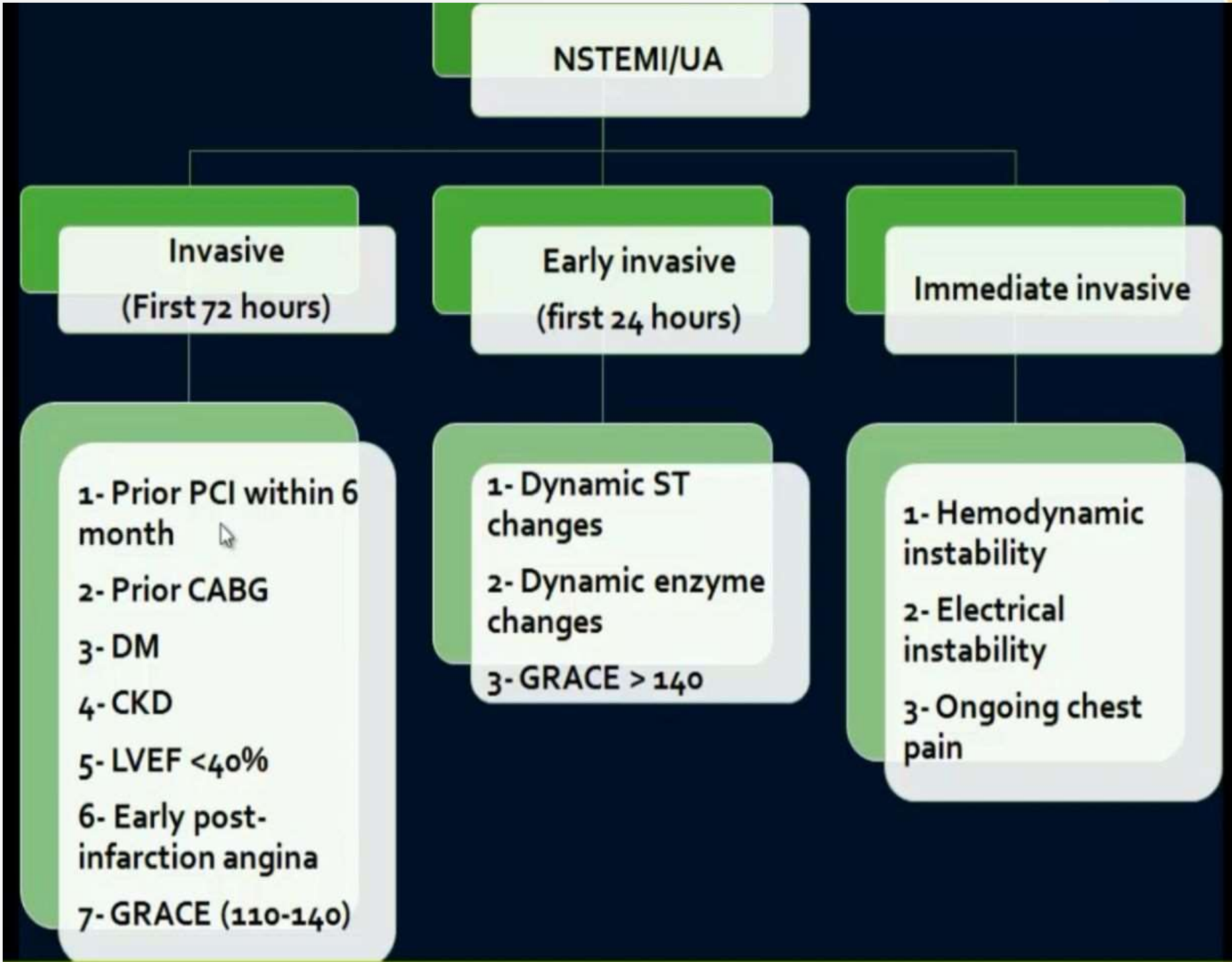
- **4- chest x ray** : to exclude other causes of acute chest pain especially aortic dissection



- **5- Leukocytosis** , ↑ **ESR**: as there is tissue damage.**LFT.KFT.PT.PTT.INR**

- **Emergency management**
- 300mg acetyl salicylic acid
- 6o0 mg clopidogril
- 8o mg atorvastatin
- 0.5mg/kg enoxaparin IV
- 5 mg SL iso-sorbide dinitrate
- 50 mg pethidine


STEMI diagnosis[a]
Primary-PCI capable center
EMS or non primary-PCI capable center
Preferably <60 min
PCI possible <120 min?
Immediate transfer to PCI center
Yes
No
Primary-PCI
Preferably ≤90 min (≤60 min in early presenters)
Rescue PCI
Preferably ≤30 min
Immediately
Immediate transfer to PCI center
No
Successful fibrinolysis?
Immediate fibrinolysis
Yes
Preferably 3–24 h
Coronary angiography
[a]The time point the diagnosis is confirmed with patient history and ECG ideally within 10 min from the first medical contact (FMC).
All delays are related to FMC (first medical contact).


NSTEMI/UA
Invasive
(First 72 hours)
Early invasive
(first 24 hours)
Immediate invasive
1- Prior PCI within 6 month
2- Prior CABG
3- DM
4- CKD
5- LVEF <40%
6- Early post-infarction angina
7- GRACE (110-140)
1- Dynamic ST changes
2- Dynamic enzyme changes
3- GRACE > 140
1- Hemodynamic instability
2- Electrical instability
3- Ongoing chest pain

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 over 60 min. may cause allergy.
- Alteplase, tenecteplase (tissue plasminogen activator - tPA)

Absolute Contraindications to Thrombolytic Therapy in STEMI

- Prior intracranial hemorrhage
- Known structural cerebral vascular lesion (e.g. arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Severe uncontrolled hypertension (unresponsive to therapy)
- Suspected aortic dissection
- Active bleeding or bleeding diathesis
- Significant closed-head or facial trauma within 3 months
- Ischemic stroke within 3 months
- Intracranial or intraspinal surgery within 2 months
- Prior treatment of streptokinase within the previous 6 months (if received streptokinase within previous 6 months, do NOT use streptokinase due to its highly antigenic nature and potential for a serious allergic reaction)

Relative Contraindications to Thrombolytic Therapy in STEMI

- Known intracranial pathology not covered in absolute contraindications
- Traumatic or prolonged (>10 min) CPR
- Systolic blood pressure > 180 mmHg or diastolic blood pressure > 110 mmHg upon presentation
- Significant history of chronic, severe, poorly controlled hypertension
- History of prior ischemic stroke that occurred more than 3 months ago
- Dementia
- Pregnancy
- Active peptic ulcer
- Major surgery within 3 weeks prior
- Recent internal bleeding within 4 weeks prior
- Noncompressible vascular punctures
- Oral anticoagulation therapy

Medications in CCU

- Acetyl salicylic acid.....75 mg 1X1
- Clopidogril.....75 mg 1X1
- Atorvastatin.....40mg 2X1
- Bisoprolol.....5mg 1X1
- Valsartan.....160 mg 1X1
- Enoxaparin.....80 mg 1x2 SC
- Nitrates infusion
- Pantoprazole..... 40mg 1X1

I) **Anti-platelet drugs**

- Aspirin for life

Loading: 300 mg 1x1

Maintenance 75-100mg

- P2Y12 inhibitors for one year
- **Ticagrelor** for all ACS unless unavailable or Cl [OAC-advanced liver disease-history of ICH)
- **Prasugrel** the same + is Cl in patients with history of ischemic stroke

Not preferred in old /renal/patients/< 6o kg

- **Clopidogril**

Loading 300/6oo mg then 75 mg 1x1

II)Statins

- for life
- Loading:80 mg atorvastatin
- Maintenance 40-80 mg atorvastatin/day
- 10-20 mg rosuvastatin/day

III)Anticoagulants

Heparin (UFH):

loading dose 60-100 U/Kg followed by maintenance dose of10-12u/kg/hour adjusted by PTT (50-70 s).

Low molecular weight heparin (LMWH)

- 0.5 mg/Kg iv bolus then 1mg/kg/12 hours subcutanequs

Fondaparinux

- 2.5 mg IV followed by 2.5 mg SC once daily.
- But Cl in patients undergoing intervention [catheter thrombosis]

- Anticoagulant therapy should be stopped after PCI is performed (unless other indication) otherwise it should be continued throughout the hospital stay (average 8 days).

IV) ACEI /ARBs

e.g captopril , enalapril, Valsartan

Indications?

1-STEMI

2- NSTEMI/UA if (DM-HPN-LV dysfunction-Renal-history of MI)

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

V) Beta blockers

- e.g Bisoprolol
- Target?
- Heart rate 60-70/minute

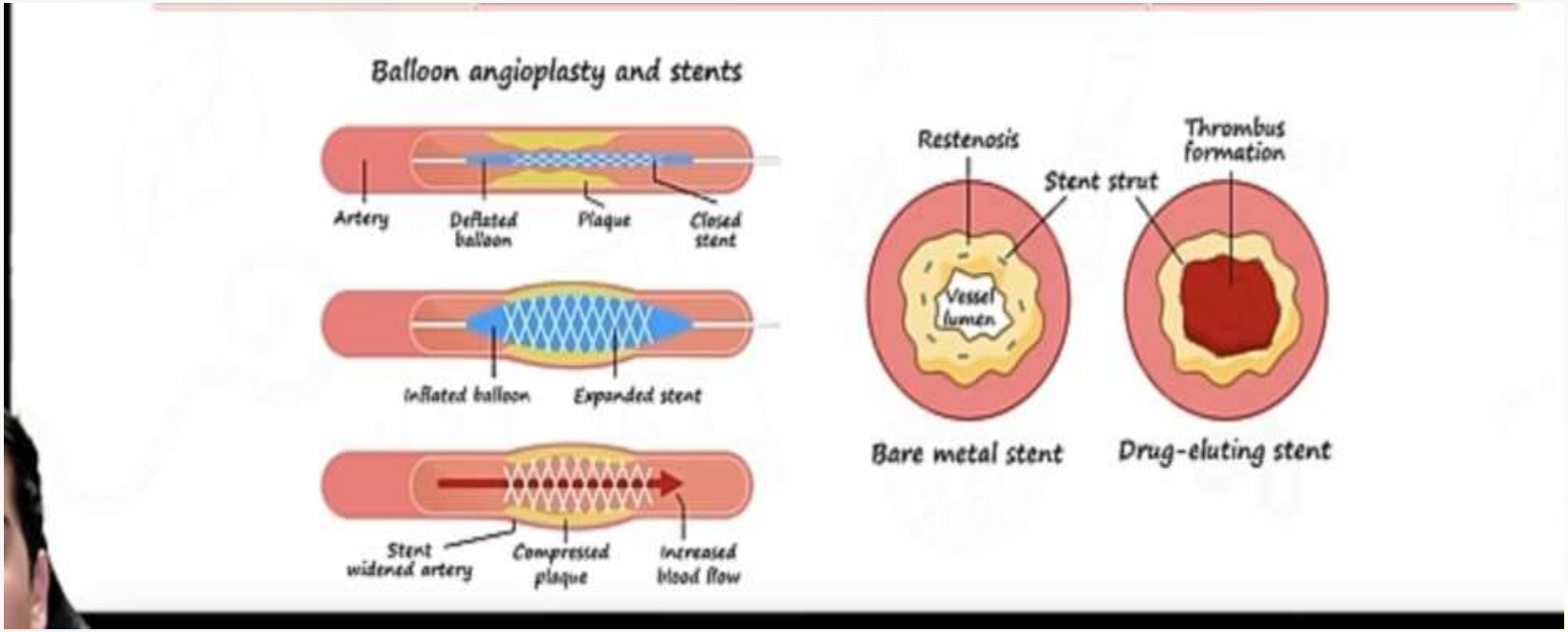
VI) Nitrates

- IV nitrates should be given in the first 24 to 48 hours then in patients with heart failure or residual ischemia shift to oral nitrates (routine use is not beneficial).

Percutaneous Coronary Intervention (PCI)

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

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



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.


THANK
YOU