Isfahan Cardiovascular Research Institute

THE NAMES OF
GOD

WHO Collaborating Center

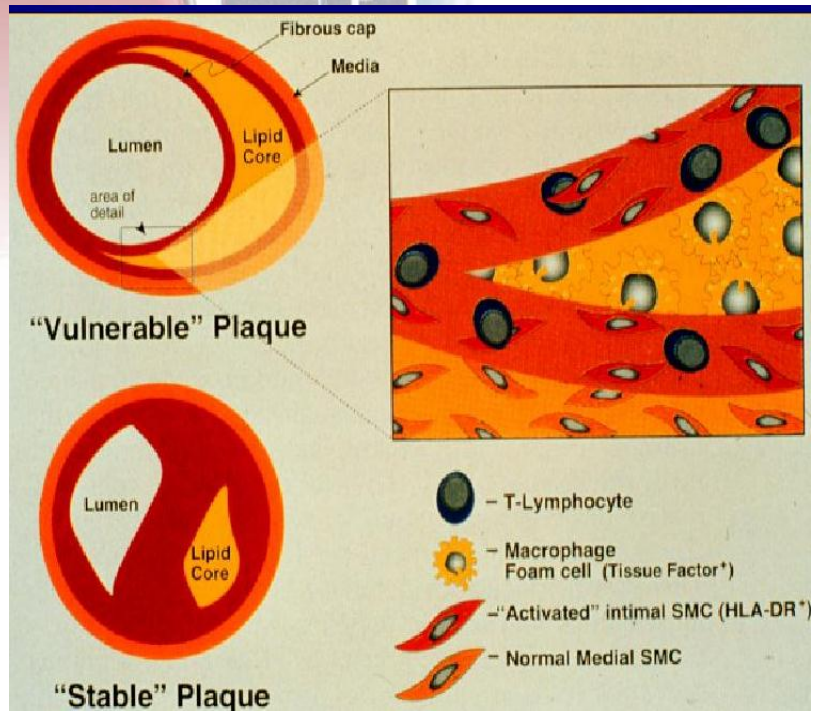
Masoumeh sadeghi, MD
Professor in cardiology
Cardiovascular Research Institute, Isfahan University of Medical Sciences

Paraclinic tests in AMI

Definition of Myocardial infarction

- Myocardial infarction is an ischemic necrosis of the myocardium, caused by occlusion of coronary artery and prolonged myocardial ischemia.
- MI is an extreme consequence of acute coronary syndromes – the spectrum of clinical states caused by instability of coronary artery lumen due to plaque instability and atherothrombosis.

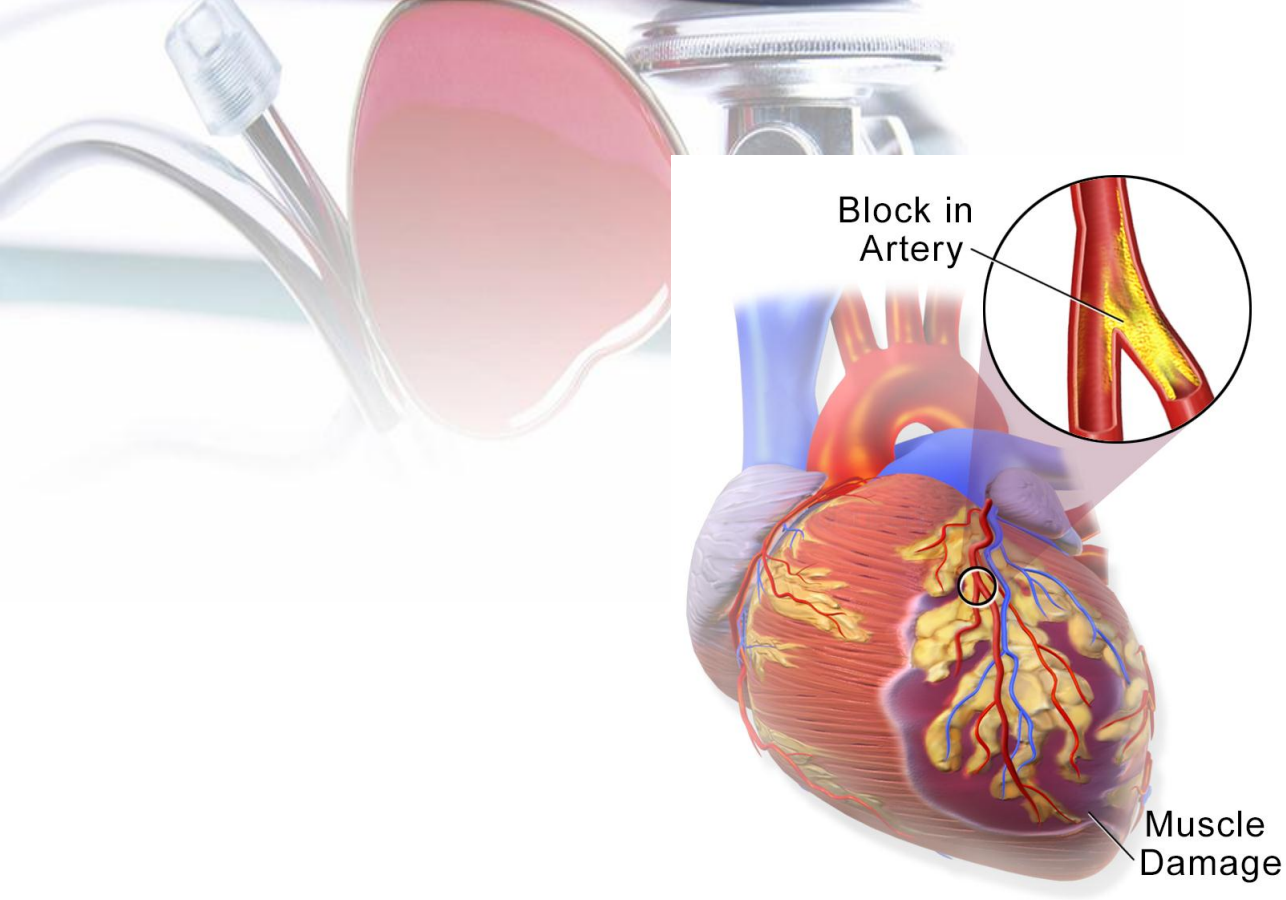
"Vulnerable" Plaque and "Stable" Plaque



Libby. *Circulation.* 1995;91:2844-2850.

What happens when you have a myocardial infarction?

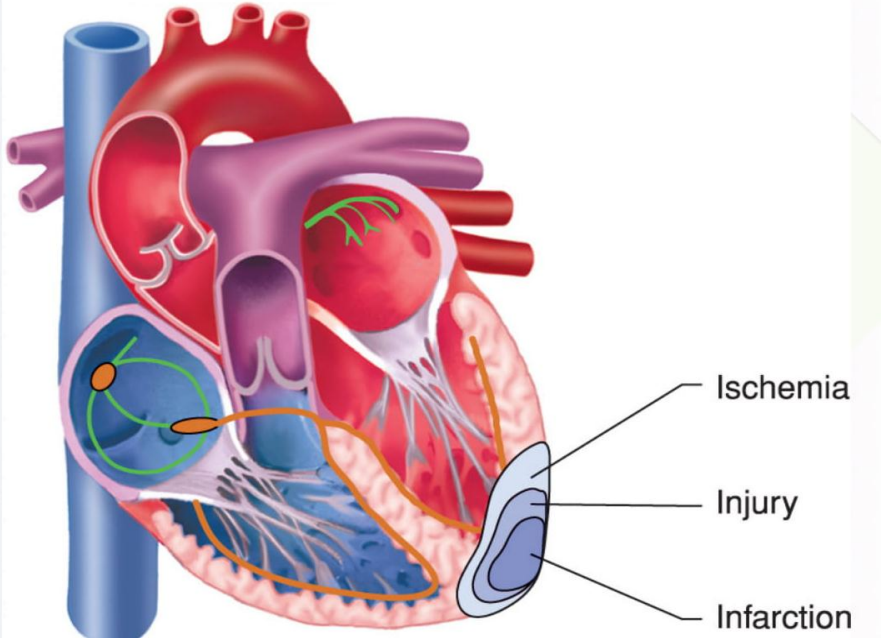
- If you have an MI, a coronary artery or one of its smaller branches is suddenly blocked.
- The part of the heart muscle supplied by this artery loses its blood (and oxygen) supply bringing it at risk of dying unless the blockage is quickly removed (the word ’infarction’ means death of some tissue).
- If one of the main coronary arteries is blocked, a large part of the heart muscle is threatened. If a smaller branch artery is blocked, a smaller amount of heart muscle is affected.
- In people who survive an MI, the part of the heart muscle that dies (’infarcts’) is replaced by scar tissue over the next few weeks to months which can cause future problems such as heart failure or arrhythmia.



Heart Attack

Myocardial Infraction

- Death of myocardial cells



Who has a myocardial infarction?

- (ACS) is the most common reason for hospitalization in ISFAHAN, 50% of patients admitted with this diagnosis develop an MI .

- Most MIs occur in people over 50, and become more common with increasing age. Sometimes younger people are affected. An MI is more common for men than women.

- People with risk factors such as smoking, diabetes, hyperlipidemia ,hypertensive, obesity have a higher risk of MI.

What are the symptoms of a myocardial infarction?

- Severe chest pain is the usual main symptom. The pain may also travel up into your jaw, and down your left arm, or down both arms.

- You may also sweat, feel sick, and feel faint. The pain may be similar to angina, but it is usually more severe and lasts longer.
- (Angina usually goes off after a few minutes. MI pain usually lasts more than 15 minutes - sometimes several hours. A small MI occasionally happens without causing pain (a 'silent MI').

- It may be truly pain-free, or sometimes the pain is mild and you may think it is just heartburn or 'wind'.

- Some people collapse and die suddenly if they have a large or severe MI.

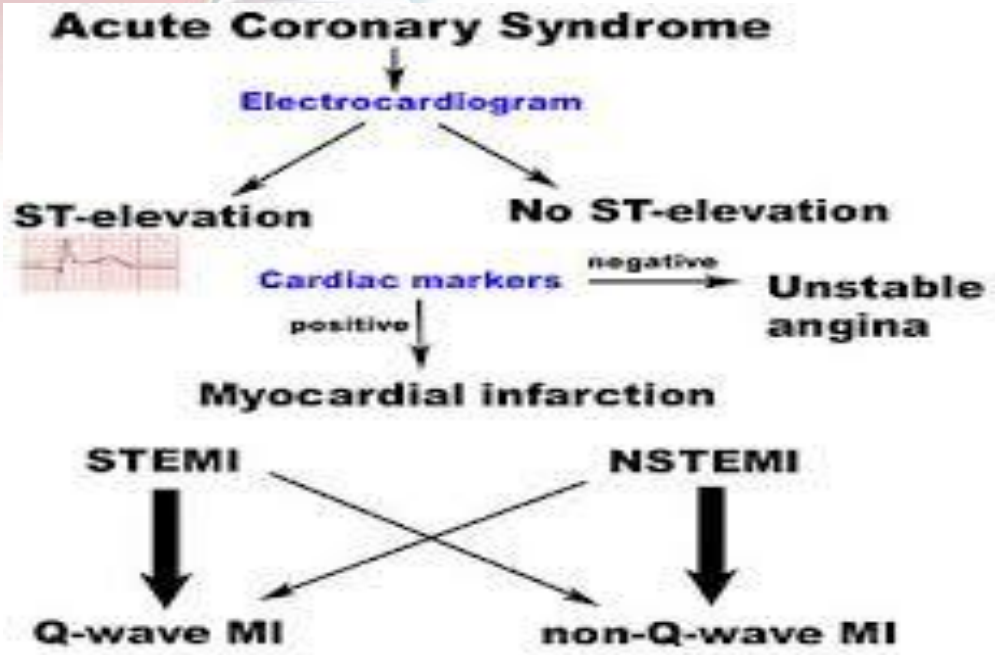
What should I do in a myocardial infarction?

- Treatment is commenced straight away and you will normally be admitted to hospital for observation, diagnosis and further treatment.

- How is myocardial infarction diagnosed and assessed?

- Many people develop chest pains that are not due to an MI.
  For example, you can have quite bad chest pains with heartburn, gallbladder problems, or with pains from conditions of the muscles or bones in the chest wall. However, tests can usually confirm MI.

Acute Coronary Syndrome



MI Classifications

- MI’s can be subcategorized by anatomy and clinical diagnostic information.

Anatomic:

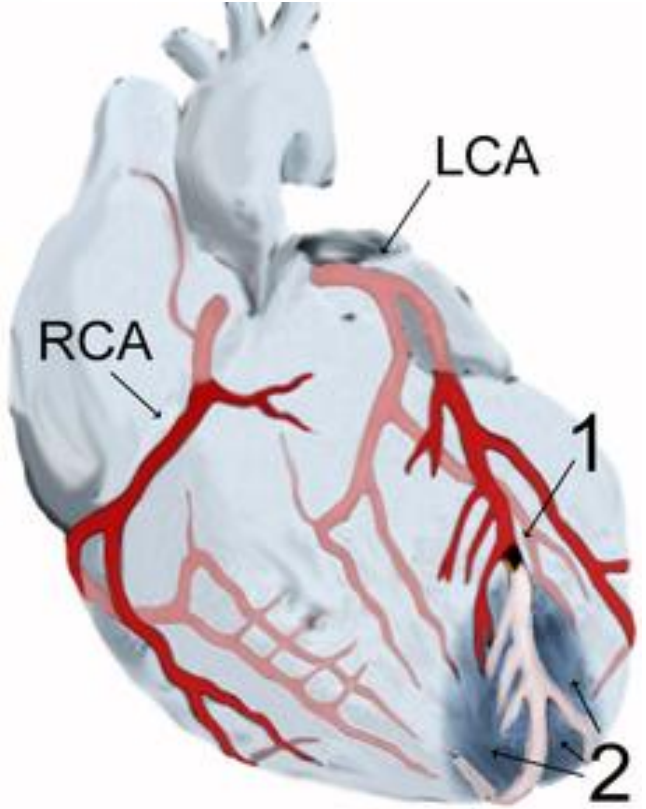
❖ Transmural and Sub endocardial

Diagnostic:

❖ ST elevations (STEMI) and non ST elevations (NSTEMI).

LOCATION OF THE INFARCTION

- MIs can be located in the anterior, septal, lateral, posterior, or inferior walls of the left ventricle.



Location of Myocardial Infarction, Electrocardiographic (ECG) Findings, and Clinical Implications

Anatomical Location	Coronary Artery	ECG Evidence	Clinical Implications
Anteroseptal wall	Left anterior descending: Supplies blood to the anterior wall of left ventricle, the interventricular septum, and the ventricular conducting tissue	V_1 through V_4, Q waves and ST segment elevations	Potential for significant hemodynamic compromise; congestive heart failure, pulmonary edema, cardiogenic shock; intraventricular conduction disturbances
Lateral wall	Left circumflex: Supplies blood to the left lateral and left posterior walls and to the SA node in 45% of people and AV node in 10% of people	I, aVL, V_5, and V_6, Q waves and ST segment elevations	Evaluation for posterior wall involvement; some hemodynamic changes; dysrhythmias caused by SA and AV node dysfunction
Posterior wall	Left circumflex: Supplies blood to the left lateral and left posterior walls and to the SA node in 45% of people and AV node in 10% of people	V_1 and V_2, tall upright R waves with ST segment depression; Q waves and ST segment elevation in V_7 through V_9	Evaluation for lateral wall involvement; some hemodynamic changes; dysrhythmias caused by SA and AV node dysfunction
Inferior wall	Right coronary artery: Supplies blood to the inferior wall of the left ventricle, the right ventricle, and the SA node in 55% of people and the AV node in 90% of people	Q waves and ST segment elevation in II, III, aVF	Evaluation for right ventricular wall involvement; some hemodynamic changes; potential for significant arrhythmias caused by SA and AV node dysfunction
Right ventricular wall	Right coronary artery: Supplies blood to the inferior wall of the left ventricle, the right ventricle, and the SA node in 55% of people and the AV node in 90% of people	Q waves and ST segment elevations in right precordial chest leads (RV_1 through RV_6)	Evaluation for inferior wall involvement; some hemodynamic changes; potential for significant dysrhythmias caused by SA and AV node dysfunction

INVESTIGATIONS

- ECG
- Biochemical Cardiac Marker: Troponin I, Troponin T, CK-MB, Myoglobins, AST and LDH
- Echocardiography
- Chest X ray
- ECHO
- Fasting lipid profile and Blood sugar
- ETT (Exercise Tolerance Test)
- Other stress testing:

• Stress echocardiography

• Myocardial perfusion scan

• Transthoracic echocardiography

Laboratory Tests

- *Creatine Kinase*

- CK-MB appears in the serum in 6 to 12 hours, peaks between 12 and 28 hours, and returns to normal levels in about 72 to 96 hours.

- Serial samplings are performed every 4 to 6 hours for the first 24 to 48 hours after the onset of symptoms

- *Creatine Kinase Isoforms:* CK-MB1 is the isoform found in the plasma, and CK-MB2 is found in the tissues. In the patient with an MI, the CK-MB2 level rises, resulting in a CK-MB2 to CK-MB1 ratio greater than one

Laboratory Tests

- *Myoglobin:* Myoglobin is an oxygen-binding protein found in skeletal and cardiac muscle. Myoglobin's release from ischemic muscle occurs earlier than the release of CK.

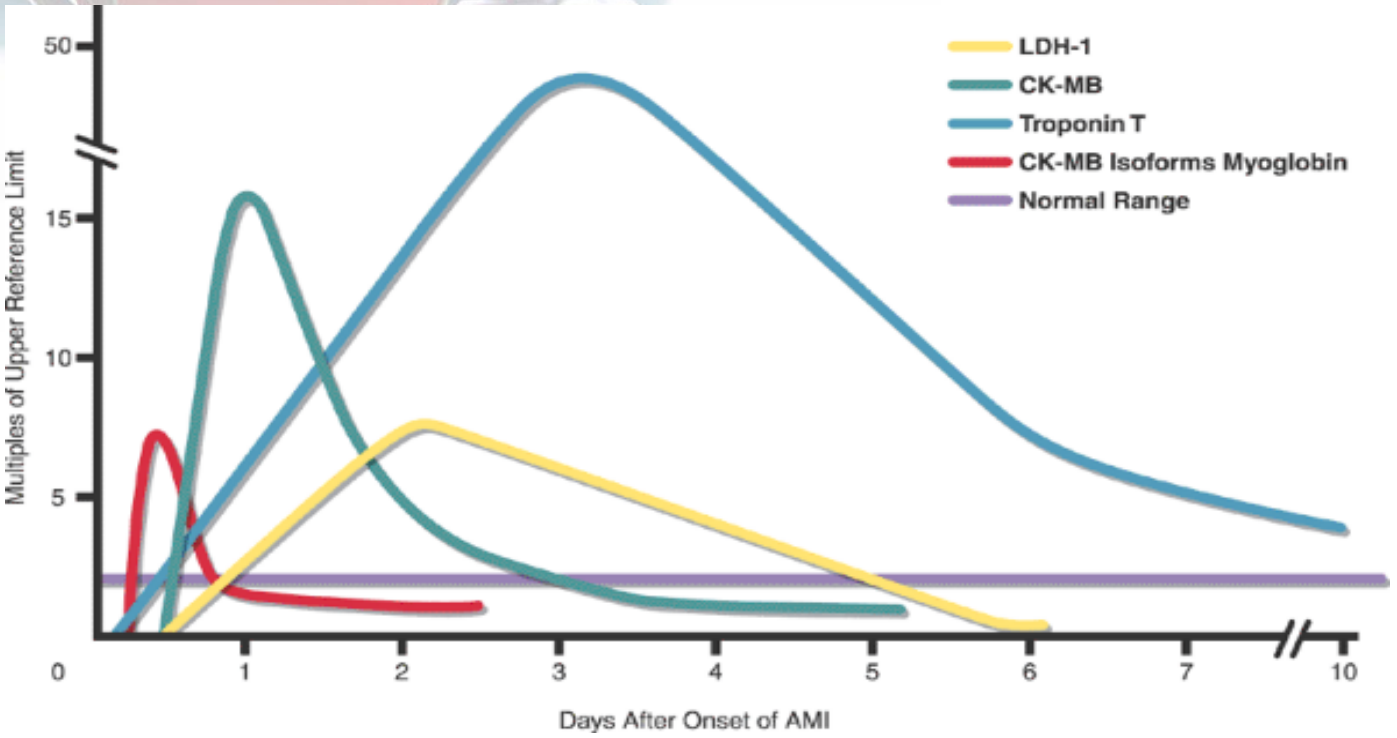
- The myoglobin level can elevate within 1 to 2 hours of acute MI and peaks within 3 to 15 hours.

- Because myoglobin is also present in skeletal muscle, an elevated myoglobin level is not specific for the diagnosis of MI. consequently, its diagnostic value in detecting an MI is limited

Laboratory Tests

- Troponin. (troponin T and troponin I):
- Troponin I levels rise in about 3 hours, peak at 14 to 18 hours, and remain elevated for 5 to 7 days.
- Troponin T levels rise in 3 to 5 hours and remain elevated for 10 to 14 days

Release of cardiac markers after acute myocardial infarction (AMI)



Findings of cardiac marker

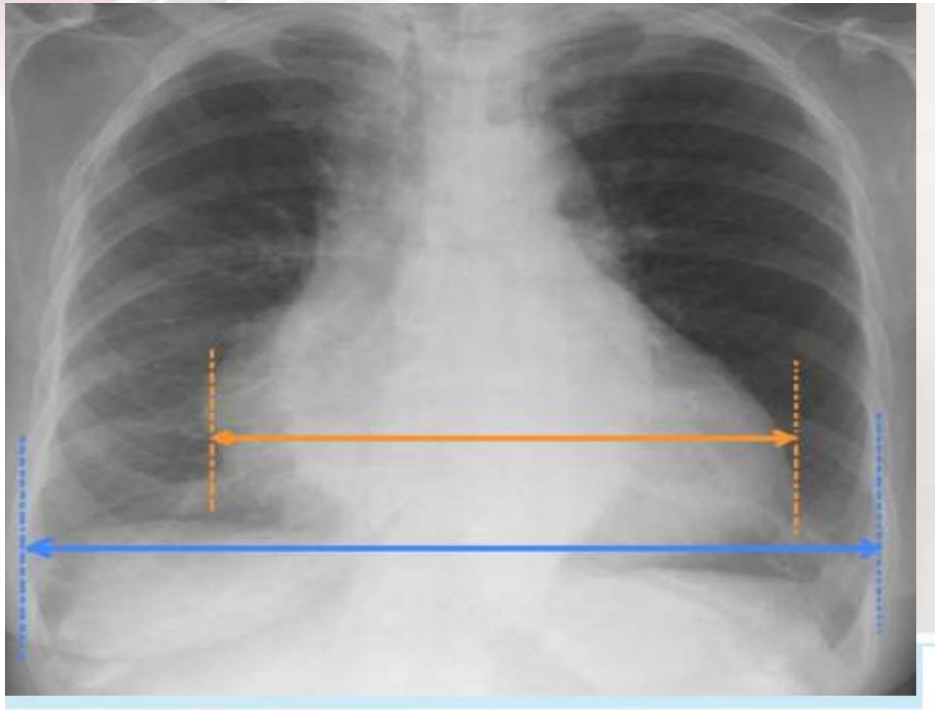
CK-MB	TROPONIN I, TROPONIN T	MYOGLOBIN	AST	LDH
Rise with in 4-6 hours	Rise with in 3 hours		Rise in about 12 hours of attack	Rises relatively late after 12 hour
Peak appears 12-24 hours			Reaches to peak in 24-36 hours	Reaches its peak by 36-48 hours
Normalize after 48-72 hours	Normalize after 2 weeks	Normalize in 3-5 days	Normalizes in 3-5 days	Normalizes by 3 weeks

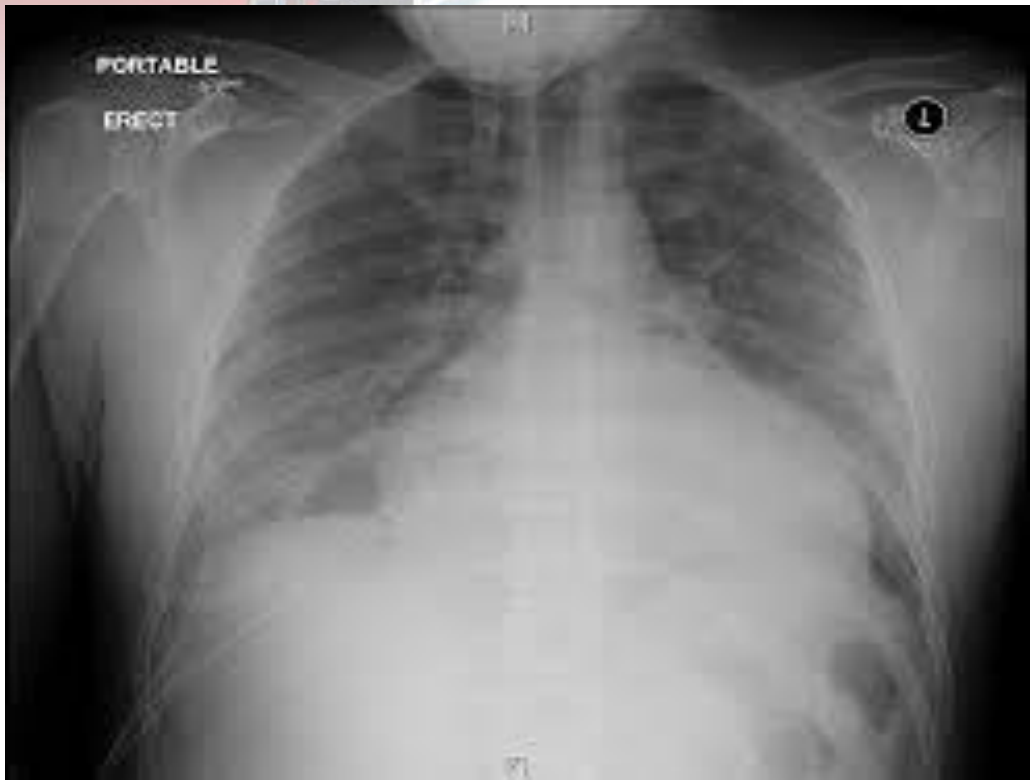
CXR MI

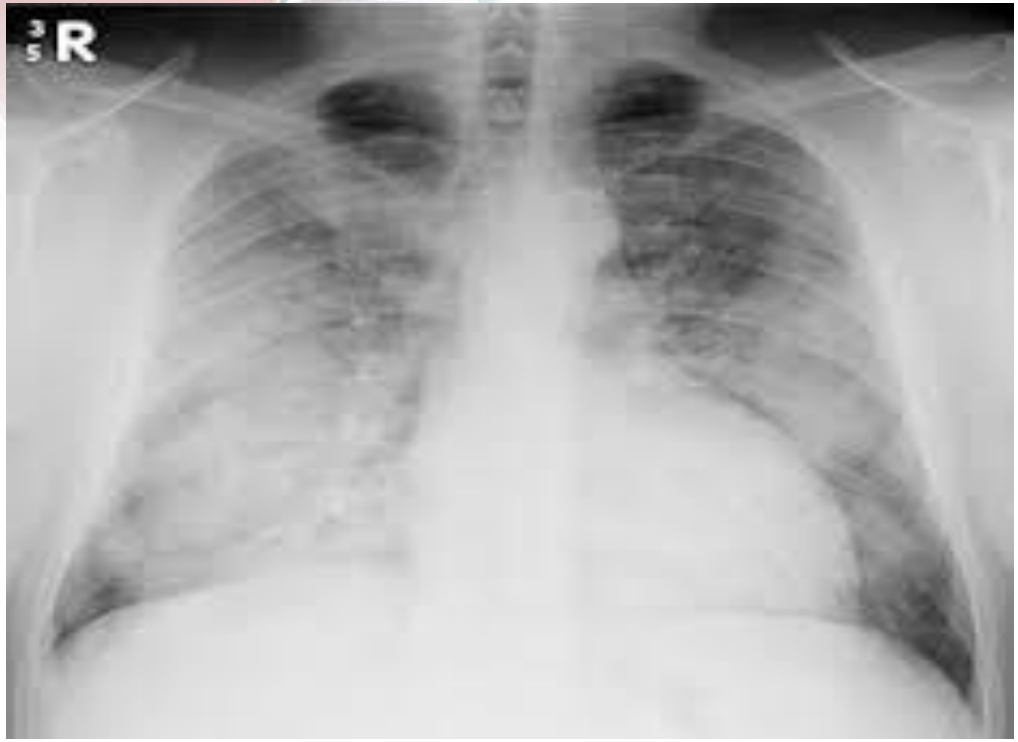

Airspaces
Bones
Cardiac
Diaphragms
Examination Technique
Fluid
Gastric Bubble
Hilum
Instrumentatio
n

Investigations-Chest Xray

- Chest Xray to determine cardiomegaly and pulmonary edema

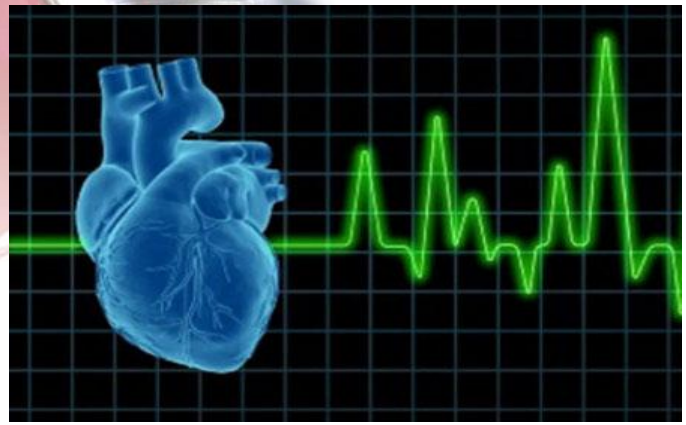



PORTABLE
ERECT
L

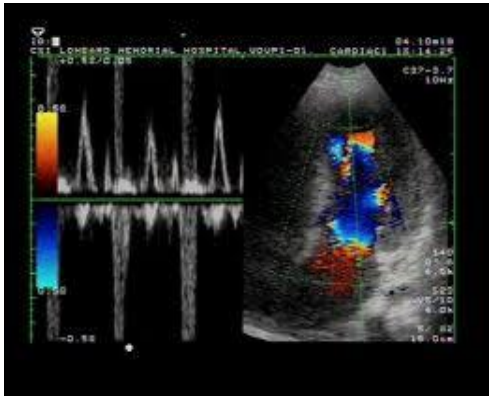

3
5
R

Paraclinical tests in patients with acute myocardial infarction

exercise test

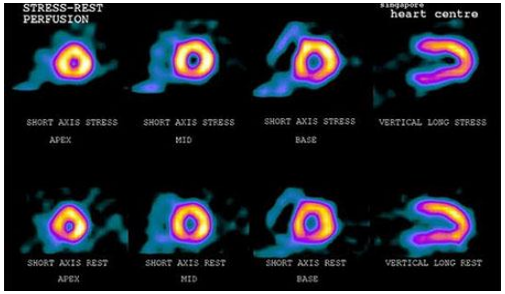


ECG



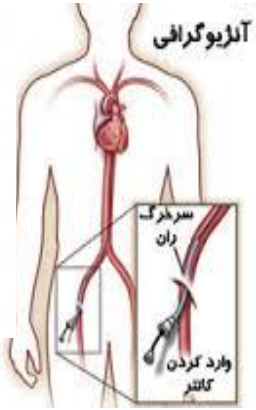
echocardiography

CTA



Heart Scan

Holter monitoring



angiography