ISCHEMIC HEART DISEASE

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Ischemic Heart Disease (IHD)

When to suspect patient with IHD

Basic: coronary circulation

Myocardial oxygen supply and demands

Causes of IHD

Management

Case presentation

A 65 Year old male, presented to outpatient clinic
complaining of chest pain of 5 months duration.

What are the possible anatomical causes of chest pain?

The pain is retrosternal, compressive in nature, precipitated by wakening of 400 meter , relieved by rest, radiated to left shoulder, associated with sweating.

Patient is diabetic
And smoker

On examination:
Blood pressure:160/100. pulse rate: 88 bpm
Heart auscultation : normal
What is the Problem? STABLE ANGINA
What is abnormal physical findings? HIGH BLOOD PRESSURE

Symptoms of Angina

Angina can spread anywhere between the belly button and the jaw, including to the shoulder, arm, elbow or hand- usually on the left side.

Angina Chest Pain:

Clinical Diagnosis

Ischemic Heart Disease

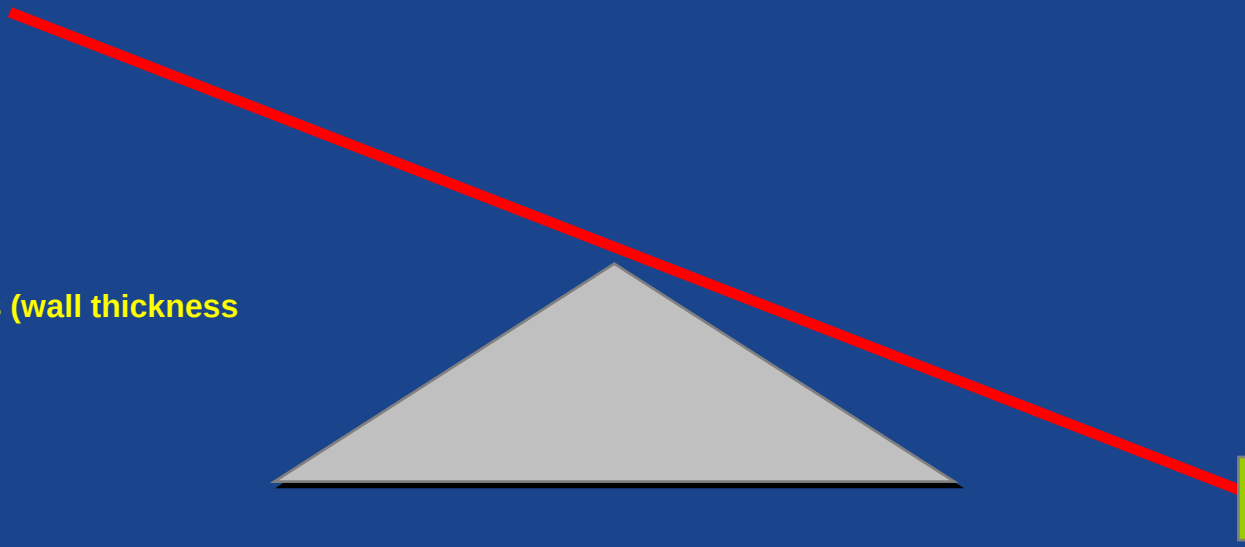
demand

1- Heart rate

2- Contractility

3- Wall tension

4- Muscle mass (wall thickness



supply

1- Coronary flow (patency of coronary artery)

2- Hemoglobuline level

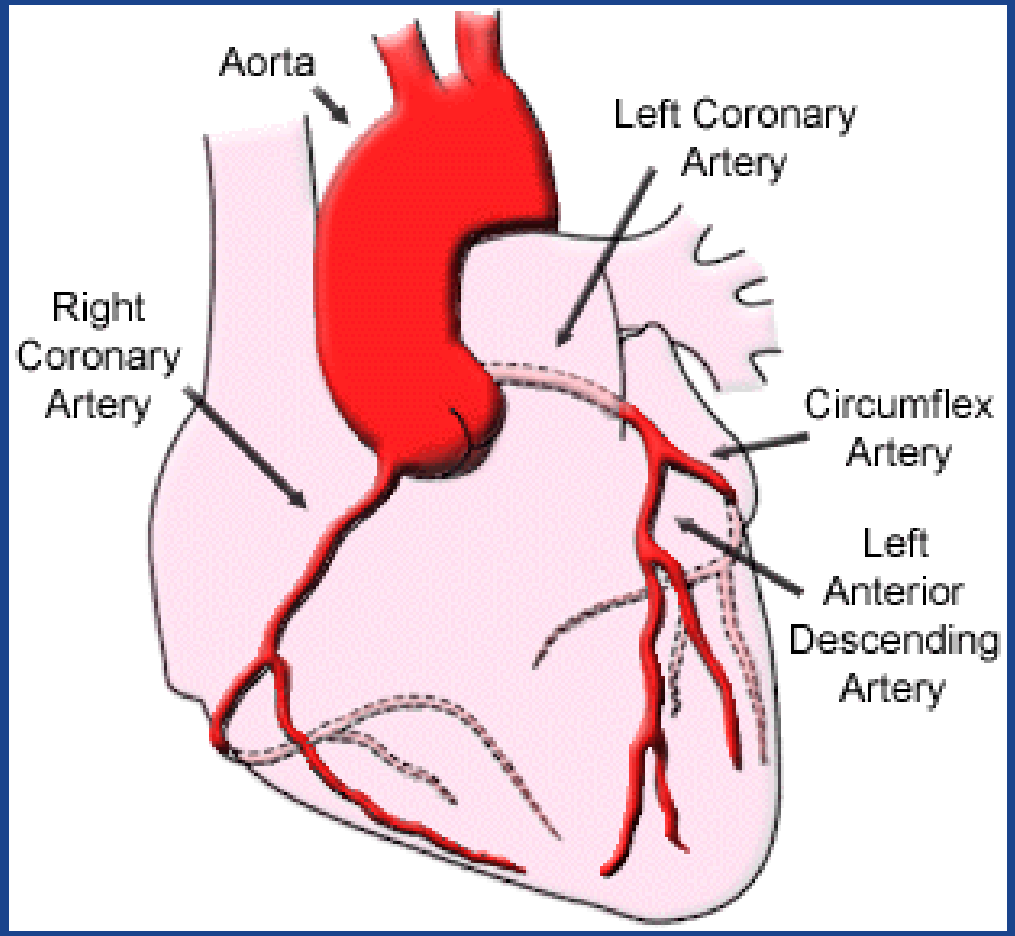
3- Myocardial oxygen extraction

4- Arterial oxygen saturation

Causes of coronary artery disease

95% Atherosclerosis	***5% Nonatherosclerosis***	
Risk factors:		

Coronary Anatomy



Risk Factors for Cardiovascular Disease

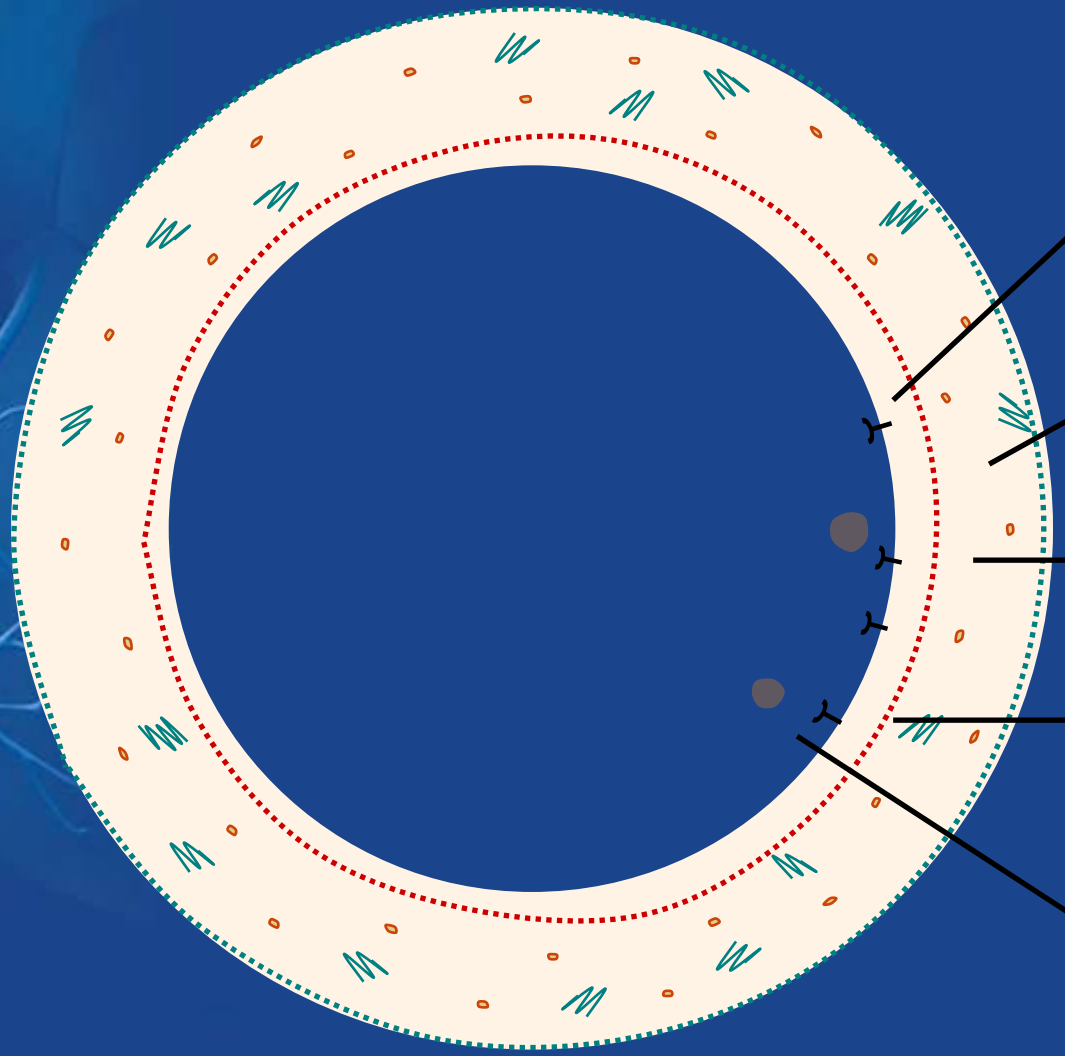
Modifiable

- Hypertension
- Smoking
- Hyperlipidaemia
  - Raised LDL-C
  - Low HDL-C
  - Raised triglycerides
- Diabetes mellitus
- Dietary factors
- Lack of exercise
- Obesity
- Homocysteinemia
- Lipoprotein a
- Gout
- Thrombogenic factors: fibrinogen, factors V,VII
- Excess alcohol consumption

Non-modifiable

- Personal history of CVD
- Family history of CVD
- Age: M>45, F>55
- Gender M>F (Premenopausal)
- Personality type A
- Genetic factors: ACE gene

Endothelial Dysfunction in Atherosclerosis



Upregulation of endothelial adhesion molecules

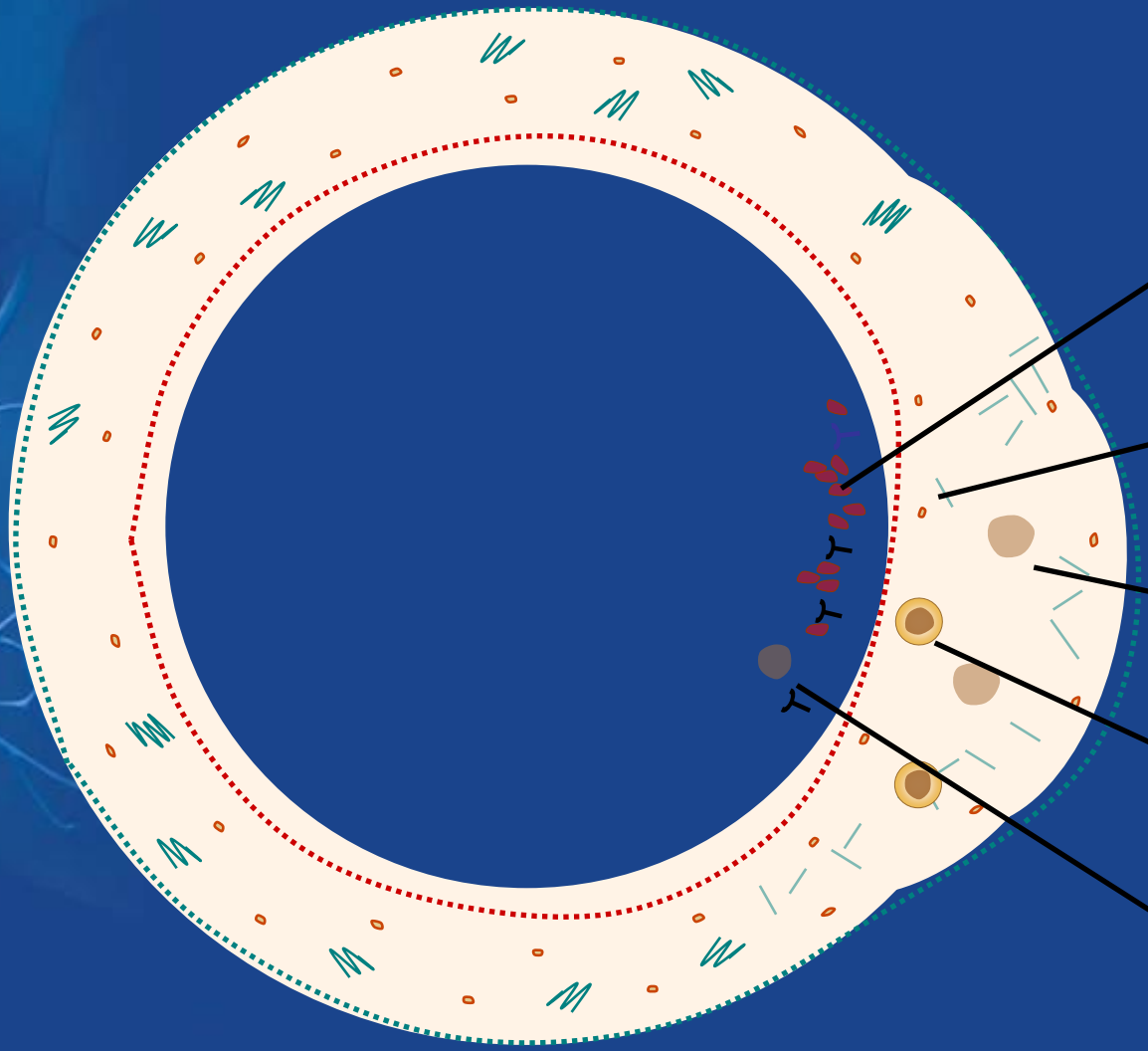
Migration of leucocytes into the artery wall

Lipoprotein infiltration

Increased endothelial permeability

Leucocyte adhesion

Fatty Streak Formation in Atherosclerosis



Adherence and aggregation of platelets

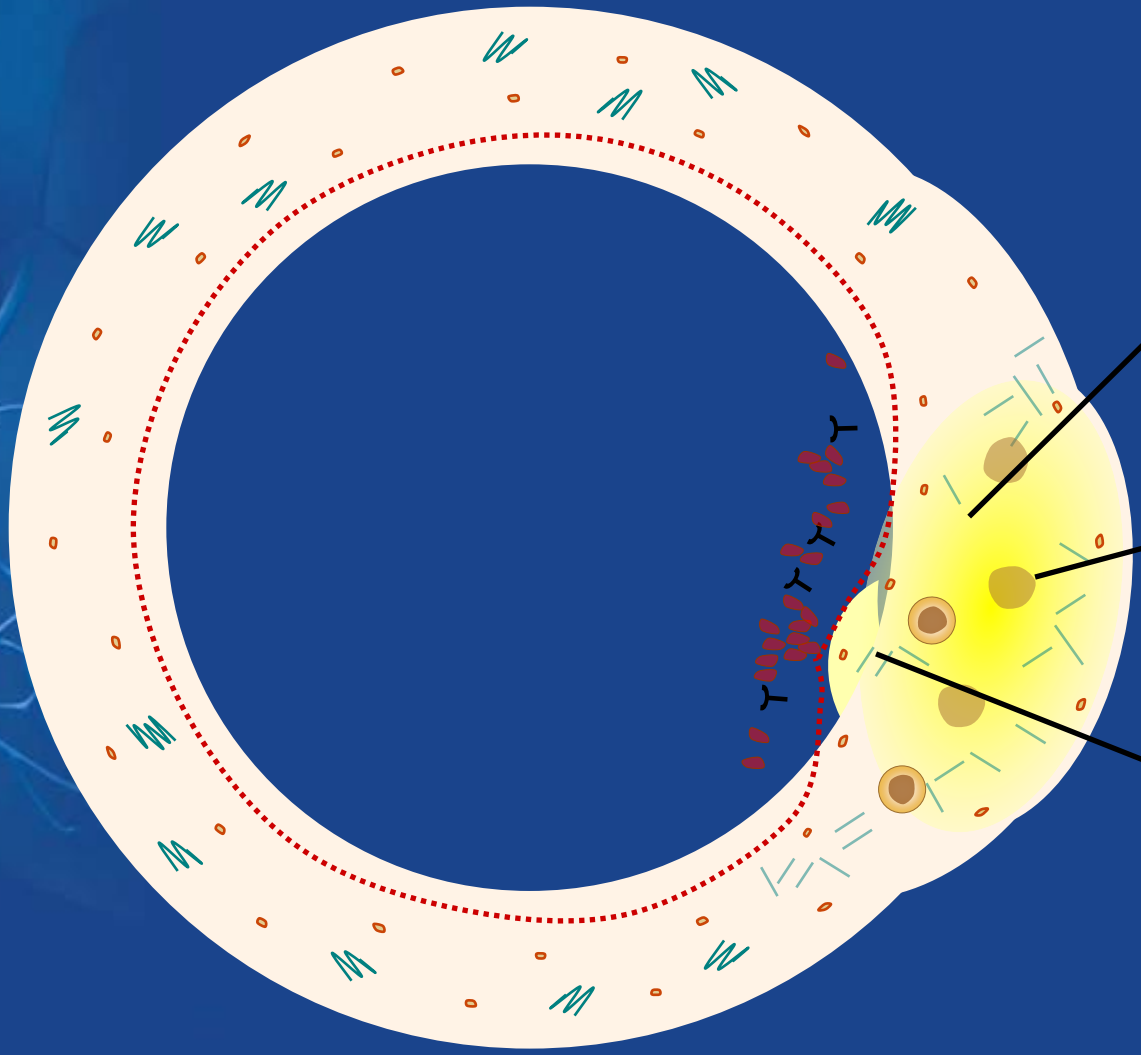
Migration of smooth muscle cells

Formation of foam cells

Activation of T cells

Adherence and entry of leucocytes

Formation of the Complicated Atherosclerotic Plaque



Formation of necrotic core

Accumulation of macrophages

Formation of the fibrous cap

What to do? Investigations

Stable angina-
Diagnosis

- History : angina pectoris is clinical diagnosis
- Physical exam
- Electrocardiogram: 12 ECG, 24 ECG
- Stress ECG : diagnostic and prognostic information
- Radioactive studies: thalium scan,..
- Echocardiography
- CT Coronary angiography
- Serum lipid(LDL, HDL, TG), FBG,CBC
- Coronary angiography

Imaging Techniques Used to Assess Atherosclerosis

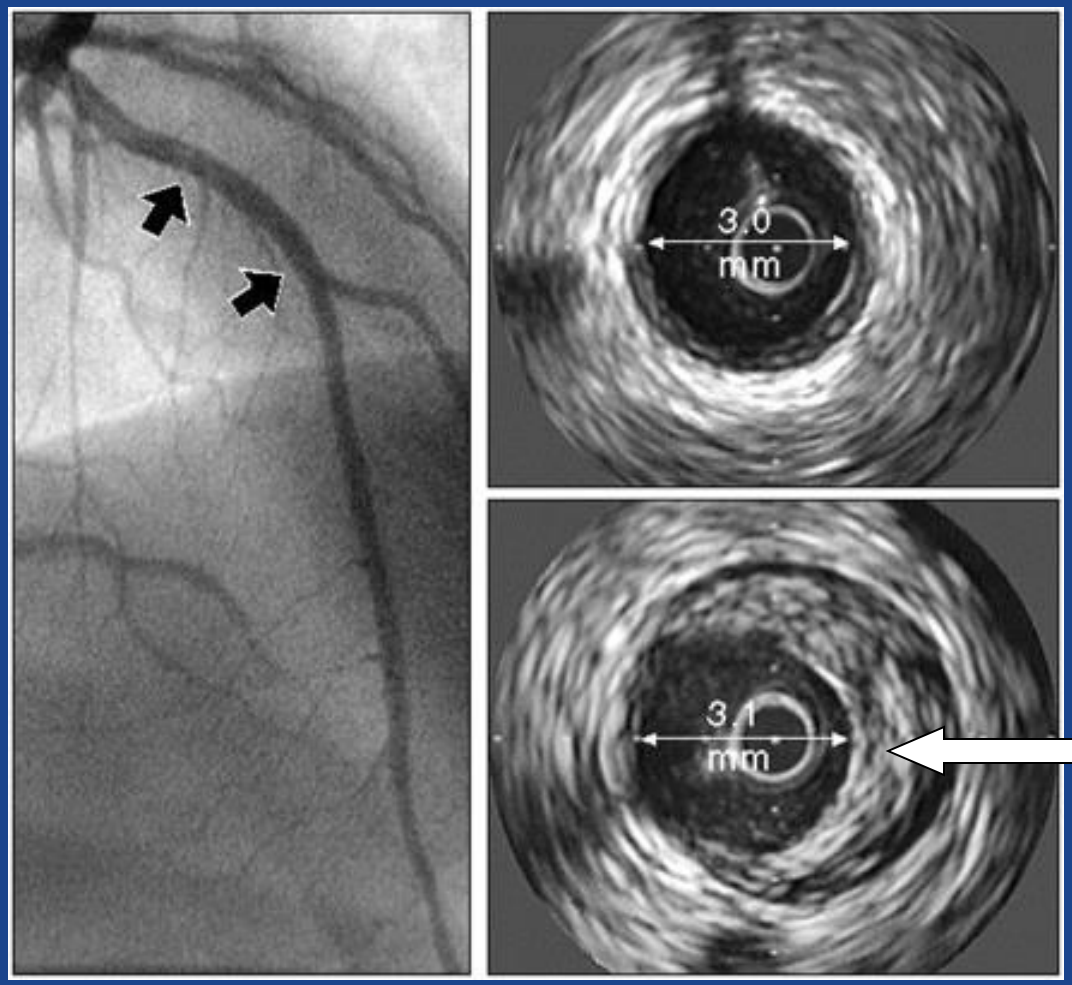
Invasive techniques

- Coronary angiography
- Intravascular ultrasound (IVUS)

Non-invasive techniques

- Magnetic resonance imaging (MRI)
- Computed tomography (CT)
- Ultrasound (B-mode)

Intravascular Ultrasound (IVUS) Showing Atheromatous Plaque



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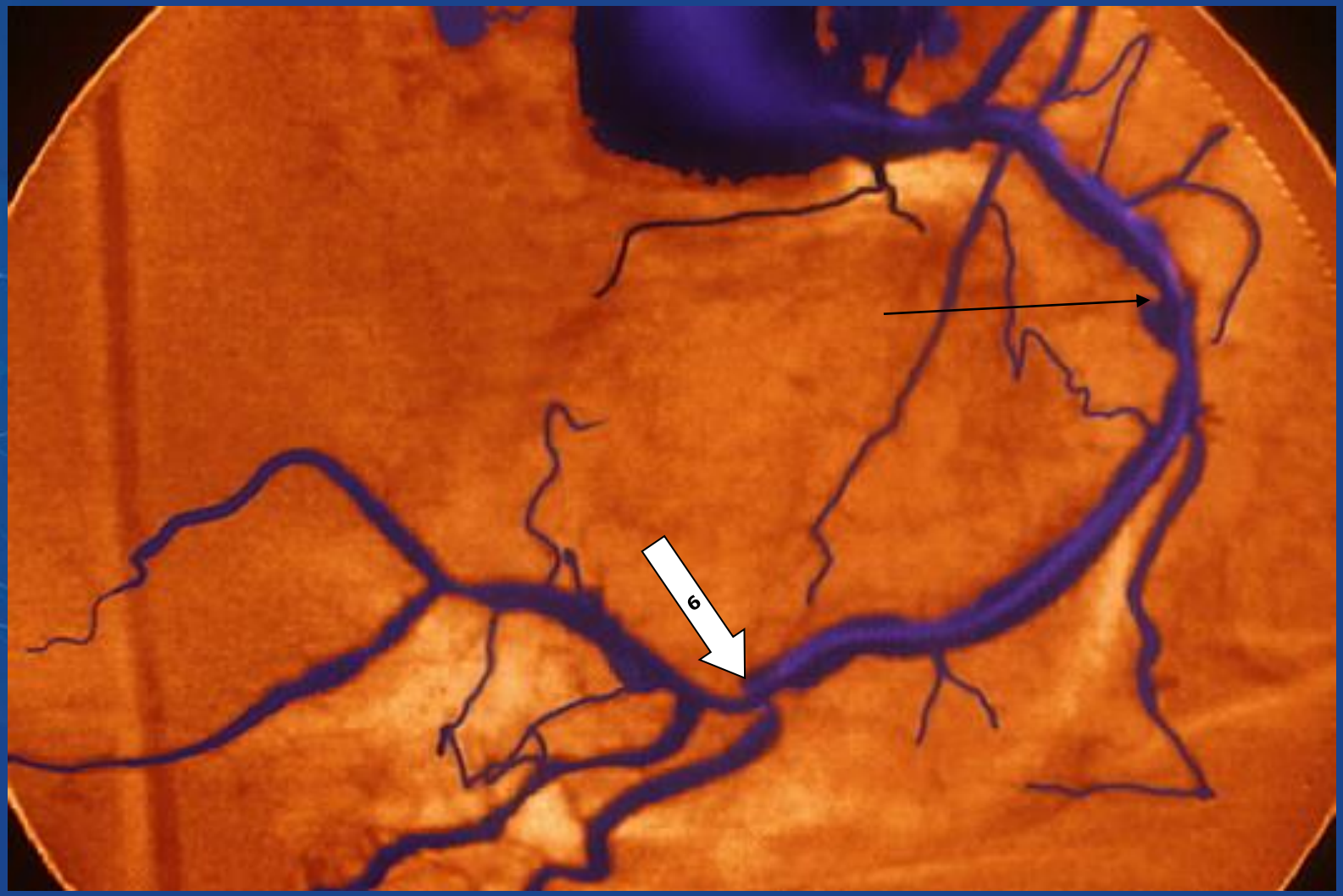
Coronary Angiography of Stenotic Coronary Artery



Arrow indicates atherosclerosis (stenosis) of the coronary artery

Management goals of stable angina

- **To improve prognosis (mortality reduction)**
  - Modification of risk factors
  - Aspirin
  - Lipid-lowering therapy
  - ACE-Inhibitor
  - Revascularization procedures (PTCA, CABG)
- **To decrease anginal symptoms**
  - Medical treatment

ACC/AHA Guidelines. *J Am Cardiol*. 1999;33:2092-2197.

ESC Guidelines. *Eur Heart J*. 1997;18:394-413.

Treatment of stable angina

1- General measures

2- Medical therapy: Increase O2 supply
Decrease O2 demand

3-Revasularization: PCI (percutaneous coronary intervension)
CABG (coronary artery bypass grafting)

TREATMENT OF STABLE ANGINA
General Measures

- Correction of established risk factors(reversible)
- weight reduction (ideal body weight)
- Areobic exercise:
  improve functional capacity, well-being sensation
- Treatment of: anemia, thyrotoxicosis, arrhythmias,..

MEDICAL THERAPY OF STABLE ANGINA

Prognostic: Aspirin, Statines, ACEI

Symptomatic: Nitrate,B-,CA-blocker, (nicorandil, ranolazine, ivabradine)

INCREASE O2 Supply

1-Increase diastolic time: B-blocker

2-Decrease coronary tone: nitrate, ca-blocker

3-Decrease LV diastolic pressure: nitrate

4-Correct coronary stenosis: PCI, CABG

5-Increase O2 capacity of blood: transfusion if anemia

DECREASE O2 Demand

1-Decrease heart rate: B-blocker, ca-blocker

2-Decrease contractility: B-blocker, ca-blocker

3- Decrease wall tension (LV pressure and cavity radius): nitrate

4- metabolic: trimetazidine

Treatment in practice (Medical, PCI, CABG)

1-General measures

2-Aspirin

3-Nitrate: S/L, Oral, dermal

3-B-blocker

4-Statins: LDL>100 mg/dl(70mg/dl)

5-Ca-blocker

6-Angio :PTCA,CABG

Case presentation

A50 year old male presented to emergency room complaining of sudden sever chest pain of 1 hour duration. It is retrosternal, compressive, and radited to left shoulder and arm.

Associated with sweating, nausea and vomiting

On examination: patient is anxious, in pain, sweaty.

BP: 100/60. PULSE: 120 BPM, RR: 26/min

Chest: basal crepitations

What is the most likely diagnosis?

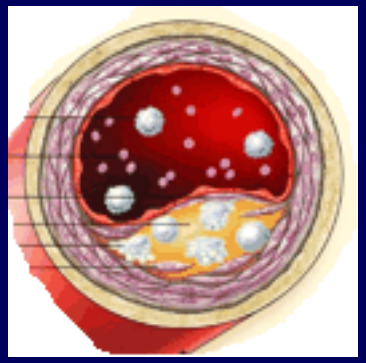
MYOCARDIAL INFARCTION

pathophysiology

Symptoms of Angina

Angina can spread anywhere between the belly button and the jaw, including to the shoulder, arm, elbow or hand- usually on the left side.

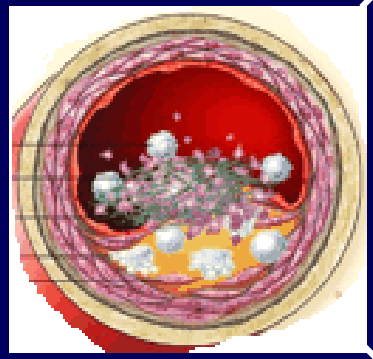
Pathogenesis of ACS



Vulnerable Plaque

Plaque rupture (55-80%)

Exertion
BP, HR
Vasoconstriction



✓Platelet aggregation
✓Thrombus formation
✓Vasospasm

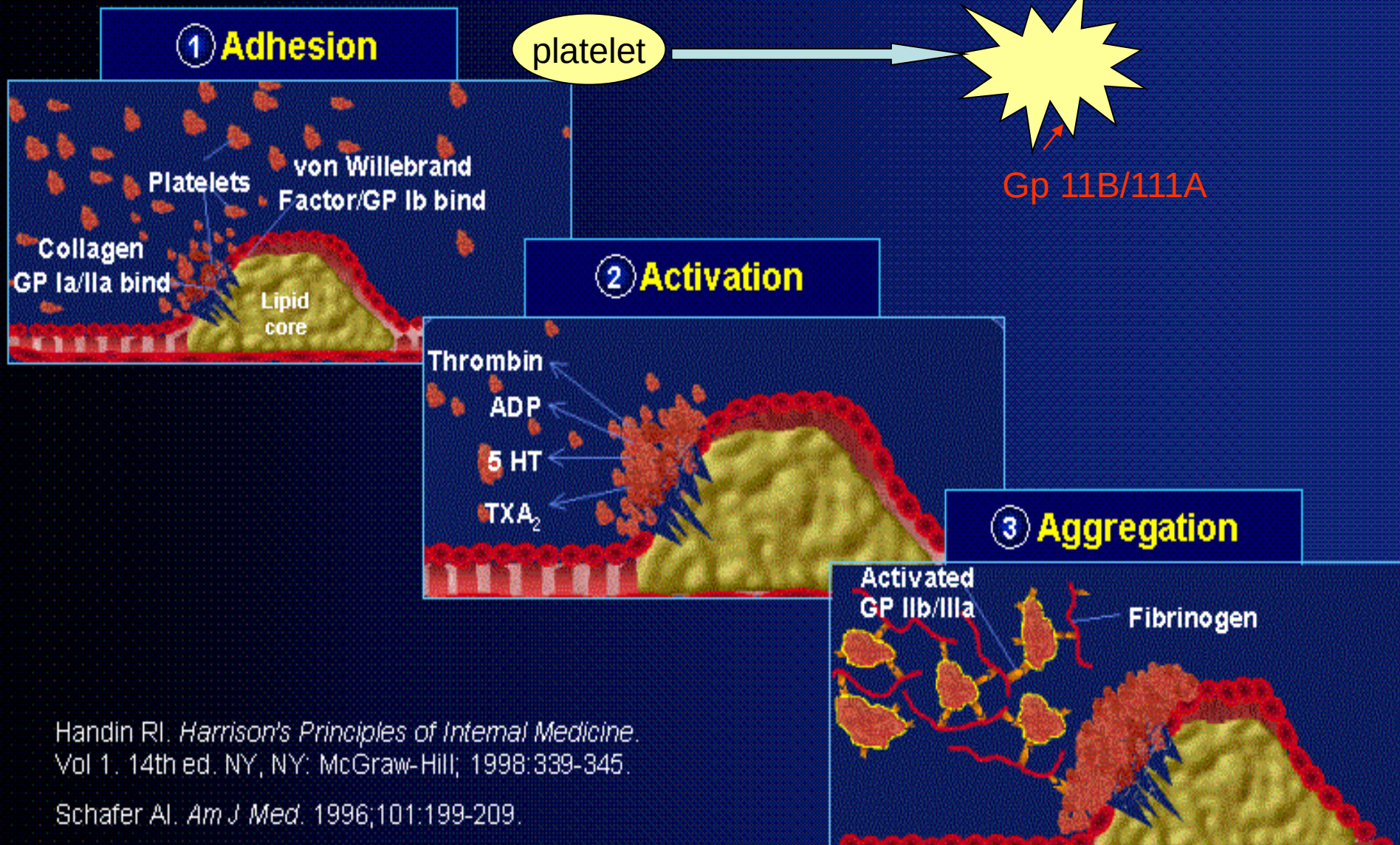
Incomplete occlusion
Distal embolization

Unstable angina
NSTEMI

Complete occlusion

STEMI

Platelet Cascade in Thrombus Formation



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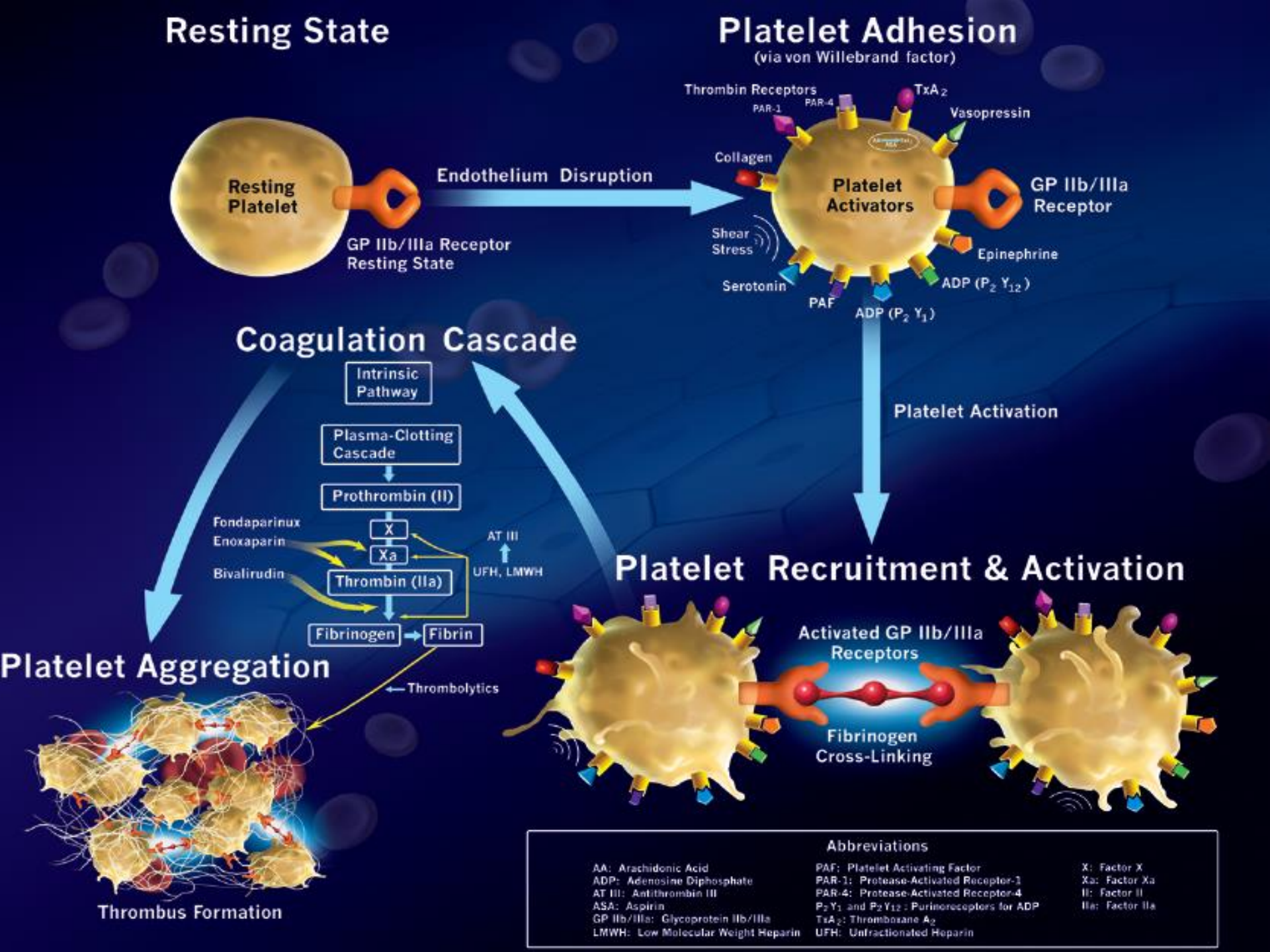
Schafer AI. *Am J Med*. 1996;101:199-209.


Resting State
Platelet Adhesion
(via von Willebrand factor)
Thrombin Receptors
PAR-1
PAR-4
TxA2
Vasopressin
Collagen
Resting Platelet
Endothelium Disruption
Platelet Activators
GP IIb/IIIa Receptor
GP IIb/IIIa Receptor Resting State
Shear Stress
Epinephrine
Serotonin
ADP (P2 Y12)
PAF
ADP (P2 Y1)
Coagulation Cascade
Intrinsic Pathway
Plasma-Clotting Cascade
Prothrombin (II)
Fondaparinux
Enoxaparin
X
Xa
AT III
Bivalirudin
Thrombin (IIa)
UFH, LMWH
Fibrinogen
Fibrin
Platelet Activation
Platelet Recruitment & Activation
Activated GP IIb/IIIa Receptors
Fibrinogen Cross-Linking
Platelet Aggregation
Thrombolytics
Thrombus Formation
Abbreviations
AA: Arachidonic Acid
ADP: Adenosine Diphosphate
AT III: Antithrombin III
ASA: Aspirin
GP IIb/IIIa: Glycoprotein IIb/IIIa
LMWH: Low Molecular Weight Heparin
PAF: Platelet Activating Factor
PAR-1: Protease-Activated Receptor-1
PAR-4: Protease-Activated Receptor-4
P2Y1 and P2Y12 : Purinoreceptors for ADP
TxA2: Thromboxane A2
UFH: Unfractionated Heparin
X: Factor X
Xa: Factor Xa
II: Factor II
IIa: Factor IIa

Diagnosis of Myocardial Infarction

1-History

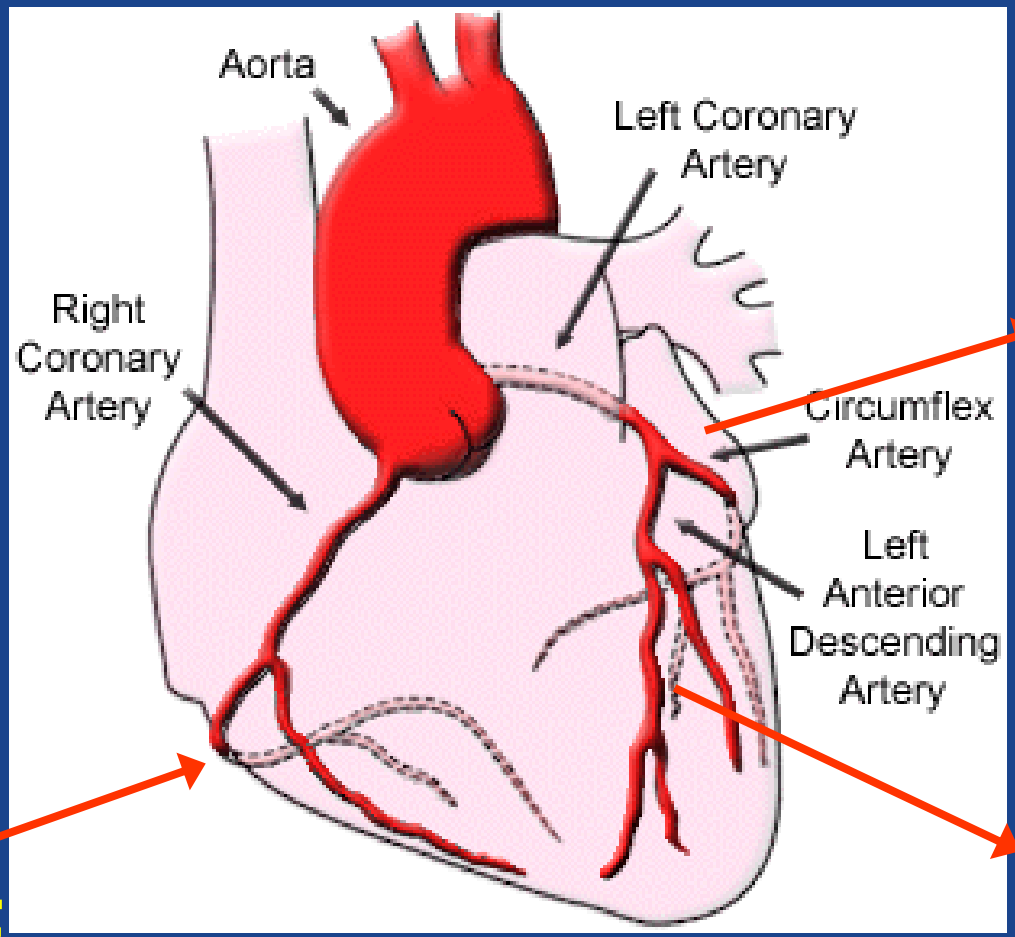
2-ECG (Electrocardiogram): STMI and NSTMI

- **Hyperacute T wave**
- **ST-segment elevation**
- **Q- wave**
- **T- inversion**
- **ST-segment depresion**
- ***normal ECG will not exclude MI***

3-Cardiac Marker: Troponin,CPK, myoglobulin,..

- **Troponin T,I: 4-6 Hr**
  - **last 10-14 days**
- **CPK:4-6 Hr, peak 17-24hr, normal 72 hr**
  - **MB(MM,BB)**
  - **MB2/MB1 >1.5**

Regions of the Myocardium

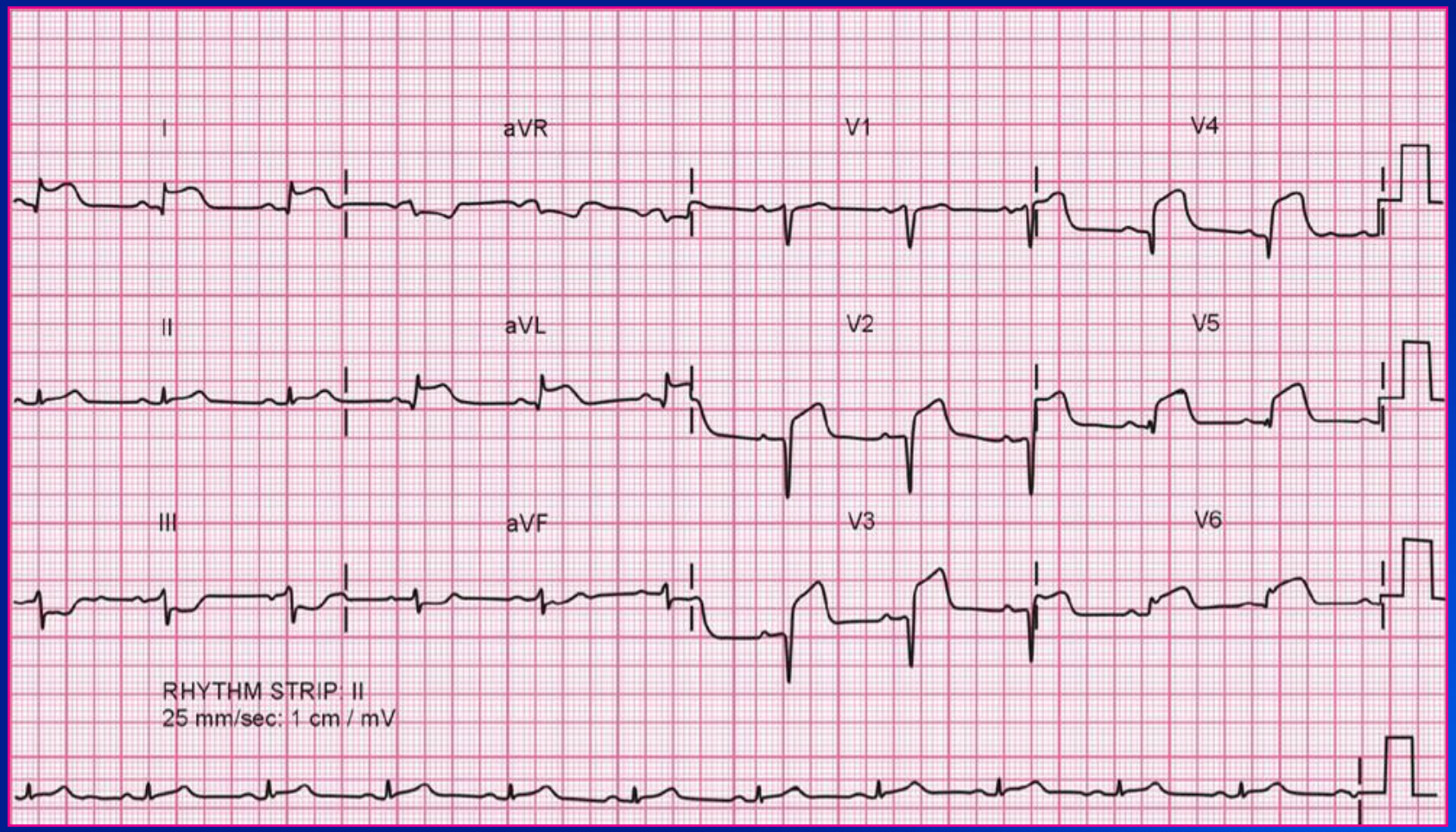


Lateral
I, AVL, V5-V6

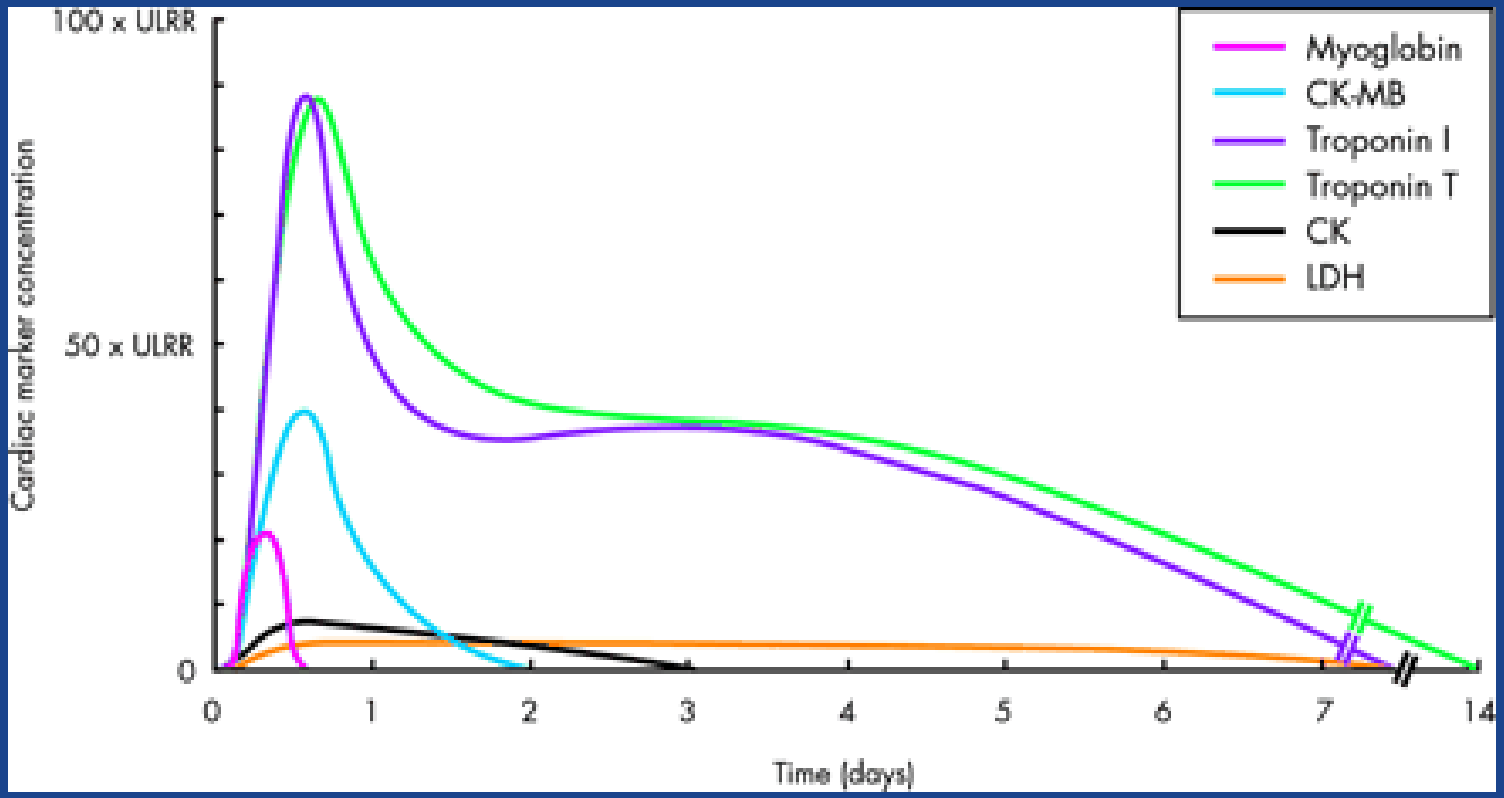
Anterior / Septal
V1-V4

Inferior
II, III, aVF

WHAT IS THE DIAGNOSIS?
Anterior ST-segment elevation(V1-V6)



Biochemical cardiac Markers



TREATMENT OF MYOCARDIAL INFARCTION

IN EMERGENCY ROOM:

1-Rapid assessment

2-Establish IV access

3-12 ECG

4- Aspirin 150-300 mg Orally, Clopidogrel

5-Oxygen

6-Analgesia: IV morphine, diamorphine 3-5 mg

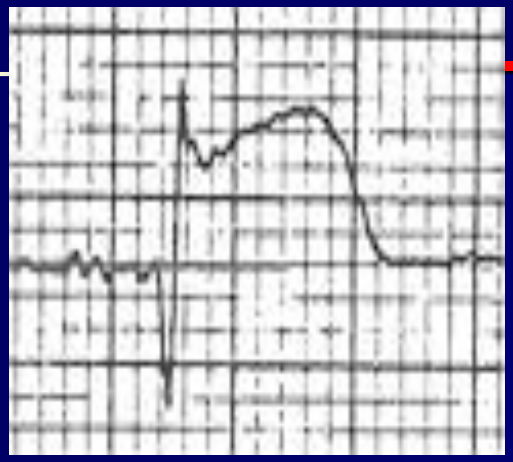
7-Antiemetic: metoclopromide 10 mg IV

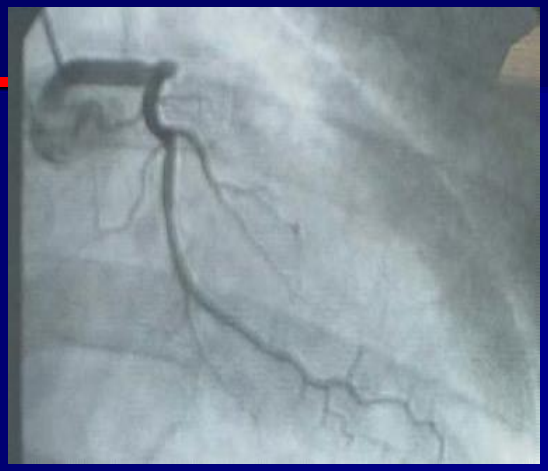
8-Sublingual nitrate: if NO hypotension, RV MI

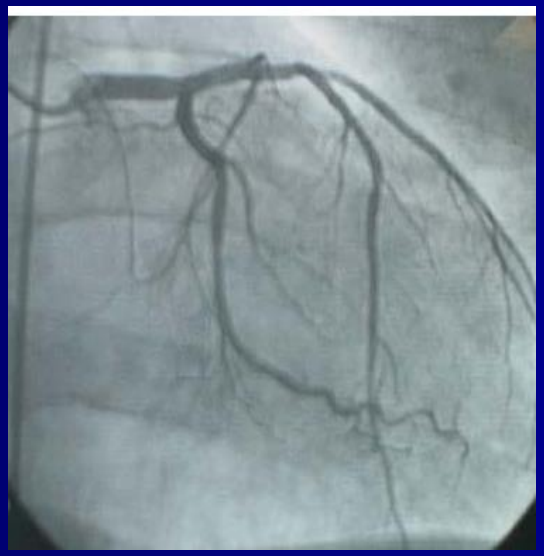
9-ECG monitor

10-Reperfusion: PCI or Thrombolytics, (CABG)

Reperfusion in STEMI







Complications of Myocardial infarction

4 WEEKS LATER PATIENT PRESENTS C/O SHORTNESS OF BREATH ON EXERTION ASSOCIATED WITH ORTHOPNEA AND PND.

WHAT DO YOU THINK?

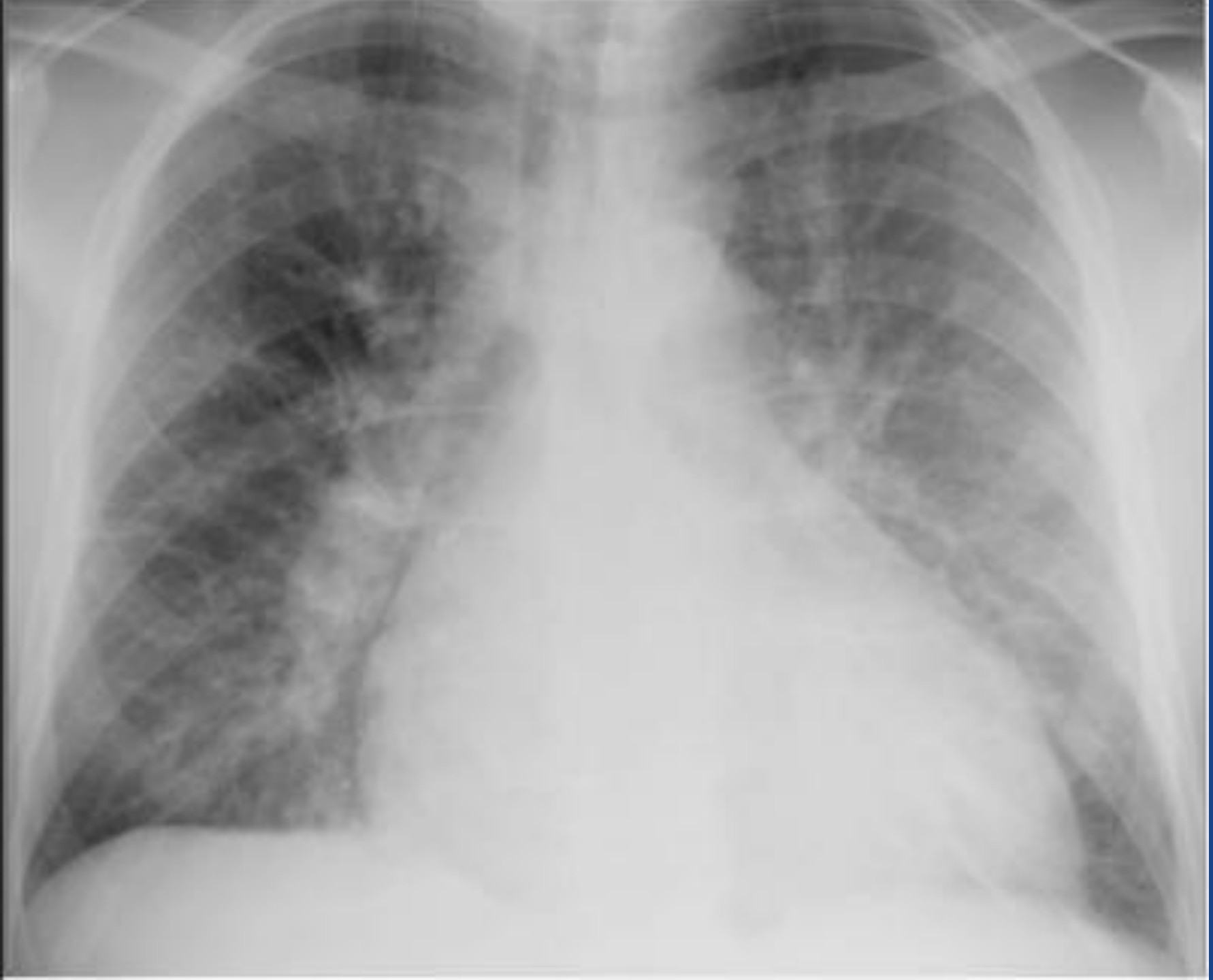
O/E: RAISED JVP

BILATERAL BASAL LUNG CREPITATION

LL EDEMA

WHAT IS THE DIAGNOSIS? Left heart failuire

WHAT INVESTIGATIONS? Chest x-ray, echo



Medical Management of Heart Failure

Drugs that improve symptoms

furosemide
thiazide diuretics
spironolactone
digoxin
ACE Inhibitors
beta blockers
aldosterone antagonists
vasodilators

Drugs that improve prognosis

ACE inhibitors, (ARB)
beta blockers
spironolactone*
Digoxin
amiodarone

Thank you