CVS

PRACTICAL

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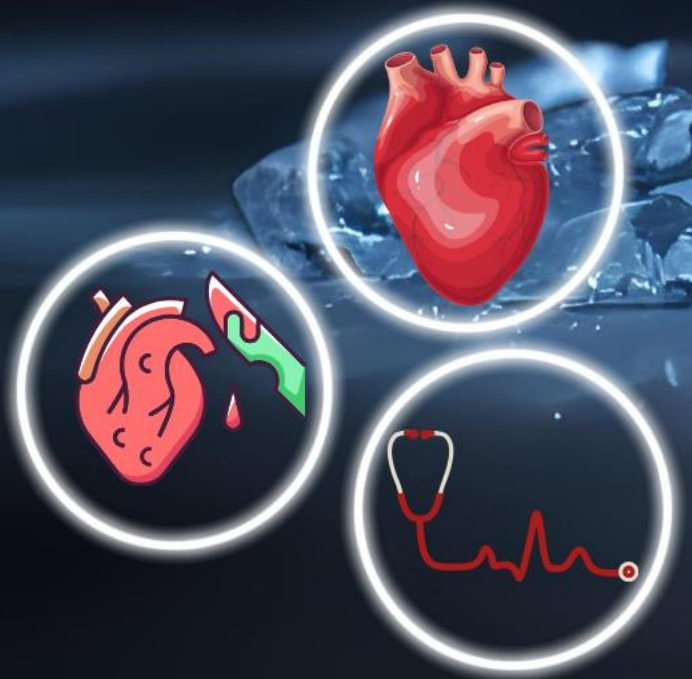

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Common Clinical Cardiology Scenarios

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Color code

Slides
Doctor
Additional info
Important

Case 1

- **History**:
- A 65-year-old gentleman presents to the ED/Emergency Department with crushing retrosternal chest pain of 2 hours duration.
- PMH/past medical history/risk factors:DM, HTN and dyslipidemia.
- Meds: Insulin, metformin, enalapril, atorvastatin.
- **Exam**: Apprehension, diaphoretic and in severe pain (impending doom)
- V/S: BP 90/50mmHG, HR 110/min
- CV: Normal S1, S2 no murmur

For his hypertension/HTN, an ACE inhibitor

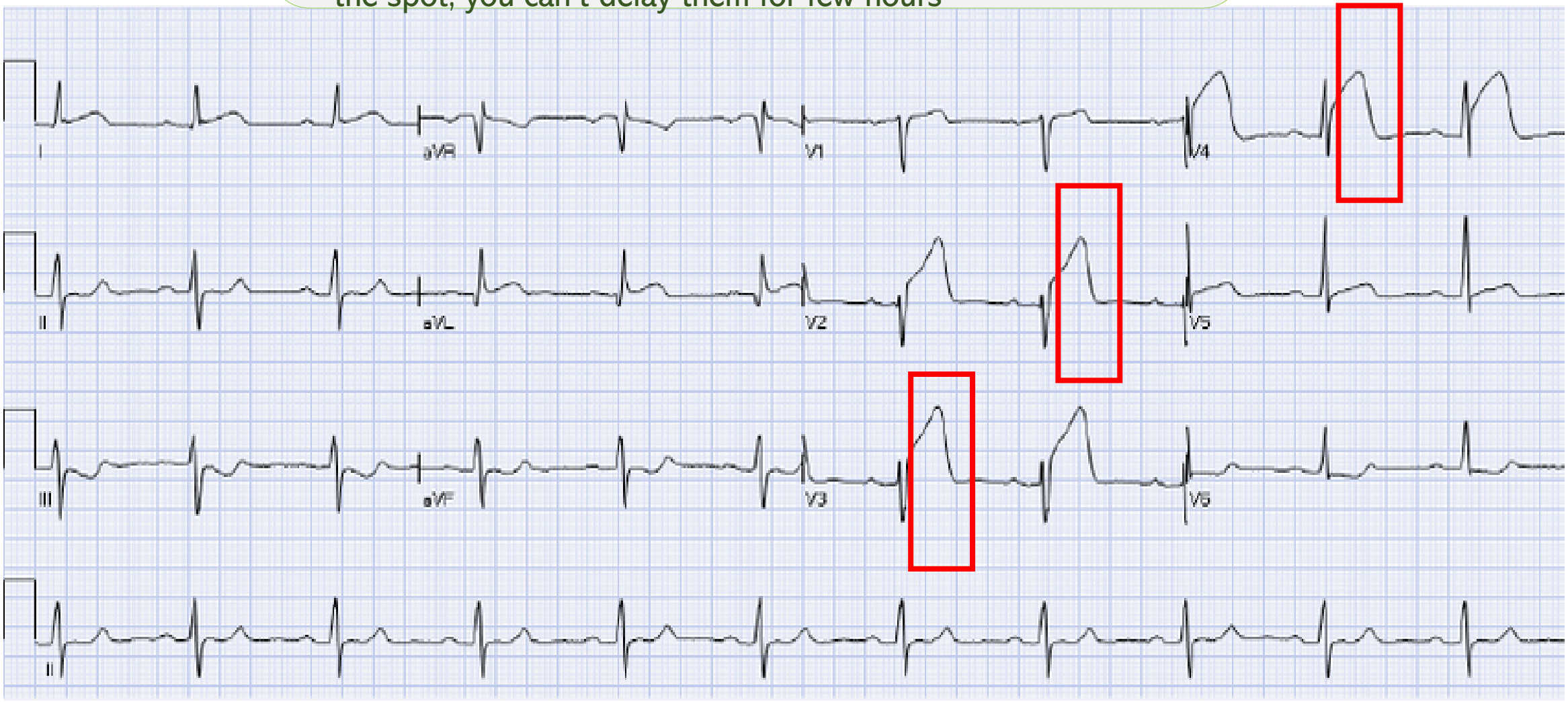
For his dyslipidemia

We notice from this information, the patient is hypotensive and tachycardic.

What is the NEXTSTEP?

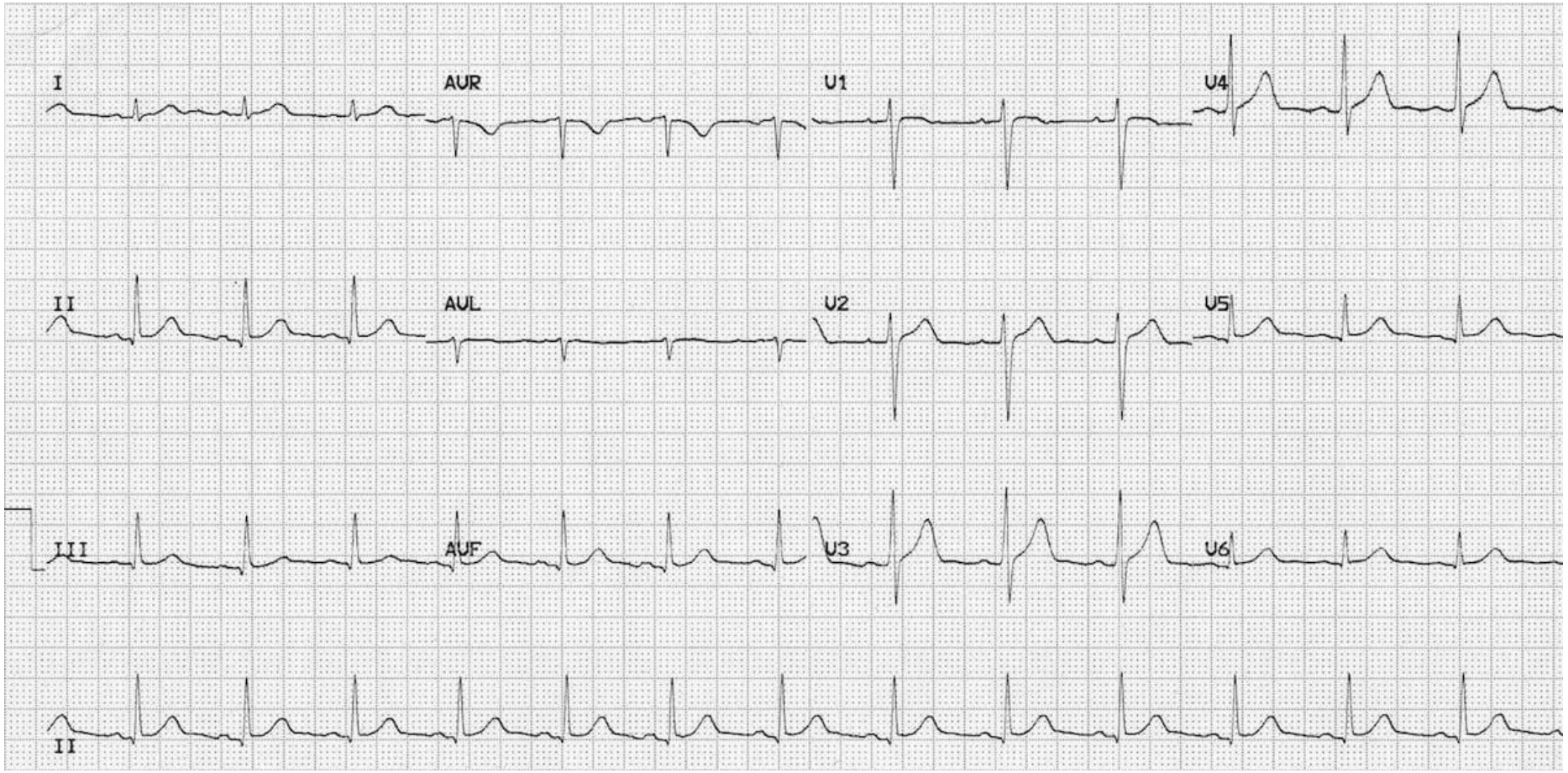
ECG

- The answer is: ECG.
- We can see the ST elevation: elevation of ST segment appear after QRS ,it appears in V2, V3 and V4 abviously.
- This is a common clinical scenarioe in emergency departments, and has a high mortality rate , yo-yo have to deal with them in the spot, you can't delay them for few hours



Nomal ECG

Compare the previous ECG with this normal ECG, which has no ST elevation, specifically in V2, V3 and V4.



Diagnosis:STEMI

- Transmural myocardial ischemia and subsequent myocardial injury or necrosis.
- Life threatening condition with high mortality
- Risk factors include: hypertension, hyperlipidemia, smoking, and diabetes which are very common in our Jordanian population.
- The pathogenic mechanism typically involves plaque rupture and thrombus formation within the coronary artery
- Diagnosis :ECG and confirmed by elevation in cardiac biomarker Troponin

STE: Refers to the ST (segment) Elevation on an ECG as we saw.

Myocardial Infarction (MI): Refers to heart muscle damage caused by insufficient blood supply.

STEMI

What happened in detail?

1) Plaque formation in the vessel wall of the coronary artery, which is a cholesterol deposition at one point in a time. It can be triggered by factors such as physical exertion, or emotional stress, or may occur spontaneously without an identifiable cause.
2) When a plaque ruptures, the lipid core of the plaque is exposed to the bloodstream. This exposure triggers the coagulation cascade and leads to platelet aggregation at the site of the rupture leading to thrombus formation too.
3) The vessel will be occluded leading to cessation /prevent blood flow to certain area of the heart=insufficient blood supply = schemia.
4) That leads to heart damage= MI.

Management

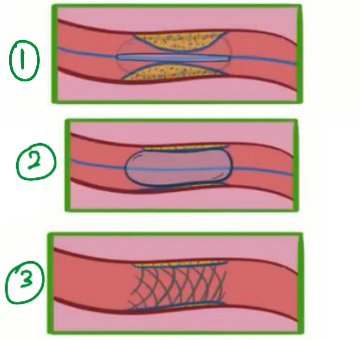
- Immediate treatment involves restoring blood flow to the affected area through reperfusion therapy
- Typically, via percutaneous coronary intervention. Early management is critical to limit myocardial damage, and adjunctive therapies, including antiplatelets and anticoagulants.
- Alternative therapy is thrombolytics

You need to restore the blood flow to the heart(this is our target), either by:
1) Mechanical/non pharmacological therapy.
As: percutaneous coronary intervention= stent.

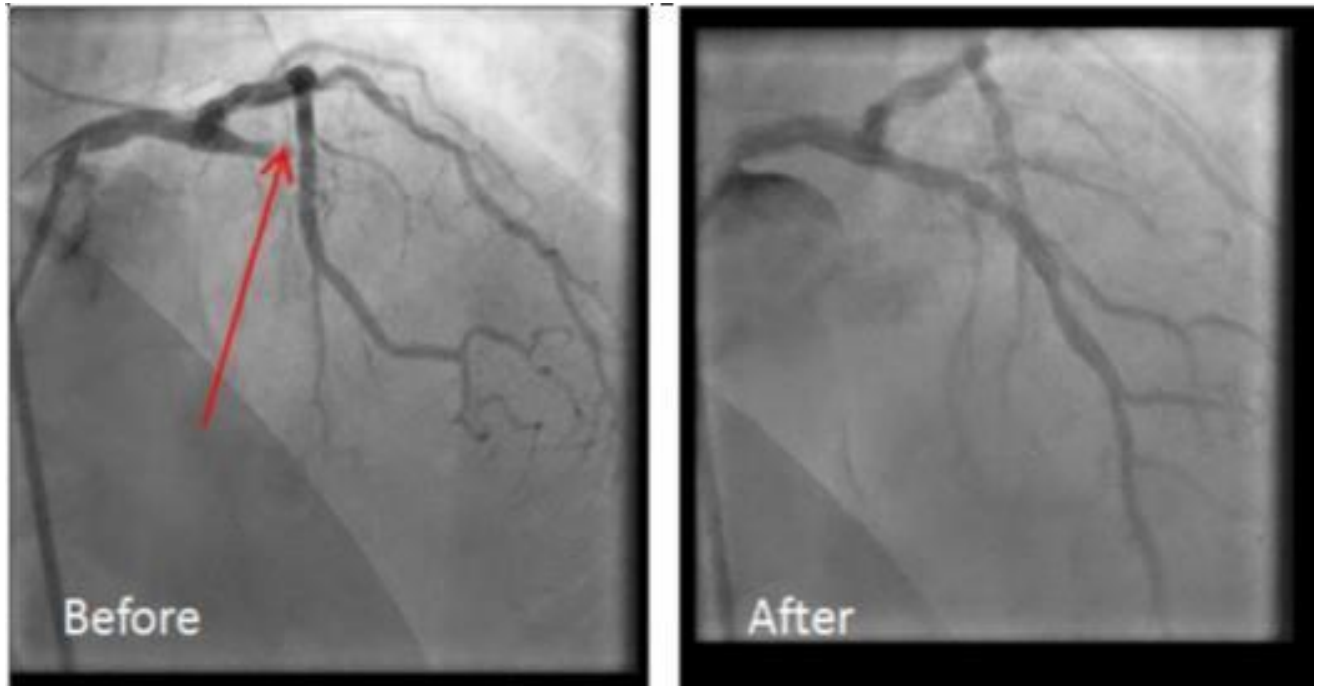
1) Pharmacological therapy.
As: Thrombolytics that bust/destroy the thrombus.

PCI

PCI stands for Percutaneous Coronary Intervention.It is a minimally invasive procedure used to open blocked or narrowed coronary arteries to restore blood flow to the heart.



- The red arrow indicates to where the blood is stopped due to thrombus.
- Once the coronary artery is recanalized by PCI, the blood flow will be restored.
- You can notice that the the vessels reach to the base, so this is the left anterior decessnding artery which was presented with anterior lead ST from V2 to V4



Case-2

This patient with no risk factors, but his symptoms are similar to previous scenario.

- **History:**
- A 20-year-old college student who is previously healthy present with sudden onset sharp retrosternal chest pain that is exacerbated by inspiration and laying in supine position but improves with leaning forward.
- Recent history of respiratory tract infection two weeks ago.
- **Exam:**

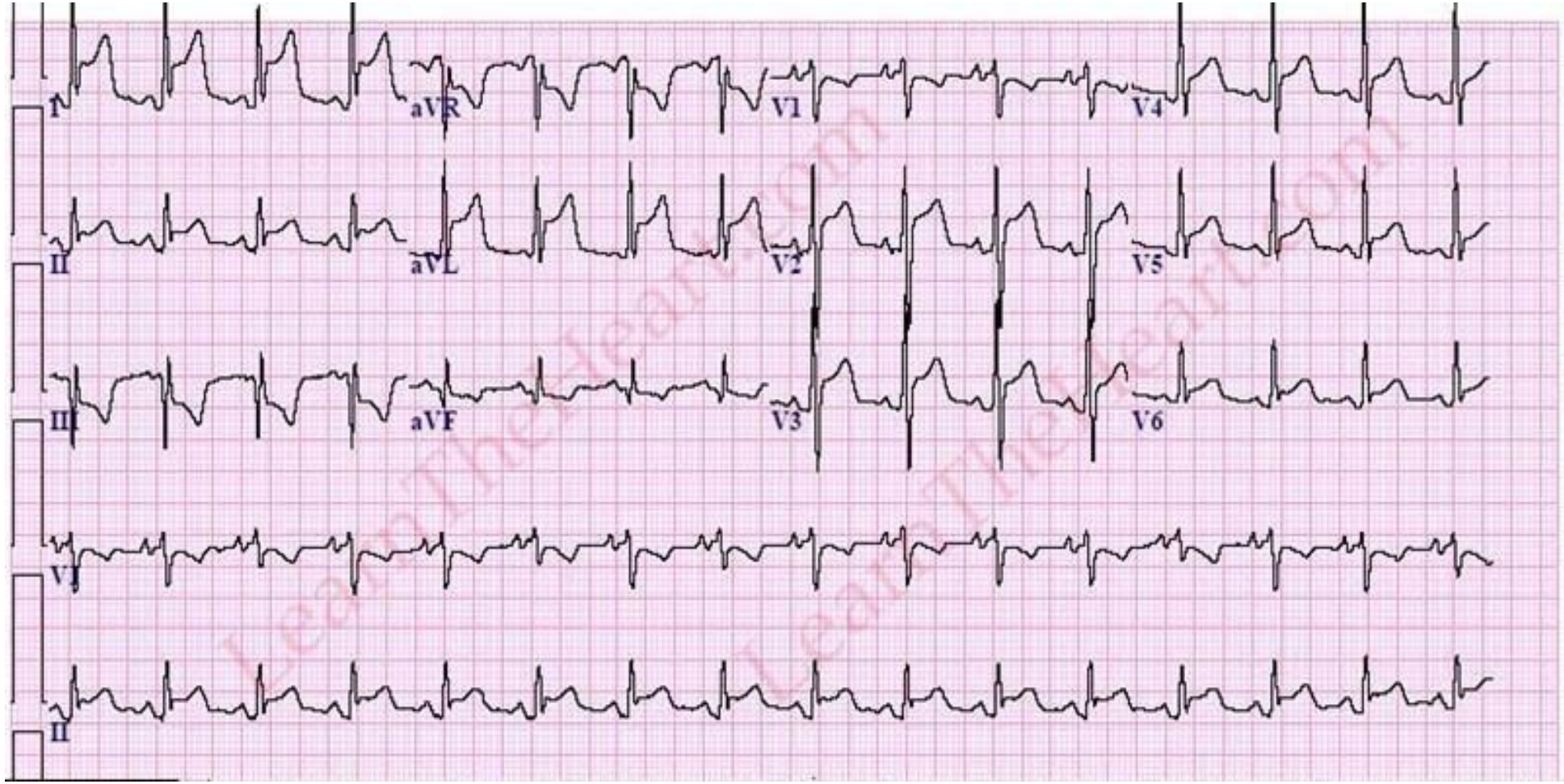
V/S :BP 120/80mmHg, HR 90/min his examination is quite unremarkable

CV: squeaking sound best heard in the left parasternal area (friction rub)

What is the NEXTSTEP?

ECG

- The answer is: ECG
- We can see diffused ST elavation, not localised.



Pericarditis

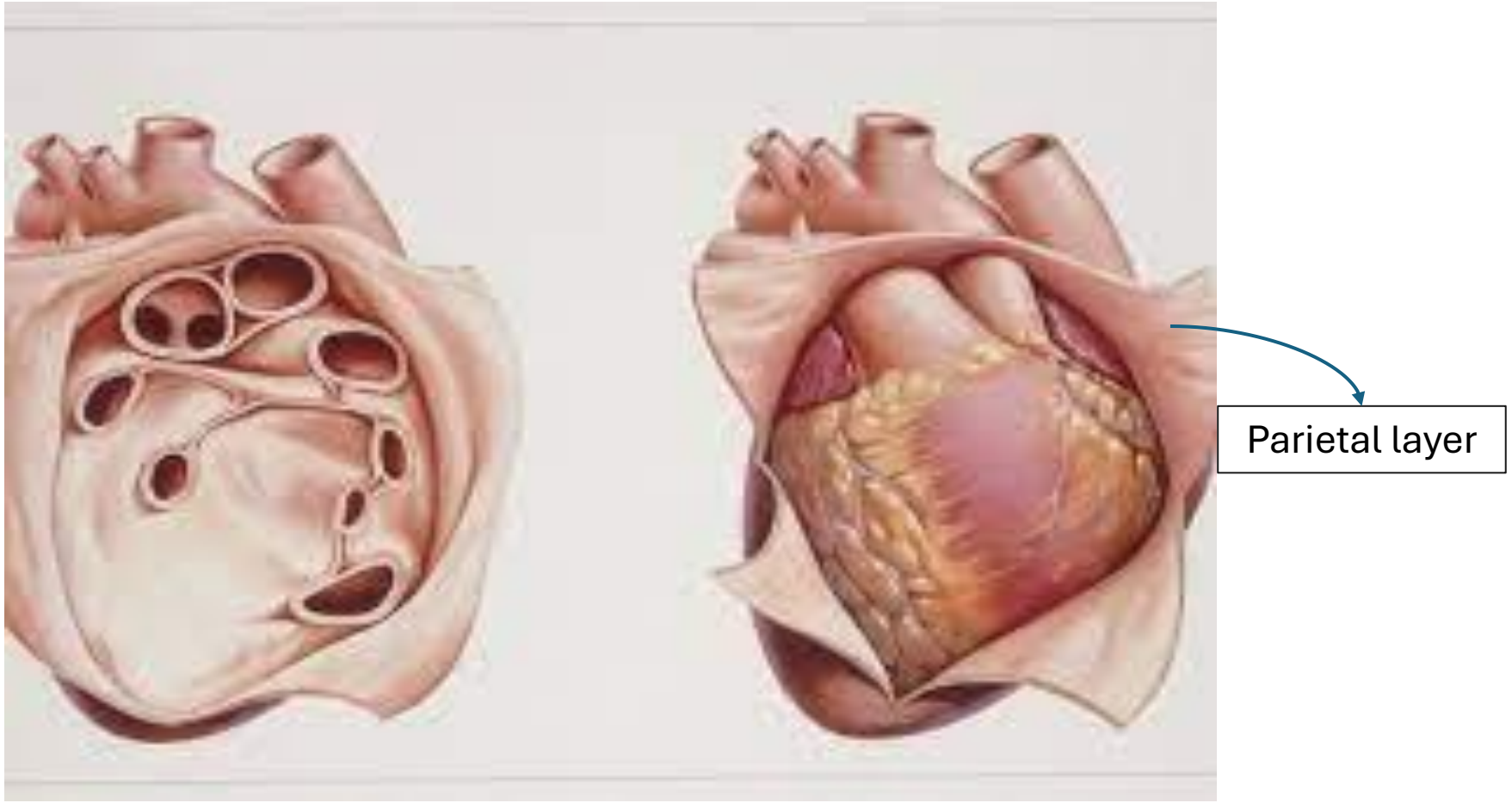
- The patient has pericarditis, inflammation of pericardium.
- The normal pericardium is composed of two layers: the visceral layer (which sits directly on the heart) and the parietal layer (a fibrous outer layer). Between these layers, there is a small amount of lubricating fluid to reduce friction during the heart's movements.
- When these two layers are inflamed, friction increases, causing pain and a characteristic squeaking or rubbing sound, known as a pericardial friction rub.

- **I-Chest pain:**
- The vast majority of patients with acute pericarditis present with chest pain (>95% of cases).
- Chest pain that results from acute pericarditis is typically fairly sudden in onset and occurs over the anterior chest.
- Chest pain due to pericarditis is most often sharp and pleuritic in nature, ***with exacerbation by inspiration or coughing.***
- One of the most distinct features is the **tendency for a decrease in intensity when the patient sits up and leans forward.**

Pericardium

- **Introduction:**
- The pericardium is a fibroelastic sac made up of visceral and parietal layers separated by a space, the pericardial cavity.
- In healthy individuals, the pericardial cavity contains 15-50 mL of an ultrafiltrate of plasma.

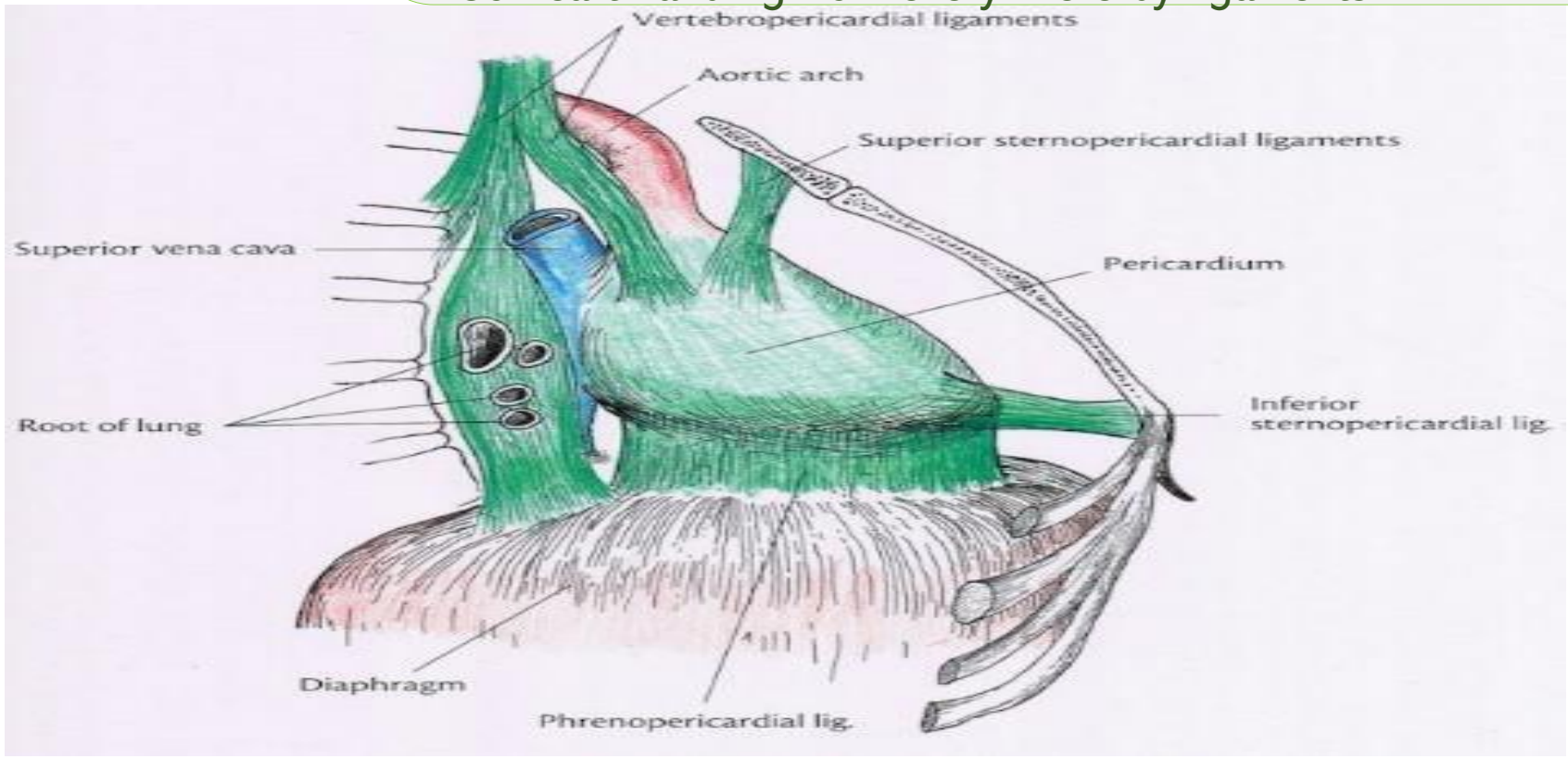
Pericardium



Pericardium

This is how the heart looks in mediastinum:

- The heart is connected through the pericardium with different structures by ligaments as:
- Posterior of the heart: vertebropericardial ligament
- Bottom of the heart: phrenicopericardial ligament
- Anterior: superior and inferior sternopercardial ligament
- So heart handing from everywhere by ligaments



Pericarditis

So the major criteria for diagnosing of such patients that you have the typical presentation (what the patient describes about the nature of the pain)

- Chest pain — typically sharp and pleuritic, improved by sitting up and leaning forward.
- Pericardial friction rub — a superficial scratchy or squeaking sound best heard with the diaphragm of the stethoscope over the left sternal border.
- Electrocardiogram (ECG) changes — new widespread ST elevation and PR depression
- Pericardial effusion.

We can see it in echocardiogram

Treatment

- For most patients with acute idiopathic or viral pericarditis, combination therapy: colchicine plus NSAIDs rather than NSAIDs alone.
- This is based upon a reduced rate of recurrent pericarditis and a low incidence of side effects with colchicine.
- Steroids are second line; the patient has side effect or allergic to NSAIDs .No response to NSAIDs

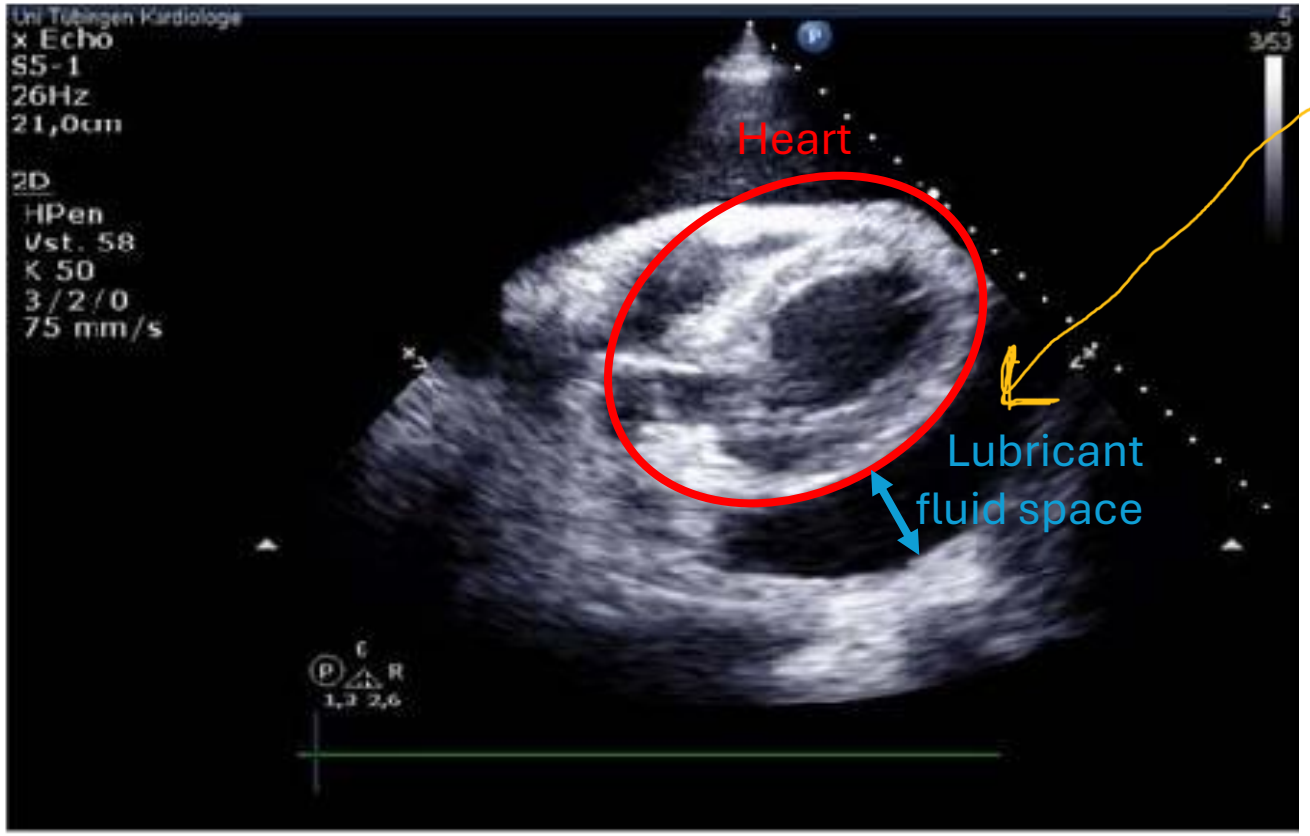
No emergency can lap, no thrombolytics therapy, this type of management is harmful for pericarditis patient.
So, what do you do?
We use anti inflammatory drugs as NSAIDs as the 1st line therapy (familiar Naproxen, ibuprofen) and colchicine (which is drug to gout , but it has anti inflammatory effects which proved to decrease the recurrence rate of pericarditis) .
How?
Give NSAIDs for 2 weeks nd colchicine for 3moths.

Feared complication

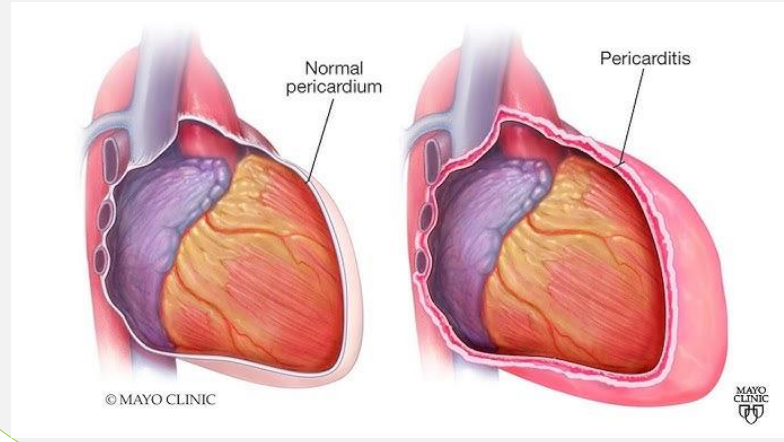
Also pericardial effusion lead to other complication called tamponade which clinically diagnoses by muffled heart sounds (meaning it is softened or unclear) and hypotension and elevated **JVP (jugular venouspressure)**

This is the Beck's triad for diagnosis

Echocardiogram



- An abnormal space develops between the two layers of the pericardium, often accumulating up to 2 liters of fluid, which is far more than the normal amount.
- This space shouldn't appear in normal echocardiogram.



Case-3

- **History:**

A 60-year-old lady with history of dyspnea, orthopnea and PND's (paroxysmal nocturnal dyspnea) of 3 weeks duration.

PMH: DM, HTN, CAD-CABG

Exam:

V/S: BP100/60mm HG, HR 95/min

CV: S3 sound, raised JVP

Lungs: crackles

LE: pitting edema

Notes about previous slide

- PND happens when someone sleep with no problems but wake up around 3-4 am with difficulty in breathing
- While orthopnea happens when someone cannot breath normally (shortness in breath) when he sleep on his back (spinal position)
- Orthopnea is worse (bcz. any lining in supine position make the short of breath)since it indicates high central venous pressure once man sleep on his back while PND it takes several hours for the central venous pressure to be high
- Both are indecators for heart failure

- Our patient's vital signs here are acceptable
- S3 sound is a diastolic sound (low-pitched), it's an early filling sound. It is normal in youngsters but above the age of 35/40 becomes pathological. S4 sound always is pathological !!
- Crackles (the sound of fluid that build up inside alveoli, which is the cause of shortness of breath due to impair gases exchange , so the patient comes dyspneic and hypoxic)
- heart failure, patients normally have volume overload in general so they have pitting edema in lower extremities

What is the NEXTSTEP?

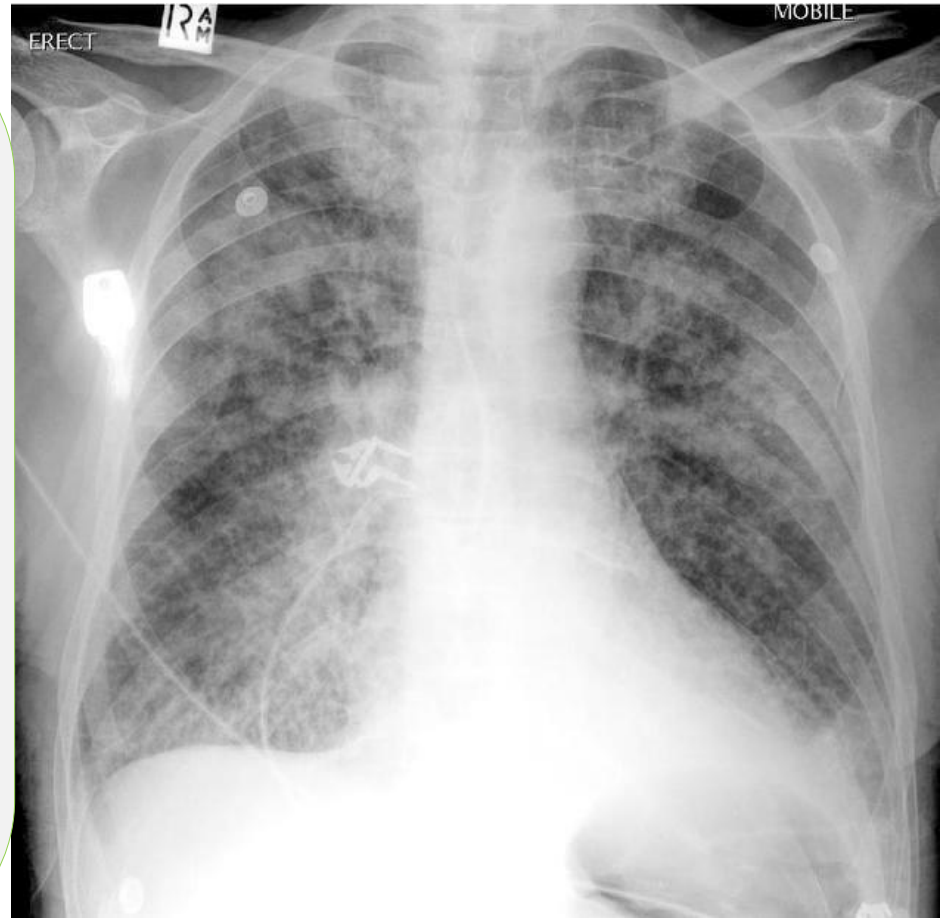
CXR (congestive heart failure) and BNP Level

- This X-ray is congested so I see here fluffy infiltrates, indicating pulmonary edema

- Sometimes we get confused is this x-ray is pneumonia or heart failure so to confirm we ask for “brain natriuretic peptide” test

- Brain: because it was discovered in animals brain , but it is released from myocytes when they are stretched & weak
- So, having a high BNP indicates indicates patient is in heart failure with very high sensitivity while negative BNP indicates this is Not heart failure with very high specificity
- So this is a highly valuable serum marker for heart failure

- This serum bio marker is very visible in our management



Heart Failure Definition

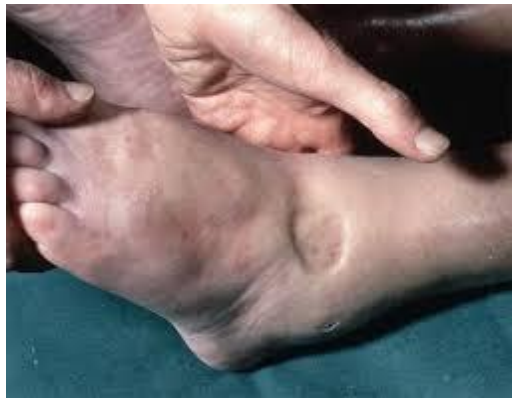
- Doctor read this box

Heart failure (HF) is a clinical syndrome in which patients have **typical symptoms and signs** resulting **from** an **abnormality of cardiac structure or function** which **impairs the ability of the ventricle to fill with or eject blood**.

- **symptoms** (e.g. breathlessness, orthopnea, paroxysmal nocturnal dyspnoea, ankle swelling, fatigue, and reduced exercise tolerance)
- **signs** (e.g. elevated jugular venous pressure, hepatojugular reflux, third heart sound [gallop rhythm], S4 sound, edema & scrotal edema in male cardiac murmur, and displaced apex beat)

Signs

Pitting edema



Weight gaining

⬆️ JVP

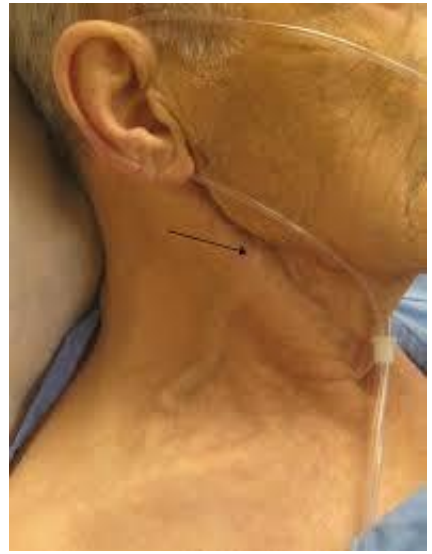
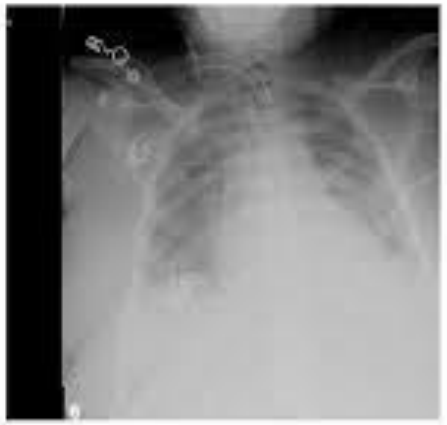
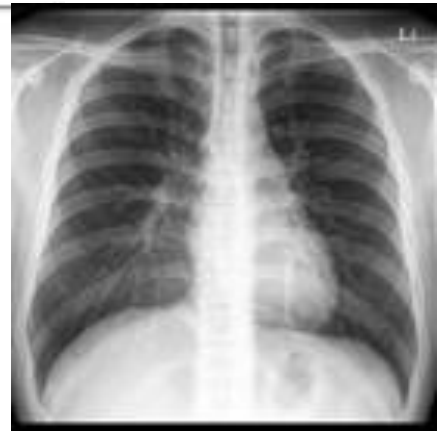


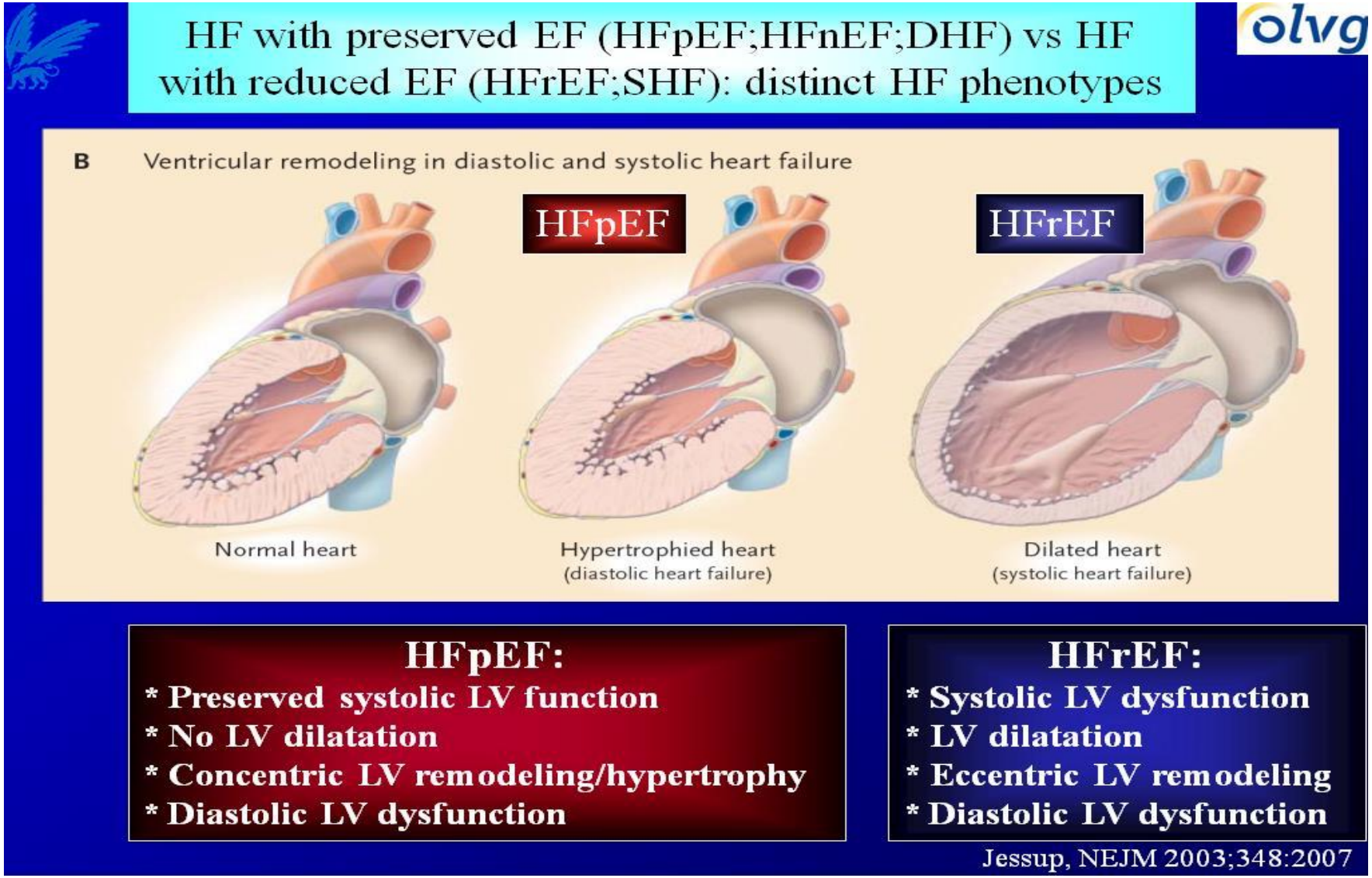
Figure 24. CXR Showing Acute Decompensated Heart Failure





X-ray with heart failure compared to a normal x-ray

Sub-types (Echocardiogram)



- Notes about previous slide

- There is two main sub types :
- -reduced ejection fraction (HFrEF : heart failure with reduced ejection fraction)

- – preserved ejection fraction (HFpEF: heart failure with preserved ejection fraction)

- They present the same **So we distinguish between them by echo**

- The previous image shows the normal heart with normal thickness, chamber lumen and Lt. atrium
- In HFpEF, heart wall is thickened, chamber lumen is compromised & Lt. atrium is dilated
- In HFrEF, lumen is severely dilated and Lt. atrium is dilated too.

Classification of Heart Failure

- This helps us to know how bad patient situation is

Functional Classification (New York Heart Association [NYHA])

Class	Severity of symptoms and limitation of physical activity
I	**No limitation** of physical activity Ordinary physical activity does not cause symptoms of HF (breathlessness, fatigue, or palpitations)
II	**Slight limitation** of physical activity Comfortable at rest, but **ordinary physical activity results in symptoms of HF**
III	**Marked sever limitation** of physical activity Comfortable at rest, but **less than ordinary physical activity causes symptoms of HF***
IV	**Unable** to carry on any physical activity without discomfort/symptoms of HF, or symptoms of HF at rest may be present At rest

Here patient has minimal exertion
(يعني بيتعب لما يتحرك من الغرفة للمطبخ)

Classification of Heart Failure

- Keep in mind HF is the most cause of hospital administration after the age of 65

• Heart Failure Staging

Stages of HF	Development and progression of HF	Corresponding NYHA Class
A	At **high risk** for HF but **without structural heart disease or symptoms** of HF	None
B	**Structural** heart disease but **without signs or symptoms** of HF	I
C	**Structural** heart disease **with prior or current symptoms** of HF	I
		II
		III
D	**Refractory HF** requiring specialized interventions	IV

Stages of HF	Development and progression of HF	Corresponding NYHA Class
A	At **high risk** for HF but **without structural heart disease or symptoms** of HF	None

- Patient with History of DM hypertension & smoker he at risk of developing heart failure due to MI

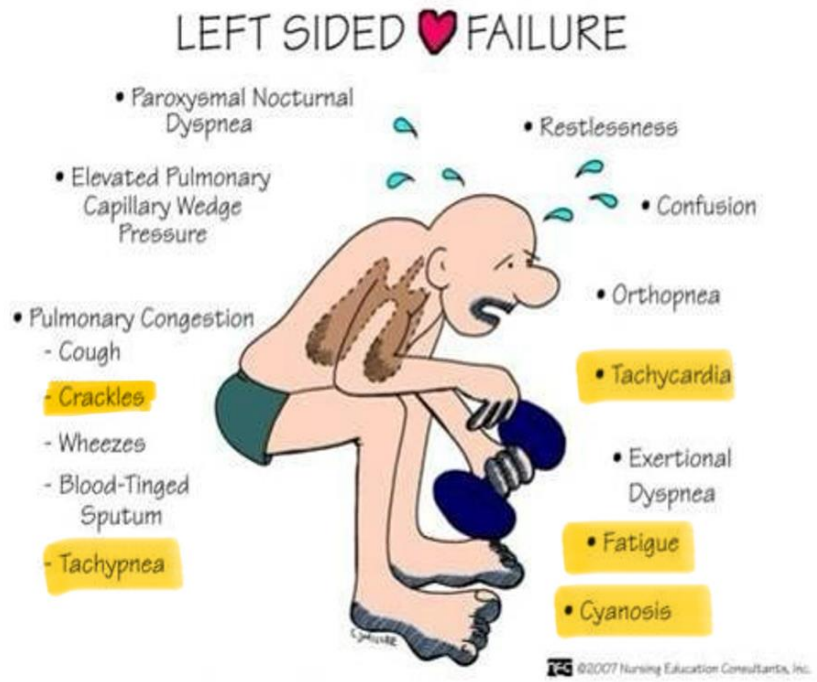
B	**Structural** heart disease but **without signs or symptoms** of HF	I

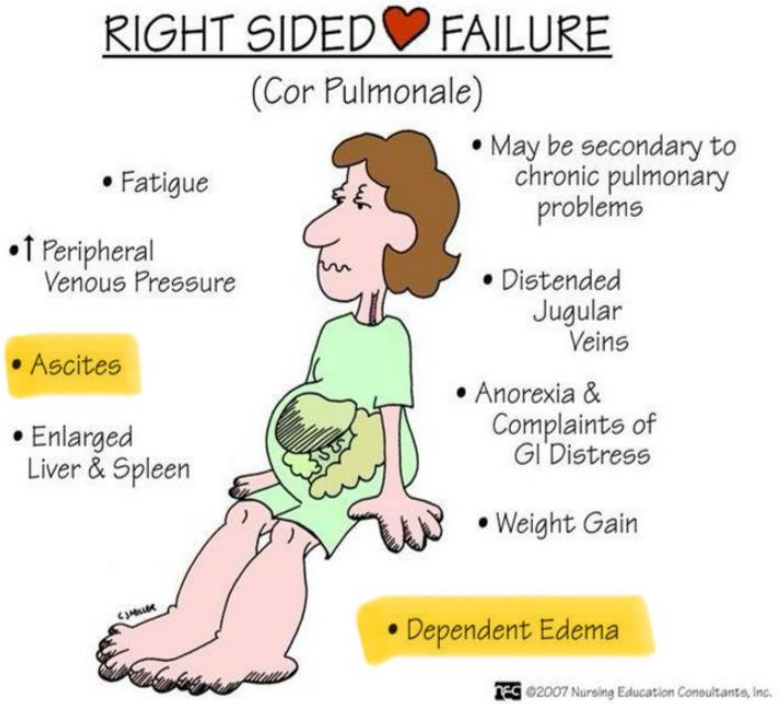
This patient's echo shows ⬇️ Ejection fraction , but he never came before with heart failure symptoms

C	**Structural** heart disease **with prior or current symptoms** of HF	I II III

C ➡️ most of the patients we follow up, they have structure problem (⬇️ EF) and have volume overload what is that here? They are classified according to NYC into categories.

Symptoms





- Mostly in Lt. side HF ▶️ pulmonary edema & highlighted above
- in Rt. Side HF ▶️ ⬆️ JVP & other highlighted above
- isolated from are rarely seen usually are combined which we call " congestive heart failure"
- When we can see right sided only? If the left ventricle is normal, but the patient have pulmonary disease (like severe cystic fibrosis, severe COPD)

HFrEF Management

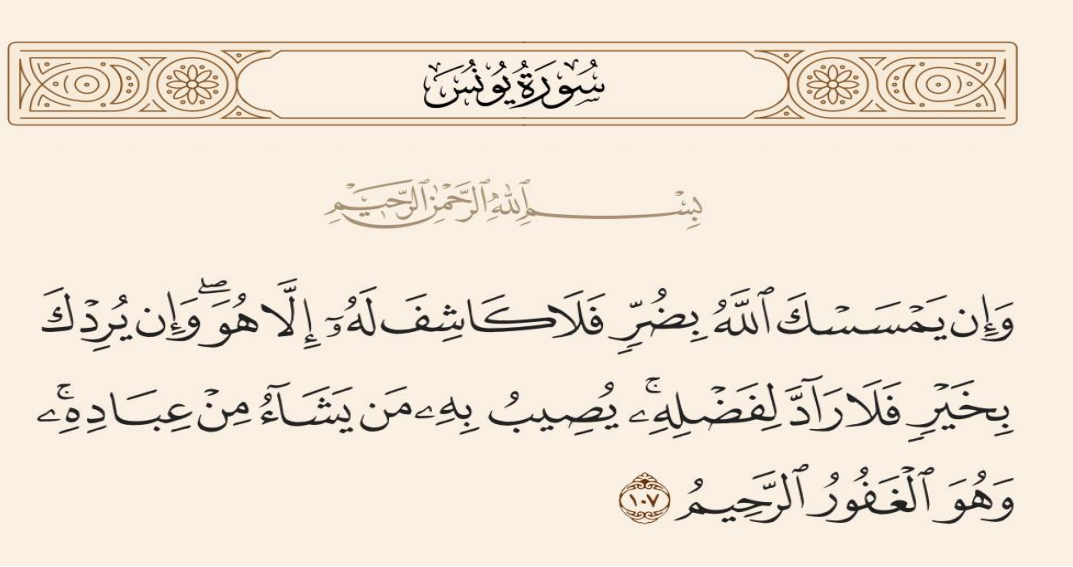
Pharmacological treatments indicated in patients with HFrEF (LVEF ≤40%; NYHA class II-IV)

Recommendations	Class of recommendation	Level of evidence
An ACEi is recommended for patients with HFrEF to reduce the risk of HF hospitalization and death	I	A
A BB is recommended for patients with stable HFrEF to reduce the risk of HF hospitalization and death	I	A
An MRA is recommended for patients with HFrEF to reduce the risk of HF hospitalization and death	I	A
Dapagliflozin /empagliflozin are recommended for patients with HFrEF to reduce the risk of HF hospitalization and death	I	A
Sacubitril/valsartan is recommended as a replacement for an ACEi in patients with HFrEF to reduce the risk of HF hospitalization and death	I	B

- Management is based on four types
- 1st class: ACEI/ARB /ARNI ” Angiotensin Receptor-Neprilysin Inhibitors”
- 2nd class: beta blockers
- 3rd class: Aldosterone Blockers (spironolactone)
- 4th class : diabetic medications “Na+/ glucose transport 2 inhibitors” , it was found that they have a good effect in treating heart failure patients

Thankyou

Additional sources



VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2	20	Muscle heart sound	What is in yellow
V2→V3			

امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!