

CARDIOVASCULAR PATHOLOGY

ISCHEMIC HEART DISEASE

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Ischemic Heart Disease

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OUTLINE

- I) CAUSES & PATHOPHYSIOLOGY
- II) CATEGORIES OF ISCHEMIC HEART DISEASE
- III) CLINICAL FEATURE
- IV) COMPLICATIONS
- V) DIAGNOSIS
- VI) 12 LEAD ECG STEMIS

- VII) TREATMENT
- VIII) APPENDIX
- IX) REVIEW QUESTIONS
- X) REFERENCES

I) CAUSES & PATHOPHYSIOLOGY

- Ischemic heart disease is also known as **coronary artery disease (CAD)**
- The underlying mechanism is a reduction in coronary blood flow, which supply oxygenated blood to the myocardium of the heart

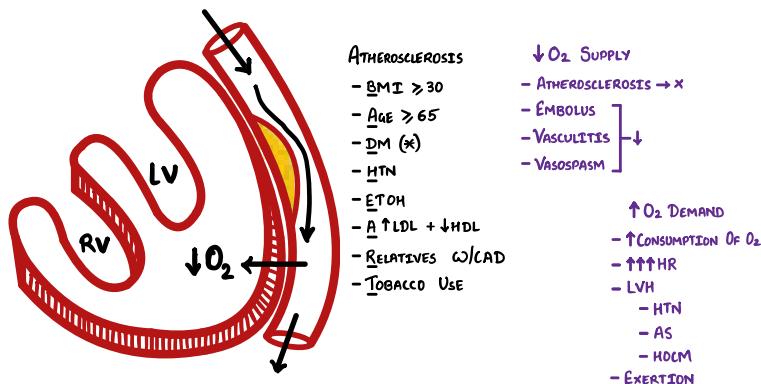


Figure 1. Causes of Ischemic Heart Disease

(A) ATHEROSCLEROSIS

- The most common cause of ischemic heart disease is the reduction or complete loss of coronary blood flow due to **atherosclerosis**
- Recall: Coronary blood vessels deliver oxygen to the myocardium of the heart and pick-up waste products (e.g. carbon dioxide)
- In this disease, atherosclerotic plaques develop within coronary vessels, causing a decrease in blood flow
- The plaques form a “big block” along the coronary vessels, resulting in:
 - Reduced blood flow and oxygen supply
 - Reduced amount of carbon dioxide or waste products being picked up
- When the myocardium has decreased or no oxygen supply,
 - it cannot generate ATP to initiate muscle contraction
→ myocardial failure

(1) Risk Factors for Plaque Formation (Mnemonic: BAD HEART)

- **B**MI ≥ 30 (**obesity**; modifiable risk factor)
- **A**ge ≥ 65 (non-modifiable risk factor)
- **H**ypertension (most common risk factor)
- **E**tOH (alcohol abuse)
- **A**n increase in LDL and a decrease in HDL (**hyperlipidemia**)
 - LDL – increased deposition of fat and cholesterol on the vessel wall
 - HDL – decreased removal of fat and cholesterol along the vessel wall
- **R**elatives with CAD
 - e.g. relatives who passed away due to heart attack/MI
- **T**obacco use
 - Smoking increases endothelial injury and plaque formation

(B) OTHER CAUSES

- When there is a mismatch between oxygen supply and demand, this can lead to **ischemia**
 - When ischemia is not reversed, it can lead to cell death or **infarction**

(1) Decreased Oxygen Supply

- Embolus
- Vasculitis
 - Inflammation of vessels can narrow the lumen
- Vasospasm

(2) Increased Oxygen Demand

- Tachycardia
 - Increased heart rate → increased myocardial contractility → increased oxygen consumption
- Left Ventricular Hypertrophy
 - Seen in patients with hypertension, aortic stenosis, and hypertrophic obstructive cardiomyopathies)
- Exercise/Exertion
 - Increases heart rate and myocardial contractility



II) CATEGORIES OF ISCHEMIC HEART DISEASE

(A) STABLE ANGINA

(1) Pathophysiology

- **Stable plaque**, with a strong fibrous cap preventing it from rupturing
- Occludes $\geq 70\%$ of the lumen

(2) Clinical Features

- Substernal chest pain characterized as squeezing, tight choking
- Chest pain occurs with exertion and can disappear with rest
 - there must be increased oxygen demand for the chest pain to manifest

(3) Blood Flow

- Patients with stable angina have **subendocardial ischemia**
 - The endocardial layer is the farthest layer from the coronary blood vessels; thus, it is the most susceptible to ischemia

STABLE ANGINA

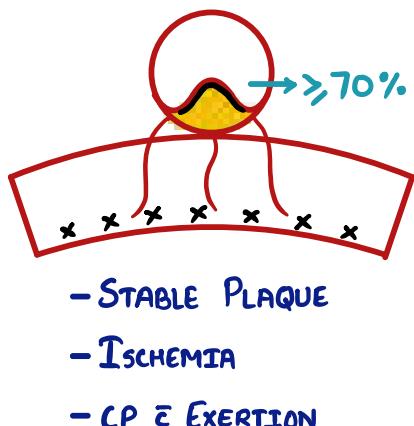


Figure 2. Stable Angina

(B) UNSTABLE ANGINA

(1) Pathophysiology

- **Unstable plaque**, with a weak fibrous cap that is prone to rupture
- Fatty center of the plaque is exposed; platelets attach to the center, leading to thrombus formation
- Occludes $\geq 90\%$ of the lumen (**near total occlusion**)

(2) Clinical Features

- Chest pain occurs at rest and worsens with exertion

(3) Blood Flow

- Patients with unstable angina also have **subendocardial ischemia**

UNSTABLE ANGINA

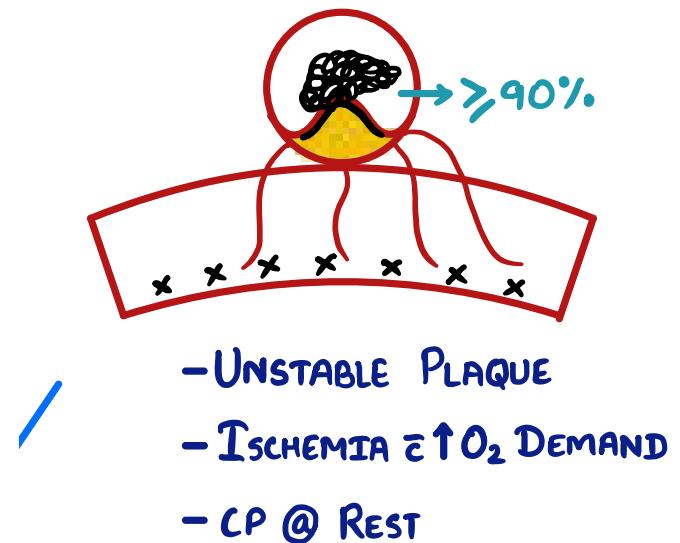


Figure 3. Unstable Angina



(C) SUBENDOCARDIAL INFARCTS / NSTEMI

(1) Pathophysiology

- **Unstable plaque**, with a weak fibrous cap that is prone to rupture
- Fatty center of the plaque is exposed; platelets attach to the center, leading to thrombus formation
- Occludes $\geq 90\%$ of the lumen (**near total occlusion**)

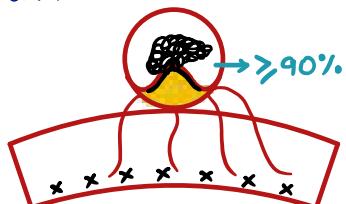
(2) Clinical Features

- Chest pain occurs at rest and worsens with exertion

(3) Blood Flow

- In subendocardial infarction, there is significant reduction of blood flow leading to infarction (cell death)
- Infarction results from at least **30 minutes of unreversed ischemia**

SUBENDOCARDIAL INFARCT (NSTEMI)



- UNSTABLE PLAQUE
- INFARCTION ✓ (≥ 30 MIN)
- CP @ REST

Figure 4. Subendocardial Infarct / NSTEMI

(D) TRANSMURAL INFARCT/STEMI

(1) Pathophysiology

- **Unstable plaque**, with a weak fibrous cap that is prone to rupture
- Fatty center of the plaque is exposed; platelets attach to the center, leading to thrombus formation
- Occludes **100%** of the lumen (**total occlusion**)

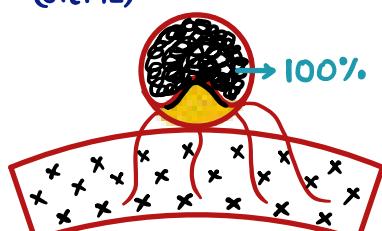
(2) Clinical Features

- Chest pain occurs at rest
- Severe chest pain with exertion

(3) Blood Flow

- There is complete loss of oxygen supply
- The entire myocardium (from the endocardium to the epicardial portion) will infarct ("transmural")

TRANSMURAL INFARCT (STEMI)



- UNSTABLE PLAQUE
- INFARCTION
- CP @ REST

Figure 5. Transmural Infarct / STEMI

Acute Coronary Syndrome

- A general term which encompasses:
 - Unstable angina
 - Subendocardial infarction / NSTEMI
 - Transmural infarct / STEMI

(E) VASOSPASTIC ANGINA / PRINZMETAL'S ANGINA

- With vasospastic angina, the chest pain is not related to/caused by atherosclerosis

- Typically occurs in young females with a history of:
 - Using illicit drugs (e.g. cocaine, methamphetamine)
 - Smoking
 - Alcohol abuse
 - Triptan use (for migraines or Reynaud's)
 - No cardiovascular risk factors

- Chest pain occurs at night

(2) Pathophysiology

- Chest pain is due to **vasoconstriction of coronary vessels**, narrowing the lumen and causing ischemia to the entire myocardium ("transmural")

(3) Diagnostic Features

- ST elevation on EKG
- Negative troponins
 - Troponins are a marker of destruction/infarction
- Reproduced by acetylcholine or ergonovine
- Reproduced by hyperventilation

(4) Treatment

- Prescribe **calcium channel blockers** or **nitroglycerin** to dilate blood vessels
- Avoid **beta blockers**
 - Recall: Beta receptors facilitate dilation; alpha receptors facilitate constriction
 - If beta receptor activity is blocked, only alpha receptor activity will register, leading to worsening vasoconstriction

PRINZMETAL'S (VASOSPASTIC) ANGINA

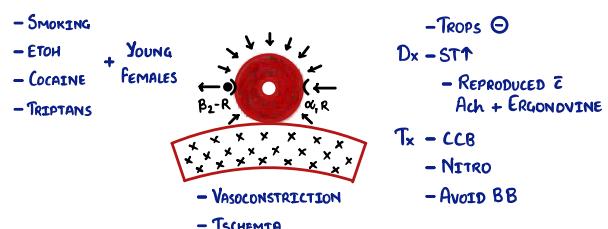


Figure 6. Prinzmetal's Angina / Vasospastic Angina



III) CLINICAL FEATURE

Taking history is important, use the OPQRST approach

Table -1. Presentation of IHD when taking history

History (OPQRST)	Stable Angina
Onset	Stable Angina: 5-10 mins Unstable angina, NSTEMI and STEMI (Acute Coronary Syndrome): >10 mins
Provocation	Worse on exertion, improved on rest, also improves with Nitroglycerin → Suggestive of Stable Angina When it occurs at rest, but can intensify with exertion → Unstable angina, NSTEMI and STEMI (Acute Coronary Syndrome)
Quality	Squeezing / Stabbing/ Pressure “Feeling like an elephant is sitting on your chest”
Radiation	Left Arm, Left Neck, Left Jaw <i>Epigastric pain can be indicative of inferior STEM</i>
Severity	Variable
Time	Constant

Atypical (silent) presentations in:

- Diabetics (neuropathy of particular nerves)
- Elderly
- Post heart transplant (alteration with the nerve signaling)
→ These patients can have MI **without** any chest pain

(A) CHEST PAIN:

- Heart has autonomic fibers that synapse on the dorsal grey horn of the spinal cord (cervicothoracic region)
- Sensory fibers from the left arm, left side of the neck and left side of the jaw synapse in the dorsal grey horn too.
 - So when an infarct develops in the heart, neurons traveling to the dorsal grey horn might travel at the same time as the sensory fibers from the left arm, neck and jaw
 - When both of these fibers travel to the brain, the brain interprets the chest pain coming from the left arm, left side of the neck and the left side of the jaw.
- The **autonomic nervous system** can activate in response to this resulting in:
 - Nausea, vomiting, diaphoresis, syncope, sense of impending doom.

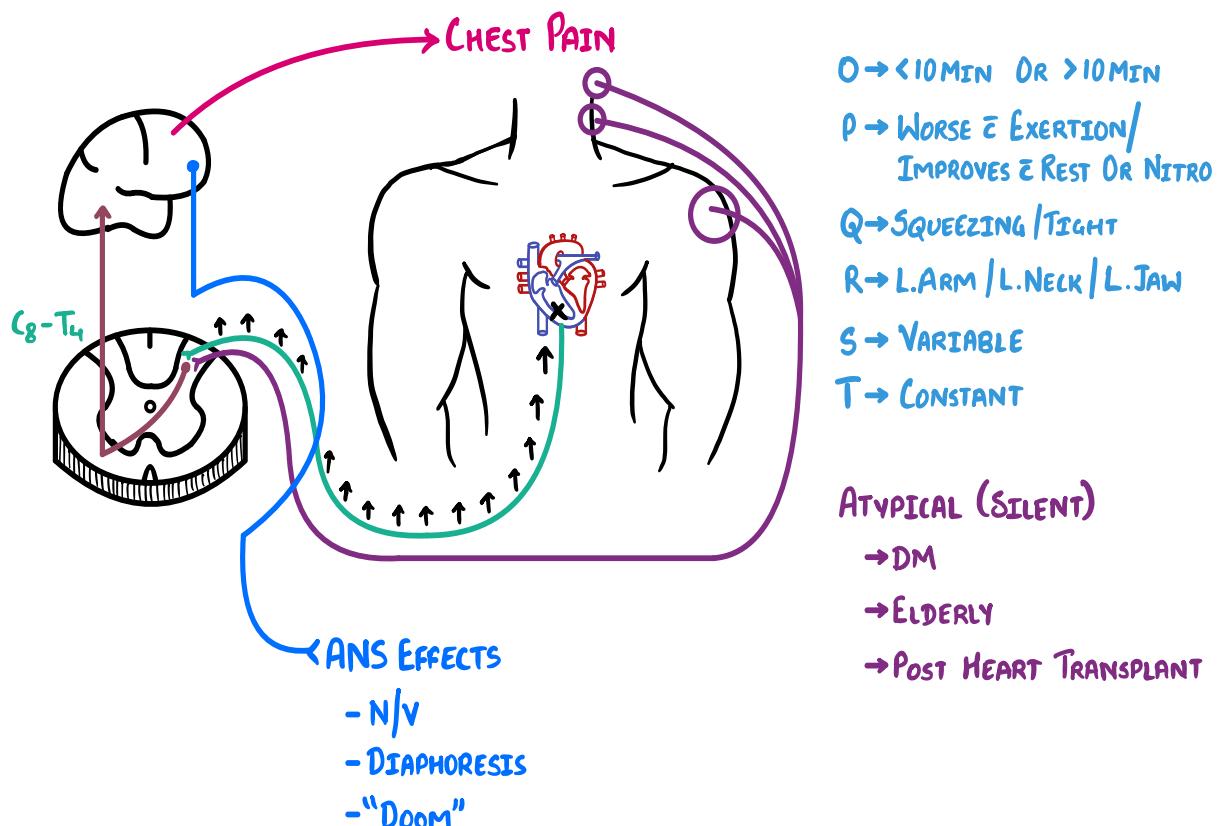


Figure 7. Chest pain radiation to the left arm, left side of the neck and left side of the jaw



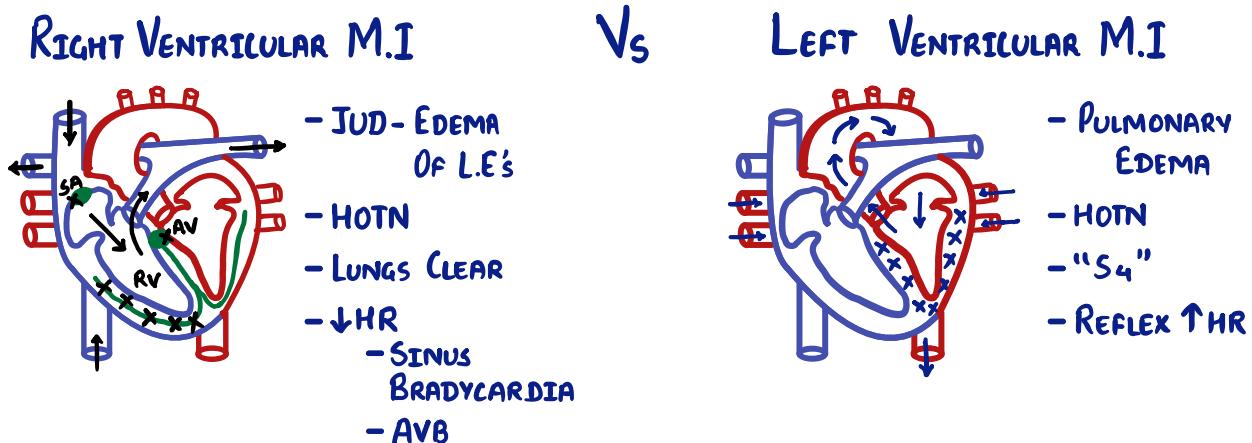


Figure 8. Right Ventricular MI Vs Left Ventricular MI

RIGHT VENTRICLE MI:**JVD & Lower Extremities Edema**

- Normally, the right ventricle receives blood from the superior/inferior vena cava → right atrium → right ventricle → to the lungs
 - If the right ventricle undergoes ischemia and there is a decrease in the heart's ability to pump blood
 - A back flow of blood into the vena cava occurs
 - If it backs up into the *superior vena cava* it will travel to the brachiocephalic → jugular veins causing **Jugular Venous Distension (JVD)**
 - If it backs up into the *inferior vena cava* it can cause lower extremities edema, hepatomegaly, ascites, etc.

Hypotension

- When there is less blood in the right ventricles, there is less blood being pumped to the lungs
 - Preload decreases = decreases stroke volume = decreases the cardiac output
 - Causing **hypotension**
 - Lungs are clear (no edema)

Decreased Heart Rate

- Right coronary artery supplies the SA and AV node
 - If the right coronary artery is occluded (as in IHD) SA and AV node (especially the AV node) do not receive proper blood supply
 - This affects the conduction through the heart
 - Can lead to **decrease in heart rate**
 - Sinus bradycardia
 - AV block (it can be 1st, 2nd or 3rd degree, *it depends on the severity of the AV dysfunction*)
- It is important to avoid giving drugs that ↓ preload

LEFT VENTRICLE MI:**Pulmonary Edema**

- Pulmonary Circulation → Left Atrium → Left Ventricle
 - if the left ventricle undergoes ischemia and stops working, it will cause a back flow into the pulmonary circulation and leak out into the pulmonary capillaries
 - Causing **pulmonary edema**
 - Presentation: shortness of breath

Hypotension

- When there is less blood in the left ventricles, there is less blood being pumped to the aorta
 - Decreased ejection fraction leads to **hypotension**
 - Hypotension decreases perfusions to organs
 - Leading to **cold mottled pale extremities**

Reflex Tachycardia

- Hypotension triggers this reflex
 - As there is a ↓ cardiac output, the autonomic nervous system compensates by ↑ heart rate to ↑ cardiac output and blood pressure
 - This is called **reflex tachycardia**

S4 heart sounds

- Since the ventricles are infarcted they become really stiff
 - When the ventricles fill it produces a **S4 heart sound**

Right Ventricular MI	Left Ventricular MI
Lungs are clear	Pulmonary Edema
↓Preload → ↓SV → ↓CO → ↓BP (HOTN)	↓EF (Ejection Fraction) → HOTN
Jugular Venous Distension (JVD)	↓Perfusion → Cold, mottled skin, Pallor
Hepatomegaly + Ascites	
Edema in the lower extremities	
Sinus Bradycardia OR AV block (degree depends on severity)	
	S4 heart sound
	Reflex Tachycardia (↑HR)



IV) COMPLICATIONS

THE FIRST 24 HOURS

(A) 24 HOURS - 3 DAYS

Sudden cardiac death

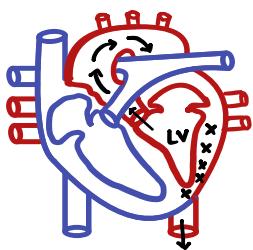
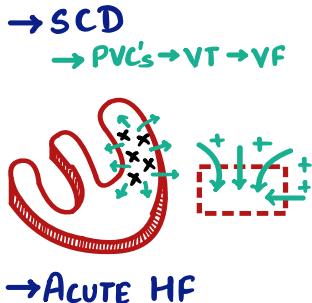
- If there is an MI, the **cell permeability** ↑ in the area of infarction.
 - high permeability: ↑ cations (calcium, sodium) load into the cells, this depolarizes the cells abnormally, and generates electric potentials causing the ventricles to contract abnormally
 - This can lead to PVC and progress into VT then VF
→ Ventricular fibrillation can result in **Sudden Cardiac Death**

Cardiogenic Shock

- If there is a significant infarct in a major vessel (e.g LAD occlusion) especially in the left ventricle
→ The heart would not be able to push blood into the systemic circulation resulting in **hypotension**
 - Hypotension causes cold extremities due to hypoperfusion
 - This can progress to **cardiogenic shock**

Flash Pulmonary Edema

- Blood backs up into the pulmonary circulation leading to **flash pulmonary edema**
 - Shortness of breath, hypoxia
 - reflex tachycardia to maintain a decent cardiac output



- HOTN → SHOCK
- COLD EXT.
- FLASH PULMONARY EDEMA
- REFLEX ↑ HR

Rupture syndrome:

• IN SEPTUM:

- When there is an infarction in the septum, the tissue becomes weak and susceptible to rupture
 - Since the left ventricle pumps blood at a higher pressure, with a weak septum it would be able to push blood into the right ventricle
 - This is called a **Ventricular Septal Defect** due to an inter-ventricular septal rupture
 - May cause:
 - Murmur (hollow systolic murmur)
 - Mixed venous oxygenation

• IN LEFT VENTRICLE:

- Infarct in the left ventricle can render it weak and if it ruptures, blood can get into the pericardial cavity, this is called a **free wall rupture**.
 - Blood in the pericardial cavity can lead to hemopericardium resulting in **Cardiac Tamponade**.
 - **Recall:** Beck's Triad occurs with cardiac tamponade, this consists of hypotension, JVD, and muffled heart sounds.

• IN PAPILLARY MUSCLES:

- Papillary muscles are found in the inner walls of the heart, and they attach to the valves via the chordae tendineae to anchor the valves.
 - If an infarct develops in the papillary muscles, the tissue can get weak and rupture.
→ Valves would no longer be anchored and become flail causing regurgitation.
 - If this occurs in the left heart, it causes **Mitral Valve Regurgitation**.
 - Presentation: mitral valve murmur, hypotension, pulmonary edema, etc.

Left Ventricular Pseudo Aneurysm

- In free wall ruptures, blood can accumulate in the pericardium and this can form a pseudoaneurysm
 - Due to blood stasis, clots can form in these areas and increase the **risk of thrombi** (mural thrombus)

→ RUPTURE SYNDROMES



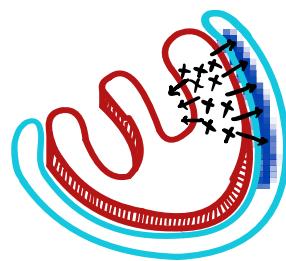
- 1) **VSD**
 - MURMUR +
 - MIXED VENOUS O₂
- 2) **FREE WALL RUPTURE**
 - TAMPOONADE
- 3) **PAPILLARY RUPTURE**
 - MR MURMUR
- 4) **LV PSEUDOANEURYSM**
 - ↑ THROMBI



Pericarditis

- If there is an infarct in the left ventricle, an inflammatory reaction can occur to clean up the debris
 - This local inflammatory reaction is called **pericarditis**
 - Presentation: *sharp and positional* chest pain, friction rub (via lung sounds)
 - The inflammation can increase the formation of fluid, leading to **pericardial effusion**.

→ PERICARDITIS



→ CP

- SHARP

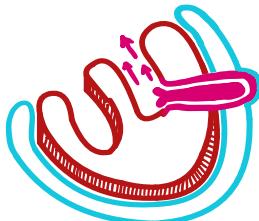
- POSITIONAL

- FRICTIONAL RUB

- PERICARDIAL
EFFUSIONSLeft Ventricular Aneurysm

- In an infarction, first there is a soft granulation tissue → fibrous tissue.
 - Fibrous tissue can "bulge out" creating a **left ventricular aneurysm**
 - There is a low risk of rupture since the fibrous tissue is stronger
 - Since the affected area of the ventricle is not contracting, this creates **blood stasis** which increases the risk of clot formation and mural thrombi.
 - **Recall:** Virchow's triad; if there is stasis, hypercoagulability or endothelial injury then there is a higher risk of clot formation
 - Thrombi also increases the risk of **emboli** formation which can occlude different organs (e.g. brain, spleen, kidney, etc.)

→ LV ANEURYSM

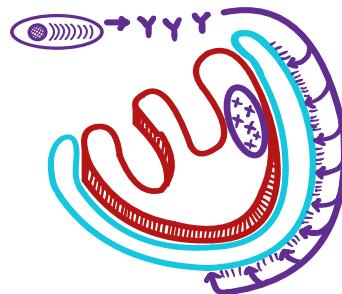


- MURAL THROMBI
- ↑ EMBOLI

Dressler's Syndrome

- Infarct in the ventricle can create an underlying immune reaction, however the etiology is unknown
 - The immune system develops antibodies against the pericardium
 - This leads to the inflammation of the pericardium
- (This occurs later, unlike the pericarditis that occurs in the 3-14 days time period)*

→ DRESSLER SYND.



V) DIAGNOSIS

(A) OVERVIEW OF DIAGNOSTICS (EKG AND BIOMARKERS)

- The diagnostics used in ischemic heart disease are:
 - 12-lead EKG
 - Biomarkers such as troponin and CK-MB

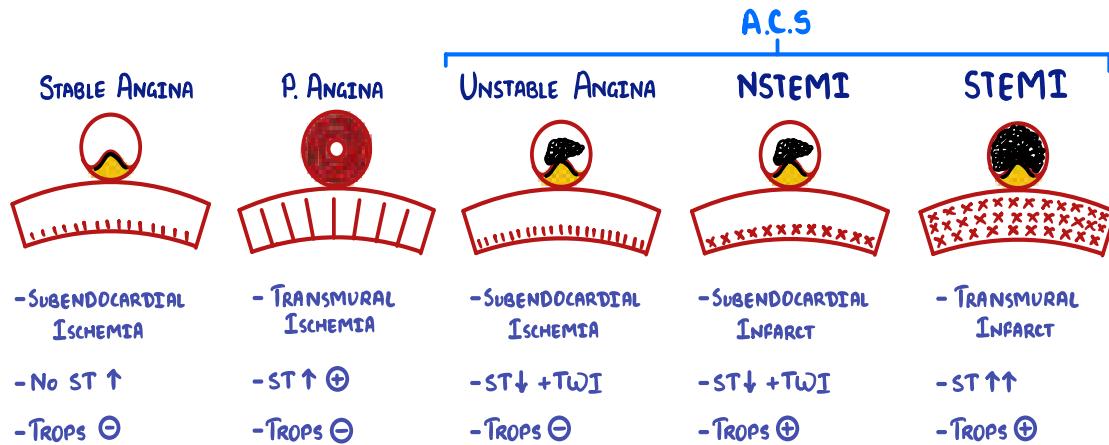


Figure 9. Diagnostic features of different ischemic heart diseases.

(1) Stable Angina

- Subendocardial ischemia**
- 12-lead EKG will show **NO ST segment elevation**
 - May have other ST wave changes but it would not be anything obvious such as ST depression, T wave inversions, Q waves
- Stable angina does NOT have infarction but it has **ischemia** → no cell destruction → no leakage of biomarkers = (-) **troponin** and **CK-MB**

For patients with ischemic chest pain, biomarkers such as **troponin** and sometimes, **CK-MB** should also be ordered (aside from EKG)

(2) Prinzmetal (Vasospastic) Angina

- Transmural ischemia** → produce **ST segment elevation** that is transient and can be reproduced by acetylcholine or ergonovine
- (-) **troponin** and **CK-MB** since vasospastic angina has ischemia; NO infarction

(3) Acute Coronary Syndromes (Unstable Angina, NSTEMI, STEMI)

- Unstable angina, NSTEMI and STEMI are categorized as **acute coronary syndrome (ACS)**
 - Most severe of ischemic heart disease

(i) Unstable Angina

- Subendocardial ischemia** → NO infarction → may show signs of **ST depression and T wave inversion** but **NO ST segment elevation**
- (-) **troponin** and **CK-MB** since there is no infarction

(ii) NSTEMI

- Subendocardial infarct** → there is tissue destruction → may show **ST segment depression and T wave inversion**; **NO ST segment elevation**
- (+) **troponin** and **CK-MB** since there is tissue destruction so these biomarkers can leak

(iii) STEMI

- Transmural infarct**
- 12-lead EKG shows **ST segment elevation**
- (+) **troponin** and **CK-MB** since it is a transmural infarct = there is tissue destruction and biomarkers can leak

(B) BIOMARKERS

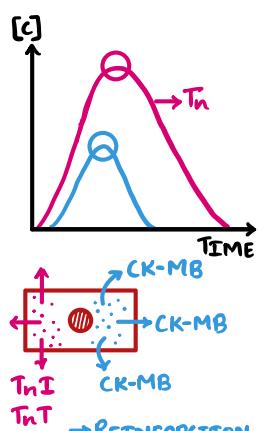


Figure 10. Biomarkers troponin and CK-MB.

- Myocardial cell has special types of molecules called **troponin (TnI and TnT)** and **CK-MB**
 - If there is tissue destruction, these biomarkers will leak out of the cells
 - CK-MB** is more specific for heart tissue destruction
- TnI and TnT usually takes **12-24 hours** before reaching the peak similar to CK-MB; however, **CK-MB normalizes quicker than TnI and TnT**
 - CK-MB can be used to determine **re-infarction**
 - If patients complain of new chest pain after an infarction, **order CK-MB**; troponins will still remain elevated and would not have normalized yet



(C) REVIEW OF CORONARY VESSELS AND THE LEADS INVOLVED IN THE EKG

- L.A.D.
 - ANT. WALL (V₁-V₄)
 - SEPTAL
 - APICAL
 - LATERAL
- L.C.X. (I/avL, V₅-V₆)
 - LAT. WALL
- R.C.A (II/III/avF)
 - INF. WALL
 - RV
- P.D.A (V₇-V₉)
 - Post. WALL

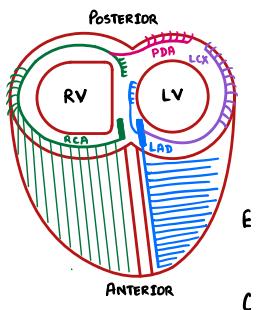


Figure 11. Coronary vessels and their corresponding ECG leads.

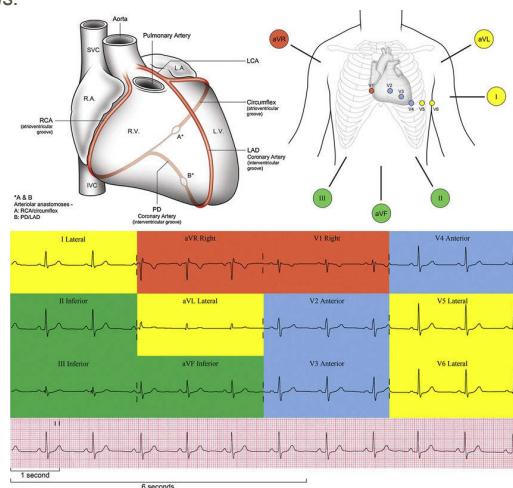


Figure 12. (Top left) Coronary arteries; (Top right) Positioning of ECG leads; (Bottom) Corresponding ECG leads [Blakeway et al, 2012].

(1) Left Anterior Descending Artery (LAD)

- Supplies the **anterior** compartment of the heart (including the septum)
- If there is infarction in the anterior wall of the heart, LAD is involved
- Leads involved: **V₁-V₄**
 - V₅ and V₆ sometimes

(2) Left Circumflex Artery (LCX)

- Branch of the LAD
- Supplies the **lateral** wall of the heart
- Leads involved: **I, avL, V₅ and V₆**

(3) Right Coronary Artery (RCA)

- Supplies the right ventricle (**marginal branch**) including the apex of the heart and the posterior part of the septum
- If there is infarction in the **inferior part** of the heart, RCA is involved
- Leads involved: **II, III, avF** and right-sided chest leads

(4) Posterior Descending Artery (PDA)

- Branch of the RCA
- Supplies the **posterior** wall of the heart
- Leads involved: **V₇-V₉**

Table -2. Summary of the coronary vessels and the leads involved when infarction occurs.

ARTERY	HEART PORTION SUPPLIED	LEADS INVOLVED
LAD	Anterior	V ₁ -V ₄
LCX	Lateral	I, aVL, V ₅ , V ₆
RCA	Inferior	II, III, aVF, right-sided leads
PDA	Posterior	V ₇ -V ₉

(D) DIAGNOSTIC FEATURES

(1) EKG

- Sequence of ischemia-infarction (**STEMI**) as seen in EKG:
 - Hyperacute T waves
 - Very particular for MI
 - ST segment elevation
 - Q waves
 - ± T wave inversion (TWI)
 - ST segment normalizes
 - T wave normalizes
- Also look for **ST segment depression** and **T wave inversion** by itself = indicative of unstable angina and NSTEMI

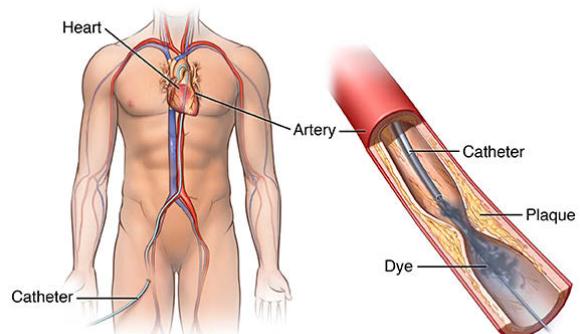
(2) Echo

- Important for diagnosing ischemic heart disease
 - Developing an infarct in the LCX territory → left lateral wall became ischemic and infarcted → not contracting
- Can show **wall motion abnormality**

(3) Cath (Cardiac Catheterization)

- **Best/most definitive test** for myocardial infarction
- A catheter is taken up from the radial or femoral artery then look into the vessels of the heart and release some contrast
 - Then, flow of the contrast can be visualized
 - If the flow of contrast stops in one area, there might be occlusion in the vessel
- Diagnostic; **AND**
- Therapeutic
 - Percutaneous coronary intervention (PCI)

Coronary angiography



Angiogram

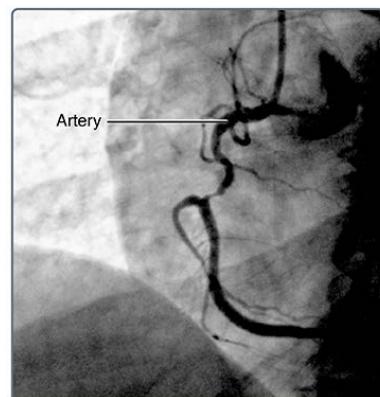


Figure 13. (Top) The process of cardiac catheterization or coronary angiography. (Bottom) Visualization of coronary vessels through injection of dye [Johns Hopkins Medicine, n.d.].



(E) DIAGNOSTIC APPROACH

- Patient complains with ischemic chest pain

(1) 12 Lead ECG

- First test to do if concerned with IHD

- Shows ST↑ → **Cath**
- No ST↑ but there is ST↓, TWI and Q waves → **Troponin**

(2) Troponin

- If (+) troponin → patients can be restratified through **TIMI Score**

- TIMI Score restratifies patients with NSTEMI or unstable angina and determines the need for catheterization based on the risk of mortality
- For patients with intermediate probability of coronary artery disease (CAD)
- TIMI ≥2 → **Cath**
- !! **may be asked in exams**

- If (-) troponin → **may be stable angina**

- Determine whether they should get a **stress test** → **(+)** stress test → elective **Cath**

(i) TIMI Score

- Restratiifies patients with NSTEMI or unstable angina and determines the need for catheterization based on the risk of mortality
- For patients with intermediate probability of coronary artery disease (CAD)

NSTE-ACS (TIMI Score) Mnemonic:

- Age ≥ 65
- Marker (Trops are elevated)
- ECG (ST↓)
- Risk factors (≥ 3)
 - Diabetes, hypertension, smoking, age
- Ischemic chest pain with at least 2 events within 24-hr period
- CAD $\geq 50\%$ stenosis of one of coronary vessels
- ASA use within the last 7 days

0-1: ↓ risk = medically managed and reevaluate
 ≥ 2 : ↑ risk = considered for catheterization

DIAGNOSTIC APPROACH

IHD?



12 LEAD ECG → ST↑

No ST↑

TROPS → +

S. ANGINA?

STRESS TEST → +

CATH



Figure 14. Summary of the diagnostic approach for patients with ischemic chest pain.

(F) STRESS TESTING

(1) How to determine the need for Stress Testing?

- Look for the Diamond Classification of Chest Pain for **Stable Angina**
 - Anginal chest pain + age/gender
 - Substernal chest pain
 - Worse chest pain with exertion
 - Relieved by nitroglycerin

(2) Stress Testing

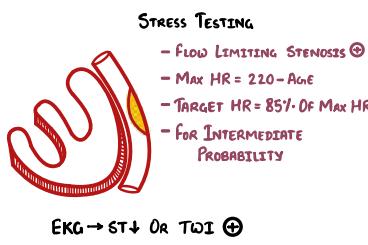


Figure 15. Stress testing and the "positives" for EKG, echo and MPI.

- Only important in **flow limiting stenosis**
 - !! can come up in exams
 - Cannot diagnose MI
- Patients should have an intermediate probability of CAD
 - Do not send if the patient has high or low probability of CAD
 - If patient has a high probability → Catheterization
- With patients with ↓O₂ supply because of occlusion, an increase in O₂ demand can **worsen** the ischemia → worsen the chest pain
- Patient walk on a treadmill → increase O₂ demand → **ischemic event**

- **Max HR = 220-Age**

- **Target HR = 85% of Max HR**

- Target HR for the patients to work out to reaching a high oxygen demand that can induce an ischemic event

- Baseline EKG, Echo and MPI (myocardial perfusion imaging) are determined and then compared post-workout

- **EKG: ST↓ or TWI = (+)**
 - Uninterpretable EKG due to a pacemaker, left bundle branch block, other abnormalities → go do an Echo
- **Echo: wall motion abnormality = (+)**
- **MPI: cold spots (areas of poor perfusion) = (+)**
 - New imaging modality that uses Technetium to visualize well perfused myocardium

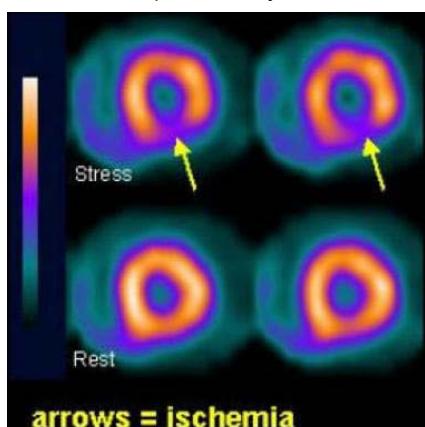


Figure 16. Cold spots (arrow) as seen in MPI [Cardona, 2021].

(3) Pharmacologic Stress Testing

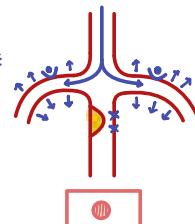


Figure 17. Pharmacologic Stress Testing.

- For patients who are NOT able to do the exercise aspect of stress testing
 - Patients who do not want to exercise; disabled; etc.
- Can replicate stress testing (workout) through certain medications
 - !! They love to ask this on USMLE Step 2
- Medications used: **dobutamine, adenosine** and **diperidamole**

(i) Dobutamine

- For Stress Echo
- Dobutamine binds on β₁ receptors on the myocardium and nodal cells → ↑HR, ↑SV → ↑contractility → ↑O₂ demand

(ii) Adenosine and/or Diperidamole

- For MPI
- Vasodilators
 - Both bind on **healthy vessels** and cause them to dilate maximally → allow blood to easily flow on the healthy vessels
 - Do not work on **diseased vessels** → no dilation → blood is "stolen" by healthy vessels (less resistance) → diseased vessels are "starved" and start to get ischemic = **Coronary Steal Syndrome**



VI) 12 LEAD ECG STEMIS

- When looking at an EKG, determine the following:
 - Rate
 - Rhythm
 - Axis
 - ST segments

Larger version of the ECG Strips can be found in the [Error! Reference source not found.](#) section.

(A) CASE 1: ANTERIOR STEMIS

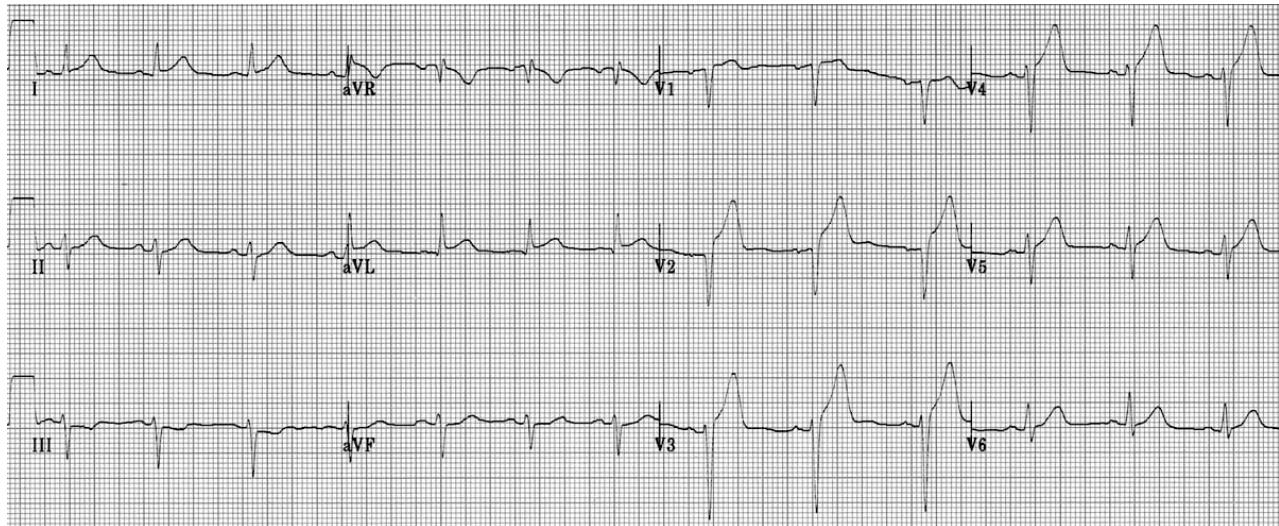


Figure 18. Case 1 ECG Strip.

- In this case, there are no rhythm strips. Therefore, rate-rhythm sequence can be skipped and move on to ST segment deviation
- Check all the limb leads (I, II, III, aVR, aVL, aVF) and then the precordial leads (V₁-V₆)

Table 3. ST segment deviation in each ECG lead.

ECG LEADS	ST SEGMENT DEVIATION
I	Subtle ST segment elevation
II	Unremarkable
III	Nothing specific – may be an ST-T wave abnormality
aVR	Unremarkable
aVL	ST segment elevation
aVF	Unremarkable
V ₁	ST segment elevation + pathologic Q wave (deep and wide Q wave)
V ₂	ST segment elevation + hyperacute T wave (funky and asymmetric T wave)
V ₃	ST segment elevation + hyperacute T wave
V ₄	ST segment elevation + hyperacute T wave
V ₅	Unremarkable
V ₆	Unremarkable

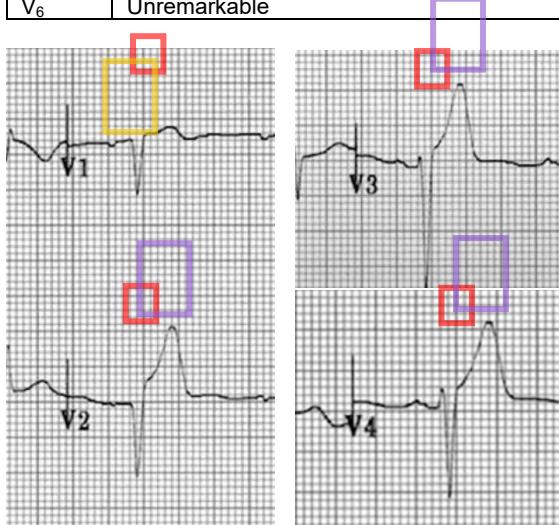


Figure 19. ST segment elevation (red) in V1-V4. Pathologic Q wave (yellow) in V1. Hyperacute T wave (violet) in V2-V4.

- Since there is ST segment elevation in V1-V4, there is occlusion in **LAD** → infarction or ischemia in the anterior heart
 - If it starts affecting some of the lateral part (anterolateral), there can be some changes in V5, V6, I and aVL
 - Suspicion: **anterior MI**

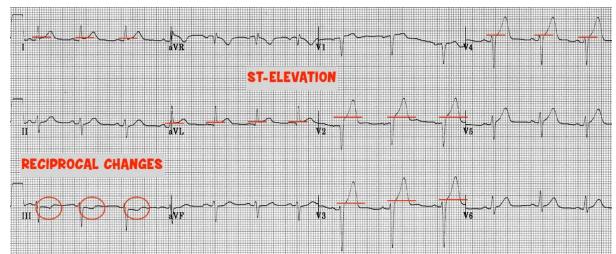


Figure 20. Labeled ECG Strip of Case 1.

- Summary:**
 - There are ST segment elevation in V1-V4, I, and aVL
 - Reciprocal changes (ST segment depression or T wave inversion) in III
 - Very significant in STEMI
 - ST segment depression:** below the TP segment
- The case is a **significant LAD occlusion** causing ischemia in the anterior septal (V2 & V3), apical (V4) and lateral (I & aVL) portion of the heart with reciprocal changes in III = **anterior STEMI**

Anterior STEMI

- ST↑ in V2-V4



(B) CASE 2: LATERAL STEMİ

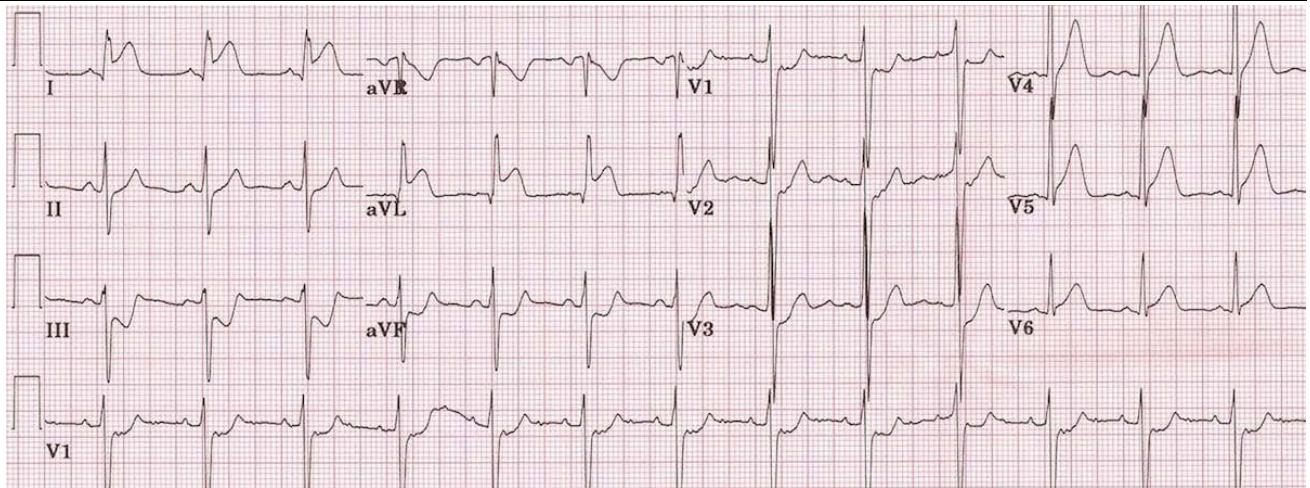


Figure 21. ECG strip of Case 2.

(1) Rate, Rhythm, Sinus

- **Rate:** regular
 - Not too fast, not too slow
- **Rhythm:** regular
 - Normal sinus rhythm – upright P wave in II and inverted P wave in aVR

(2) ST Segment Deviation

Table 4. ST segment deviation in ECG leads for Case 2.

ECG LEADS	ST SEGMENT DEVIATION
I	Large ST↑
II	Unremarkable
III	ST↓ (reciprocal change)
aVR	Unremarkable
aVL	ST↑
aVF	ST↓ (reciprocal change)
V ₁	Small ST↓ (reciprocal change)
V ₂	ST↓ (reciprocal change)
V ₃	ST↓ (reciprocal change)
V ₄	Unremarkable
V ₅	ST↑
V ₆	Subtle ST↑

Mainstem occlusion can be found in aVR

Summary:

- ST↑ in leads I and aVL → occlusion in **LCX** or distal LAD → affecting the **high portion of the left ventricle**
- ST↑ in lead V5 → affect the **anterolateral** portion of the heart
- Subtle ST↑ in lead V6

- **Diagnosis:** **Lateral STEMI**

Lateral STEMI

- ST↑ in I, aVL

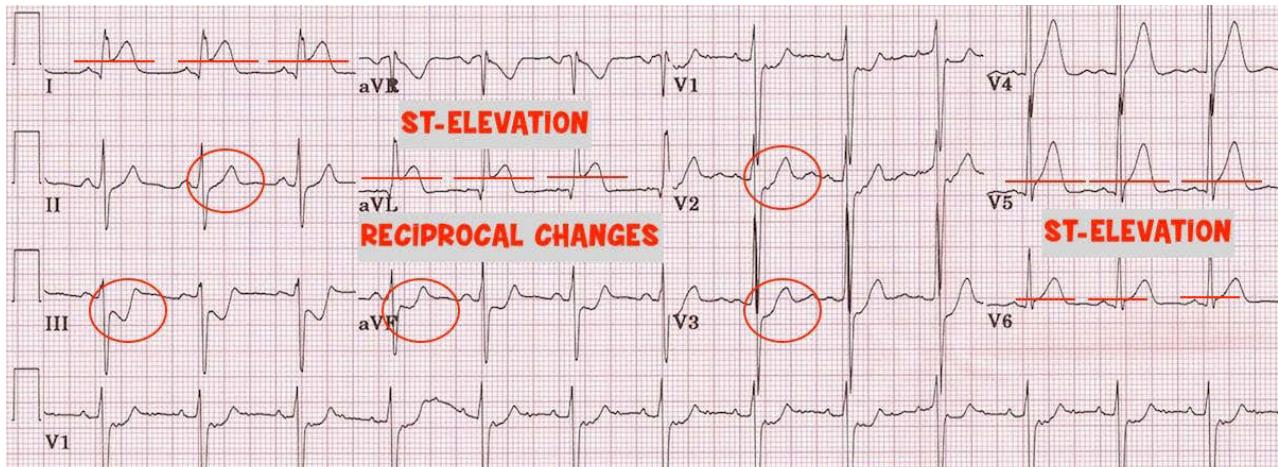


Figure 22. Labeled ECG strip of Case 2. ST elevation in leads I, aVL, V5 and V6. Reciprocal changes (encircled) in III, aVF, V2, and V3.



(C) CASE 3: INFERIOR STEMI

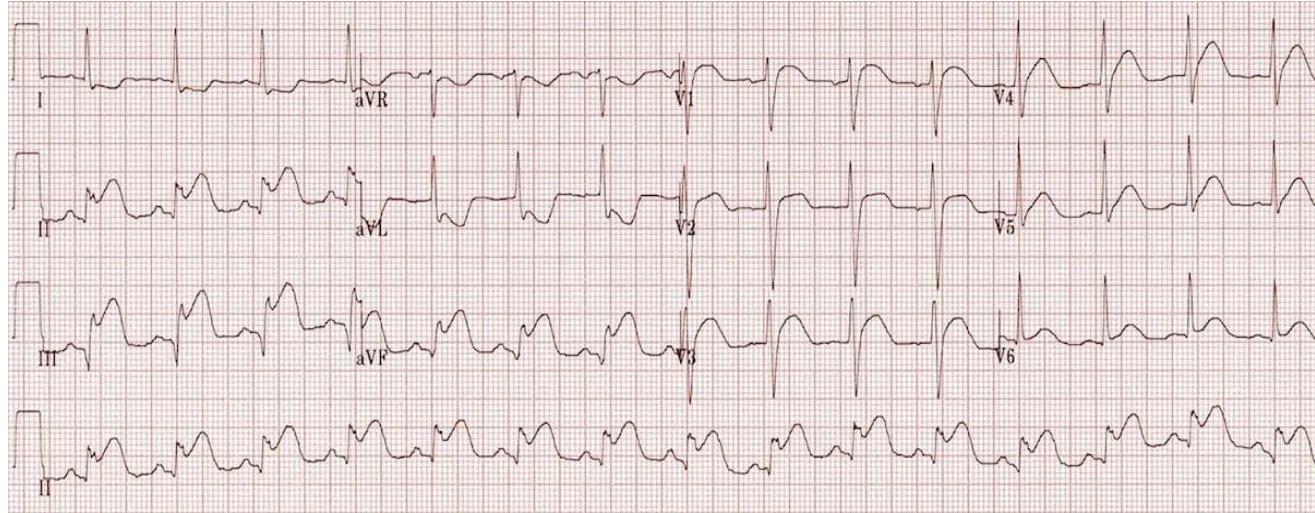


Figure 23. ECG strip of Case 3.

(1) Rate, Rhythm, Sinus

- **Rate:** regular
- **Rhythm:** regular
 - R-R intervals are similar
 - P wave in lead II is upright, inverted in aVR = **sinus rhythm**

(2) ST Segment Deviation

Table 5. ST segment deviation in ECG leads for Case 3.

ECG LEADS	ST SEGMENT DEVIATION
I	ST↓
II	ST↑
III	ST↑
aVR	Unremarkable
aVL	ST↓
aVF	ST↑
V ₁	Subtle ST↑
V ₂	Unremarkable
V ₃	Unremarkable
V ₄	Unremarkable
V ₅	Unremarkable
V ₆	Unremarkable

• Summary:

- ST↑ in II, III and aVF = **inferior STEMI** → affects the inferior portion of the heart
- Reciprocal changes in I and aVL
- Inferior STEMI is associated with occlusion of the RCA
 - Occlusion of the RCA will affect the blood flow in the inferior wall of the heart AND the right ventricle developing right ventricular infarction
 - Therefore, **evaluate right-chest leads** to assess for RV infarction

Inferior STEMI

- ST↑ in II, III and aVF

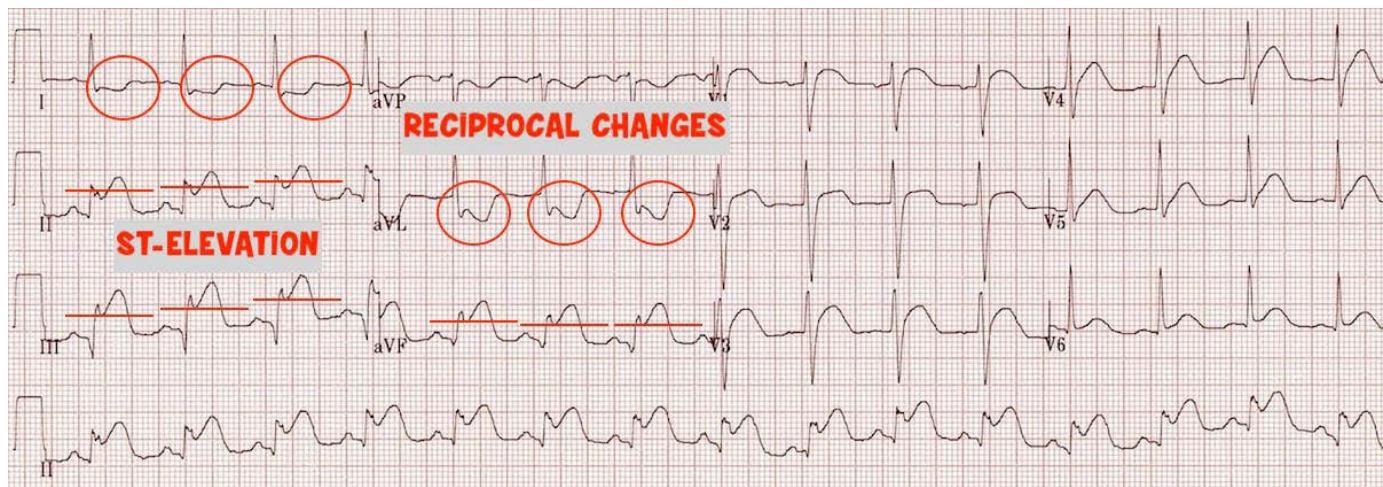


Figure 24. Labeled ECG strip of Case 3. ST elevation (line) in II, III and aVF with reciprocal changes (encircled) in I and aVL



(D) CASE 4: INFERIOR STEMI WITH ASSOCIATED RIGHT-SIDED MYOCARDIAL INFARCTION

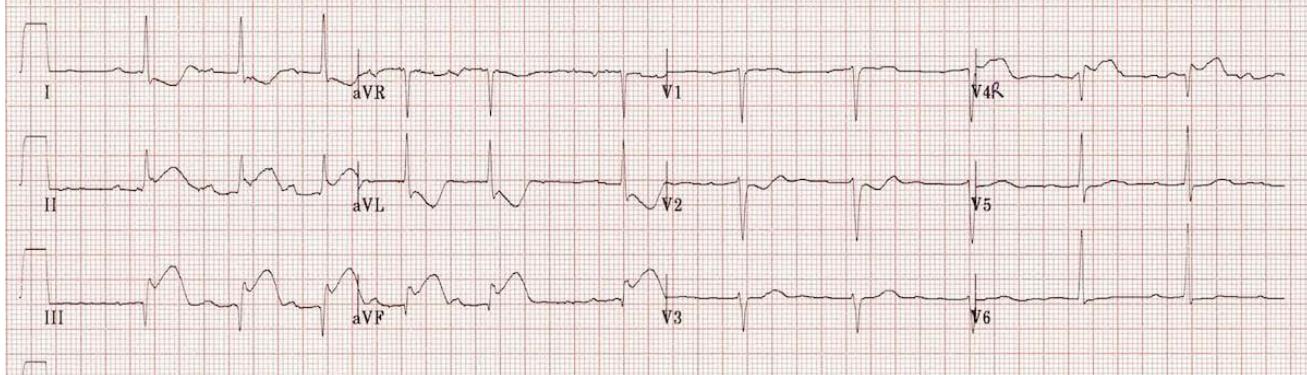


Figure 25. ECG strip of Case 4.

Table 6. ST segment deviation in ECG leads for Case 4.

ECG LEADS	ST SEGMENT DEVIATION
I	ST↓
II	ST↑
III	ST↑
aVR	Unremarkable
aVL	ST↓
aVF	ST↑
V ₁	Unremarkable
V ₂	Small ST↓
V ₃	Unremarkable
V ₄	Unremarkable
V _{4R*}	ST↑
V ₅	Unremarkable
V ₆	Unremarkable

*V_{4R}: right-sided V₄

- ST↑ in II, III and aVF with reciprocal changes in I and aVL = **inferior STEMI**
- Remember to have a high degree of suspicion for right ventricular infarction therefore track over the chest leads (V1-V6) into the right side
 - Any ST↑ (V1R-V4R) in the right-sided chest leads = high suspicion for RV infarction or right-sided MI
- **Summary:**
 - ST↑ in II, III and aVF = **inferior STEMI**
 - ST↑ in V4R = **associated right-sided MI**

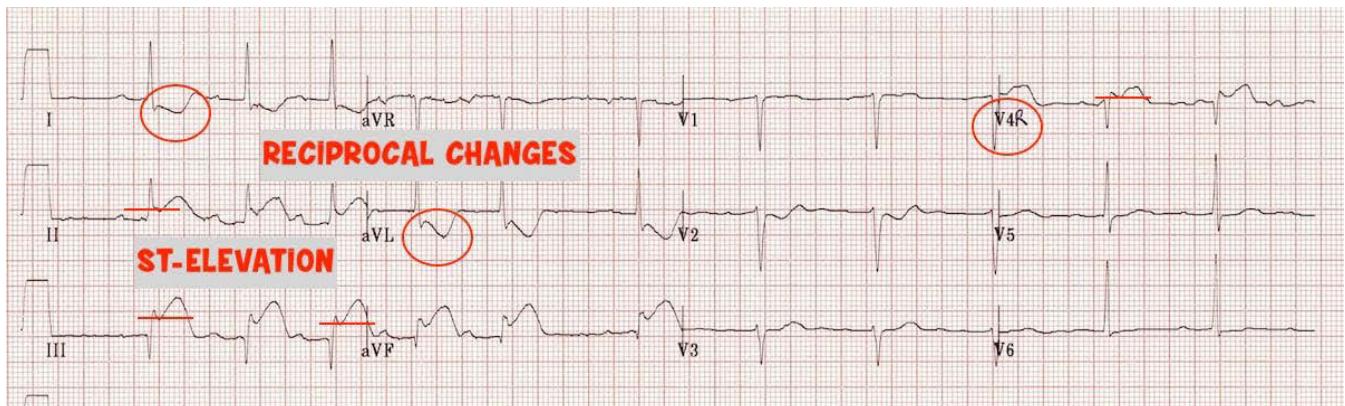


Figure 26. Labeled ECG strip of Case 4. ST elevation in II, III and aVF indicating an inferior STEMI. ST elevation in V4R indicating an associated RV infarction.



(E) CASE 5: POSTERIOR STEMİ WITH INFERIOR WALL INVOLVEMENT

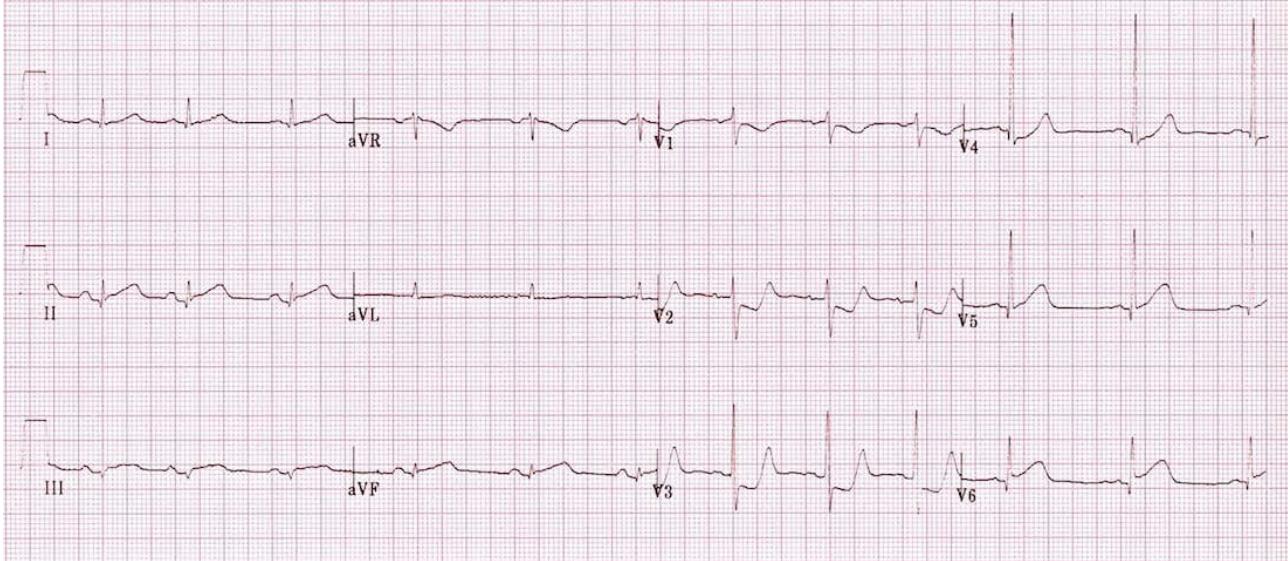


Figure 27. ECG strip of Case 5.

Table 7. ST segment deviation in ECG leads for Case 5.

ECG LEADS	ST SEGMENT DEVIATION
I	Unremarkable
II	Subtle ST↑ (~1 mm)
III	Subtle ST↑
aVR	Unremarkable
aVL	Unremarkable
aVF	Subtle ST↑
V ₁	TWI
V ₂	ST↓
V ₃	ST↓
V ₄	ST↓
V ₅	Unremarkable
V ₆	ST↑

- Subtle ST↑ in II, III and aVF but no reciprocal changes in I and aVL so there *may* be involvement of the inferior wall
 - Think of the blood supply: RCA supplies the right ventricle and the inferior portion and runs at the posterior portion to branch off as PDA that supplies the posterior heart
 - *Is there any involvement of the inferior wall that is posterior related?*
- Remember that the PDA gives way to the posterior and inferior portion of the heart
 - It does not pick up ST↑ in precordial V1-V6 chest leads. You would have to put the leads on the posterior part of the chest wall to pick up ST elevation

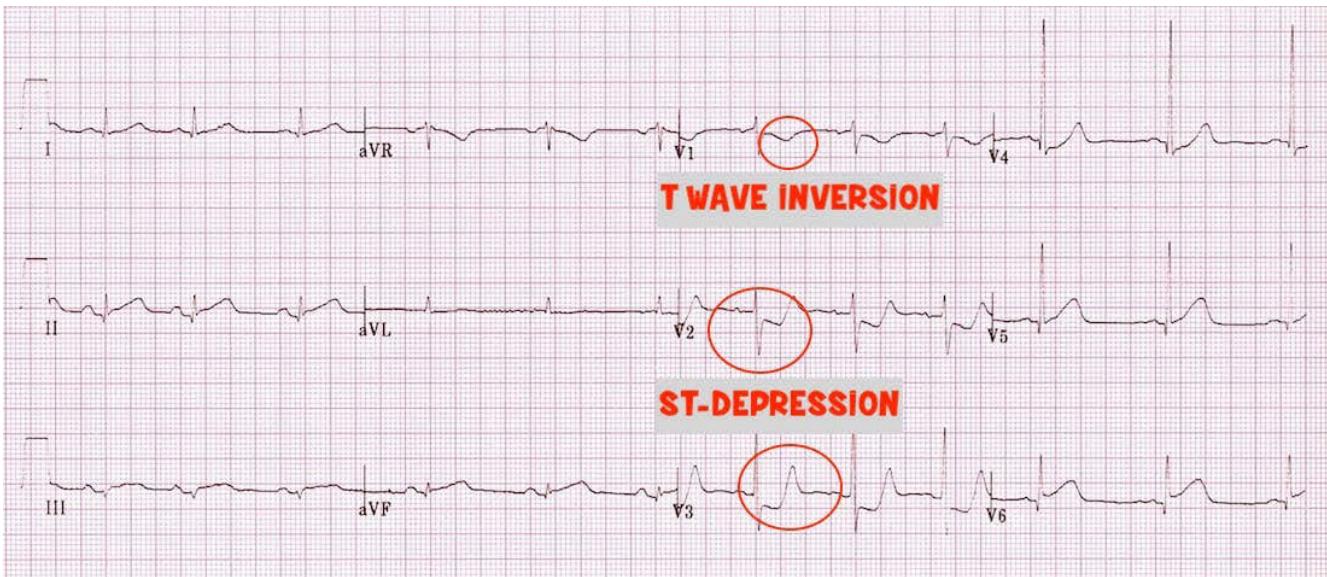


Figure 28. Labeled ECG strip of Case 5.



(1) Posterior Chest Leads (V7-V9)

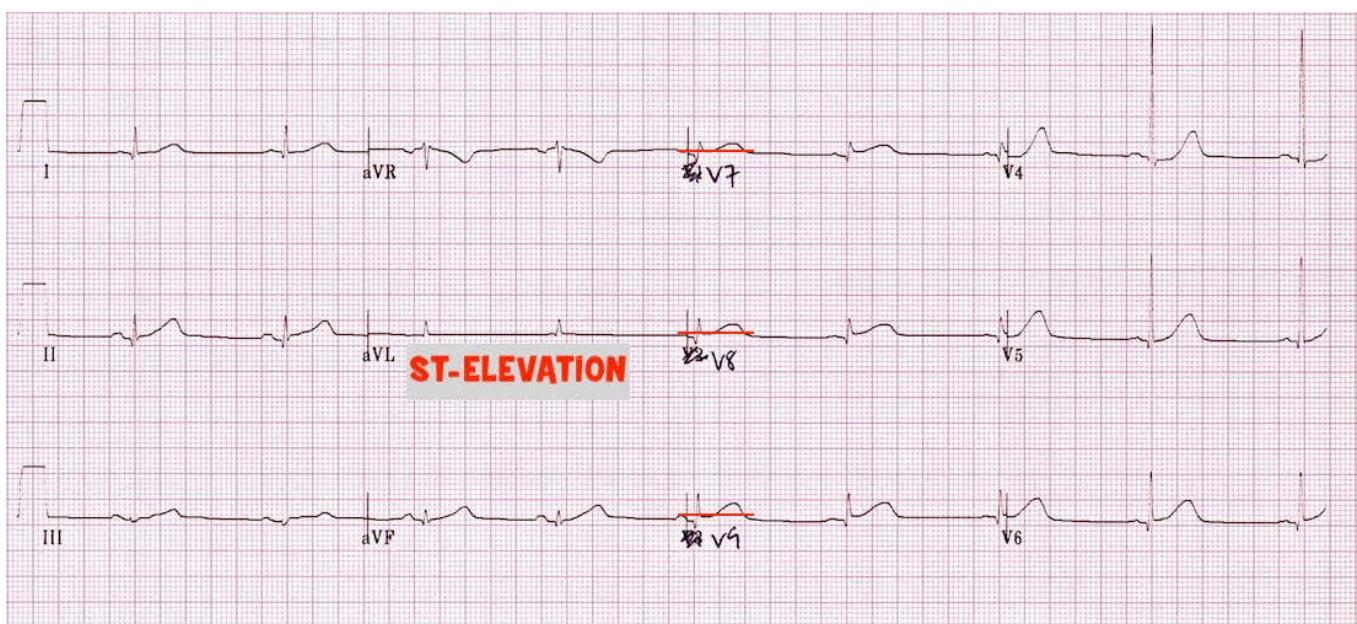
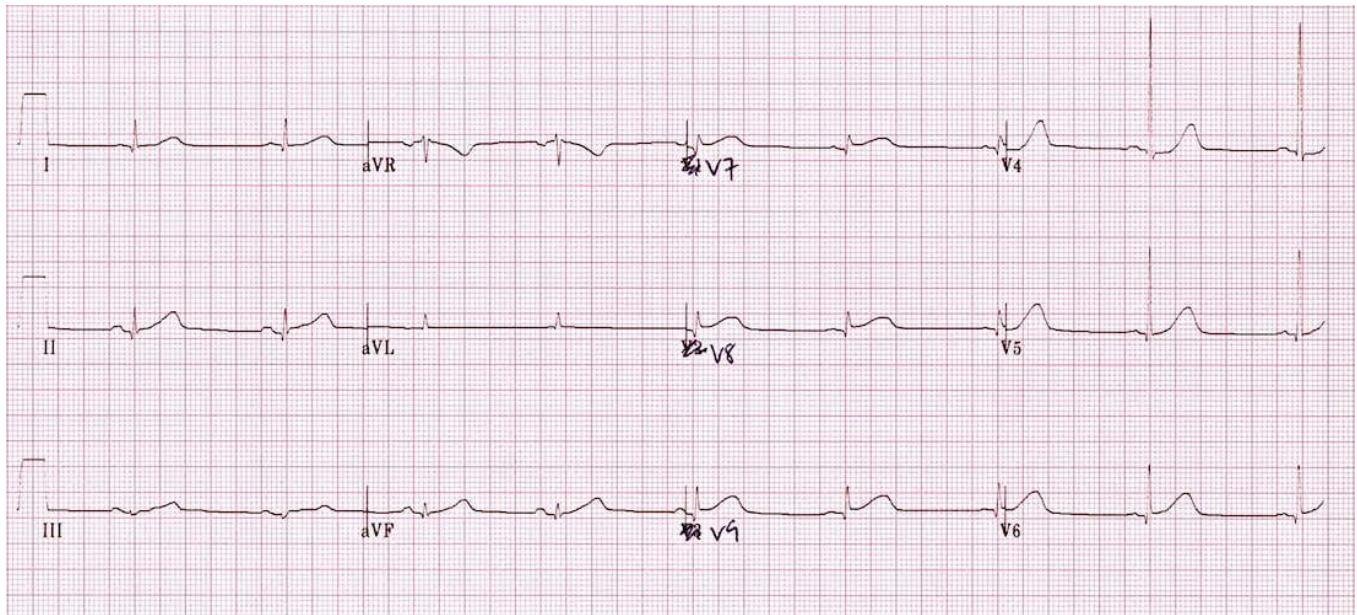


Figure 29. ECG strips (bottom: labeled) of Case 5 with posterior chest leads (V7-V9)

Table 8. ST segment deviation in posterior ECG leads for Case 5.

ECG LEADS	ST SEGMENT DEVIATION
II	Subtle ST↑ (~1 mm)
aVF	Subtle ST↑
V ₇	ST↑
V ₈	ST↑
V ₉	ST↑

- ST↑ in posterior chest leads, II and aVF = **posterior STEMI with inferior wall involvement**
 - There were ST↑ in chest leads that had ST↓ in the prior figure



VII) TREATMENT

(A) REDUCE RISK OF THROMBOSIS

- Goal:
 - ↓ risk of Plaque formation
 - ↓ risk of thrombosis
- Role of Platelets in forming Thrombosis:
 - Plaque is formed → rupture → exposure the thrombogenic core of fat tissue → platelets adhesion and activation
 - Platelet's granules secrete:
 - ADP
 - TXA₂ = Thromboxane A₂
 - 5-HT
 - These molecules can cause :
 - More platelets to come to the area = **Platelet Aggregation**
 - Increase the **stickiness** (Adhesion)
 - Stimulate the expression of proteins → Glycoproteins on the platelets like GPIIb / IIIa

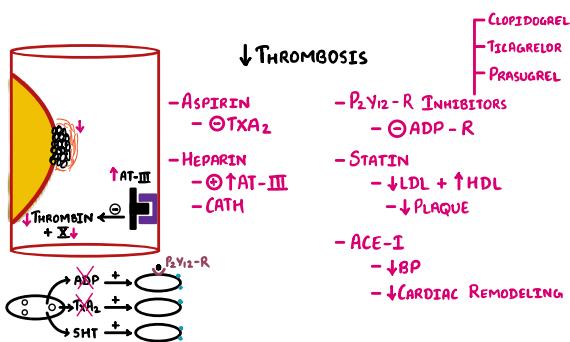


Figure 30. IHD Treatment- Reduce Thrombosis.

(1) Aspirin

- Antiplatelet
- Mechanism:
 - Irreversibly inhibits Cyclooxygenase-1 (COX-1) → Inhibits the production of **Thromboxane A₂ (TXA₂)** → **Inhibits Platelet Aggregation**
 - higher doses Aspirin → Inhibits COX-2 → Blocks prostaglandin production → analgesic and antipyretic effects

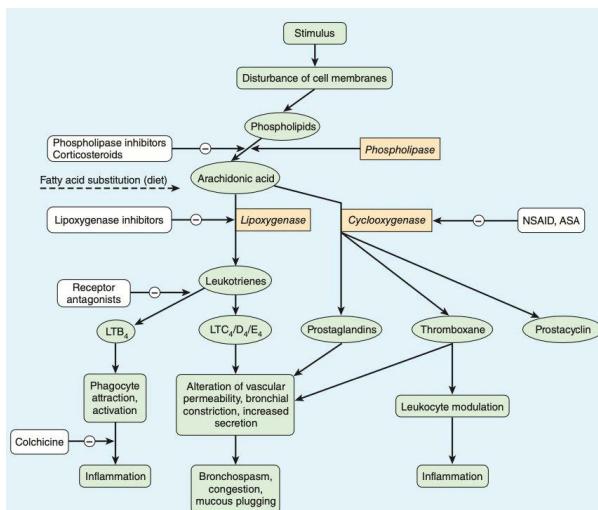


Figure 31. Prostanoid mediators derived from arachidonic acid and sites of drug action. ASA, acetylsalicylic acid (aspirin); LT, leukotriene; NSAID, nonsteroidal anti-inflammatory drug. (Pharmacology Katzung)

(2) P2Y₁₂ Receptor Inhibitors

- Antiplatelet
- ADP (= Adenosine Diphosphate) binds to P2Y₁₂ Receptors
- Medications in this group :
 - Clopidogrel = Plavix
 - Ticagrelor = Brilinta
 - Prasugrel
- Mechanism:
 - binds to the adenosine diphosphate (ADP) P2Y₁₂ receptor on the platelet surface → ↓ platelet aggregation and ↓ Stickiness

(3) Heparin

- Anticoagulant
- Heparin is produced endogenously
- Mechanism:
 - Binds to antithrombin III → enhance the activity of AT-III which is inhibition of **Thrombin and factor X** (These molecules induce coagulation) → decrease coagulation → decrease clot formation → decrease thrombosis
- Types of Heparins
 - Unfractionated Heparin → intravenous or subcutaneous use
 - Low Molecular Weight (LMW) Heparin → Preferred
- Heparin is beneficial in
 - STEMI, NSTEMI, Unstable Angina
 - Especially If you are getting **CATH**

(4) Statins

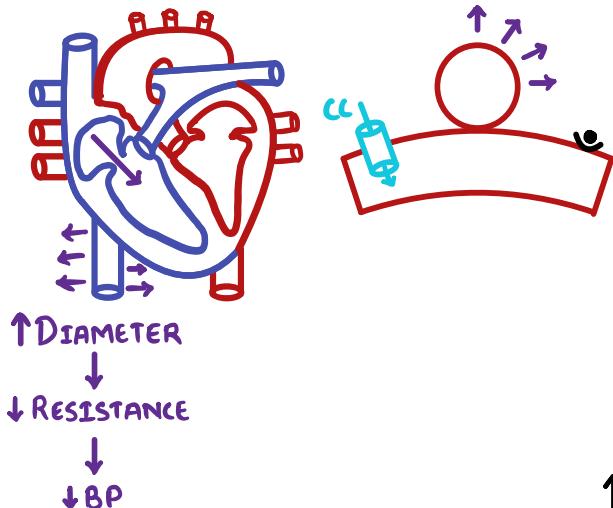
- Mechanism:
 - They do not necessarily reduce thrombosis, they ↓ Plaque formation
 - HMG-CoA reductase inhibitors → Ideally ↓ LDL & ↑HDL → ↓ Plaque formation → may ↓risk of forming future thrombosis

- **Modifiable Risk Factors:** (Reduce the risk of plaque forming)
 - Diabetes
 - Glucose control
 - Exercise
 - Diet
 - Hypertension
 - Medication
 - Lose weight
 - Exercise
 - Less salt intake
 - Obesity
 - Lose weight
 - Exercise
 - Diet
 - Smoking
 - Smoking cessation
 - Alcohol consumption
 - Reduce Alcohol use

ACE-I

- Is recommended in **long term** treatment of patients if they Blood pressure can tolerate
- Mechanism:
 - ↓ Blood pressure
 - ↓ Cardiac Remodeling (especially in patient who had MI)





↓ O₂ DEMAND / ↑ O₂ SUPPLY

- NITROGLYCERIN → ↓ PRELOAD → ↓ SV
⊖ RV-MI → CORONARY VASODILATOR

- MORPHINE → ↓ PRELOAD → ↓ SV
→ ↓ AFTERLOAD

- BETA BLOCKERS → ↓ CONTRACTILITY
(± CCB) → ↓ HR

- OXYGEN → If HYPOXIC

↑ REVASCULARISATION

Figure 32. IHD Treatment -Reduce Oxygen demand.

(1) Nitroglycerin

- Mechanism
 - Vasodilator (→ ↑ Diameter → ↓ resistance → ↓ BP)
 - Dilation of the **Venous system** → ↓ Preload → ↓ SV → ↓ O₂ demand
 - Vasodilation of the **coronary vessels** → increase perfusion of the ischemic cardiac tissue
- Contraindication
 - Avoided in patients who are **PRELOAD** dependent such as **Right Ventricular MI**
 - Phosphodiesterase inhibitor
 - Aortic Stenosis

(2) Morphine

- Mechanism
 - Vasodilator
 - Venous Dilation → ↓ Preload → ↓ SV → ↓ O₂ demand
 - Arteries Dilation → ↓ Resistance → ↓ Afterload → ↓ O₂ demand
- Some studies suggest it may have adverse effect on outcome
 - For refractory Ischemic patients
 - Reserved for patients with an unacceptable level of pain

(3) Beta Blockers

- Mechanism
 - Beta-1 receptors locations:
 - SA node, AV nodes and Bundle system

- Contractile unit of the Myocardium
- Beta-Blockers
 - Block Beta-1 receptors on the contractile unit of the myocardium → ↓ contractility → ↓ O₂ demand
 - Inhibit conduction system → ↓ HR → ↓ O₂ demand

(4) Calcium Channel Blockers

- Sometimes can be added to the treatment plan
- Mostly Dihydropyridines such as
 - Amlodipine
 - Nicardipine
 - Nimodipine
 - Nifedipine
- Mechanism
 - Act mainly on Vascular Smooth Muscle → Vasodilation of the arteries → ↓ Afterload → ↓ O₂ demand
 - Vasodilation of the coronary arteries
 - Some of them may block calcium channels on the heart and may reduce HR → ↓ O₂ demand
- Contraindications
 - Decompensated heart failure (Weak Heart)

(5) Oxygen

- In patients with Hypoxia → Supplemental O₂ → ↑ O₂ supply



(C) REVASCULARIZATION

- Overall Indications:
 - Severe chest pain
 - STEMI
 - NSTEMI and Unstable Angina refractory to medical treatment
 - Start developing complications such as :
 - Heart Failure
 - Left Ventricular dysfunction

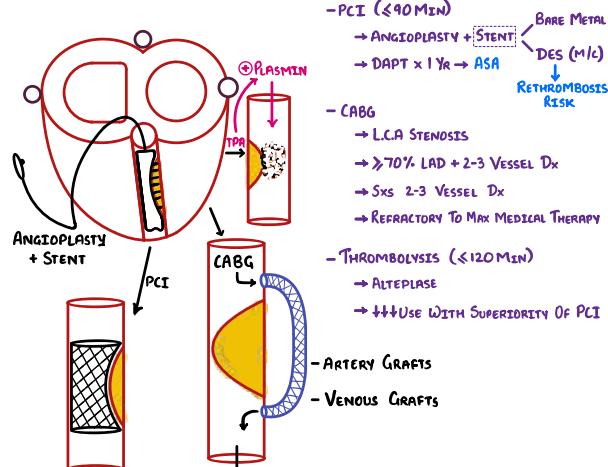


Figure 33. IHD Treatment- Revascularization.

(1) Percutaneous Coronary Intervention (PCI)

- Timeframe ≤ 90 mins
 - door-to-balloon time
 - time from presentation at a percutaneous coronary intervention (PCI) center to first balloon inflation

Indications:

- Preferred for patients with STEMI, NSTEMI, Unstable Angina with elevated troponin and high TIMI score not benefited from medical therapy
- Stable Angina ≥ 70 % stenosis , positive stress test
- Hemodynamically unstable

Mechanism:

- Angioplasty + Stent

Selection of Stent type:

- bare-metal stents (BMS)
- Drug-eluting stents (DES) (Most common, preferred)
 - antirestenotic drug → inhibit growth around the stent → prevent excessive intimal hyperplasia → reduce the risk of restenosis
- Common complication with stents → **Re-thrombosis**
 - Dual Antiplatelet Therapy (DAPT) for at least a year
 - Clopidogrel + ASA
 - Ticagrelor + ASA
 - After a year the risk of restenosis and Re-thrombosis may reduce but should stay on Aspirin

(2) Coronary Artery Bypass Graft surgery (CABG)

Types of graft:

- Arterial grafts
 - Radial Artery
 - Internal Thoracic Artery
- Venous grafts (preferred)
 - Saphenous vein

Indications:

- Left main stem Coronary Artery Stenosis
- Significant stenosis (≥ 70%) of the proximal left anterior descending artery, with 2-vessel or 3-vessel occlusion
- Severe Chest Pain + 2-3 Vessels disease
- NSTEMI, Unstable Angina not responding to maximal medical treatment

(3) Thrombolysis

- Least preferred
- Timeframe ≤ 120 mins
 - patient's presentation and delivery of fibrinolytic therapy (door-to-needle time)

Alteplase

Mechanism

- TPA=Tissue Plasminogen Activator → plasmin → fibrinolysis

Efficacy

- is inferior to PCI
- if timely primary PCI is not available → TPA → Normally after TPA the patient needs to have angiography and PCI

Contraindications

- Active bleeding
- Prior intracranial hemorrhage
- Recent surgery
- Severe hypertension

Mnemonic for Medical Management of Ischemic

Heart Diseases

- MONA BASH-C
 - Morphine
 - Oxygen
 - Nitroglycerin
 - Aspirin
 - Beta Blockers
 - ACE-I
 - Statin
 - Heparin
 - Clopidogrel and other P2Y₁₂ Receptor inhibitors



VIII) APPENDIX

Table -9. Presentation of IHD when taking history

History (OPQRST)	Stable Angina
Onset	Stable Angina: 5-10 mins Unstable angina, NSTEMI and STEMI (Acute Coronary Syndrome): >10 mins
Provocation	Worse on exertion, improved on rest, also improves with Nitroglycerin → Suggestive of Stable Angina When it occurs at rest, but can intensify with exertion → Unstable angina, NSTEMI and STEMI (Acute Coronary Syndrome)
Quality	Squeezing / Stabbing/ Pressure “Feeling like an elephant is sitting on your chest”
Radiation	Left Arm, Left Neck, Left Jaw <i>Epigastric pain can be indicative of inferior STEM</i>
Severity	Variable
Time	Constant

Table -10 RV vs LV MI clinical features

Right Ventricular MI	Left Ventricular MI
Lungs are clear	Pulmonary Edema
↓Preload → ↓SV → ↓CO → ↓BP (HOTN)	↓EF (Ejection Fraction) → HOTN
Jugular Venous Distension (JVD)	↓Perfusion → Cold, mottled skin, Pallor
Hepatomegaly + Ascites	
Edema in the lower extremities	
Sinus Bradycardia OR	S4 heart sound
AV block (degree depends on severity)	Reflex Tachycardia (↑HR)

Table -11. Summary of the coronary vessels and the leads involved when infarction occurs.

ARTERY	HEART PORTION SUPPLIED	LEADS INVOLVED
LAD	Anterior	V ₁ -V ₄
LCX	Lateral	I, aVL, V ₅ , V ₆
RCA	Inferior	II, III, aVF, right-sided leads
PDA	Posterior	V ₇ -V ₉

Table -12 Wide table sample

Type of IHD	Ischemia/Infarction	EKG	Troponin, CMB
Stable angina	Subendocardial ischemia	No ST ↑	(-)
Prinzmetal (vasospastic) angina	Transmural ischemia	ST ↑	(-)
Unstable angina	Subendocardial ischemia	ST ↓ + TWI	(-)
NSTEMI	Subendocardial infarction	ST ↓ + TWI	(+)
STEMI	Transmural infarction	ST ↑↑	(+)

Table -13 Categories of Ischemic Heart Disease

	Unstable Angina	Stable Angina	Subendocardial Infarct	Transmural Infarct
Plaque	Stable plaque	Unstable plaque	Unstable plaque	Unstable plaque
Occlusion	≥ 70%	≥ 90% Near total occlusion	≥ 90% Near total occlusion	100% Total occlusion
Clinical Features	Chest pain with exertion	Chest pain at rest; worsens with exertion	Chest pain at rest; worsens with exertion	Chest pain at rest; severe with exertion
Blood Flow	Subendocardial Ischemia	Subendocardial Ischemia	Subendocardial Infarction	Transmural Infarction (entire myocardium is affected)



Table 14. Summary of the Complications of Ischemic Heart Disease

Time	Complication	Mechanism	Clinical Manifestations	
0-24 hours	Sudden Cardiac Death	↑ cell permeability → ↑ cations load → depolarizes cells abnormally → ventricles contract abnormally → PVC → VT → VF → in Sudden Cardiac Death	Death	<p>→ SCD → PVC's → VT → VF</p>
	Cardiogenic Shock	Occlusions of major coronary vessels → ↓ output to the systemic circulation resulting in hypotension → hypoperfusion → cardiogenic Shock	Cold Extremities	<p>→ ACUTE HF → HOTN → SHOCK → COLD EXT.</p>
	Flash Pulmonary Edema	Blood backs up into the pulmonary circulation leading to flash pulmonary edema	Shortness of breath, hypoxia Reflex Tachycardia	<p>→ FLASH PULMONARY EDEMA → REFLEX ↑ HR</p>
24 hours – 3 days	Rupture Syndrome	Infarction in the interventricular septum → Rupture → Ventricular Septal Defect	Murmur (hollow systolic murmur) Mixed venous oxygenation	<p>→ RUPTURE SYNDROMES 1) VSD - MURMUR ⊕ - MIXED VENOUS O₂</p>
		Infarction in the left ventricle → ruptures → blood can get into the pericardial cavity → Cardiac Tamponade	Beck's Triad occurs with cardiac tamponade, this consists of hypotension, JVD, and muffled heart sounds	<p>2) FREE WALL RUPTURE - TAMPOONADE</p>
		Infarction in the papillary muscles → the tissue can get weak and rupture → valves flail causing Mitral Valve Regurgitation	Mitral valve murmur Hypotension Pulmonary Edema	<p>3) PAPILLARY RUPTURE - MR MURMUR</p>
	Left Ventricle Pseudoaneurysm	In free wall ruptures, blood can accumulate in the pericardium and this can form a pseudoaneurysm	Mural thrombus	<p>4) LV PSEUDOANEURYSM - ↑ THROMBI</p>
3 days – 14 days	Pericarditis	Infarction in the left ventricle leads to a local inflammatory reaction to clean up the debris ➤ The inflammation can increase the formation of fluid, leading to pericardial effusion	Sharp & Positional Chest Pain Friction Rub (Via Lung Sounds) Pericardial Effusion	<p>→ PERICARDITIS → CP - SHARP - POSITIONAL - FRICTIONAL RUB - PERICARDIAL EFFUSIONS</p>
14 days – 1 month	Left Ventricular Aneurysm	In an infarction, soft granulation tissue turns to fibrous tissue. ➤ Fibrous tissue can "bulge out" creating a left ventricular aneurysm ➤ <i>Blood stasis</i> of the affected area increases the risk of mural thrombi and emboli .	Shortness of breath Organ Damage	<p>→ LV ANEURYSM - MURAL THROMBI - ↑ EMBOLI</p>
	Dressler's Syndrome (Pericarditis)	Infarction in the ventricles can create an underlying immune reaction, however the etiology is unknown ➤ The immune system develops antibodies against the pericardium → inflammation of the pericardium	Chest Pain Pericardial Effusion Dyspnea	<p>→ DRESSLER SYND. - EMBOLI</p>



IX) REVIEW QUESTIONS

1) Which of the following patients is at risk for atherosclerosis?

- a) 58-year-old male with diabetes
- b) 42-year-old female smoker
- c) 31-year-old male with a BMI of 35
- d) All of the Above

2) Which of the following characterizes stable angina?

- a) Squeezing chest pain which occurs at rest
- b) Plaque with a strong fibrous cap
- c) ST elevation on EKG
- d) Near total occlusion of coronary vessel

3) Which of the following is incorrect about subendocardial infarct?

- a) Chest pain can occur at rest
- b) There is thrombus formation
- c) There is near total occlusion of coronary vessel
- d) Unreversed ischemia was present for at least 2 hours

4) The following are categorized under Acute Coronary Syndrome except:

- a) Stable Angina
- b) Unstable Angina
- c) NSTEMI
- d) STEMI

5) Which of the following medications should be avoided in patients with Prinzmetal's angina?

- a) Amlodipine
- b) Nitroglycerin
- c) Metoprolol
- d) Nifedipine

6) The EKG of a patient complaining of chest pain shows depression of the ST segment and T wave inversion. Her troponin level is elevated. The patient might have:

- a) Subendocardial ischemia
- b) Transmural ischemia
- c) Subendocardial infarction
- d) Transmural infarction

7) Which of the following should be ordered once a patient who was admitted for MI experiences another chest pain?

- a) EKG
- b) Troponin
- c) CK-MB
- d) ANP

8) If the patient has ischemia on the inferior part of the heart, which of the following coronary vessels is involved?

- a) LAD
- b) LCX
- c) RCA
- d) PDA

9) A patient undergoing stress test has a baseline myocardial perfusion scan. Which of the following drugs should NOT be used to replicate stress?

- a) Adenosine
- b) Dobutamine
- c) Diperidamole
- d) None of the above

10) An ECG shows ST elevation in II, III and aVF. What STEMI is indicated?

- a) Posterior STEMI
- b) Inferior STEMI
- c) Lateral STEMI
- d) Anterior STEMI

11) Aspirin is effective in ischemic heart disease treatment because:

- a) Inhibition of ADP production
- b) Analgesic effect
- c) Inhibition of TXA2 production
- d) Antipyretic effect

12) Which statement is false about the medications used in ischemic heart disease?

- a) Beta blockers decrease the Oxygen demand
- b) Morphine is contraindicated in Right Ventricular MI patients
- c) Nitroglycerin increase the perfusion to the Ischemic tissue
- d) Calcium Channel Blockers should be avoided in Decompensated Heart Failure patients

13) If a patient is presented with severe chest pain and occlusion of 3 vessels, which Revascularization approach is recommended?

- a) PCI
- b) CABG
- c) TPA+ PCI
- d) TPA

X) REFERENCES

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