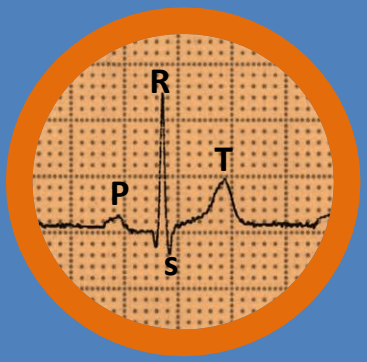

ECG Review : Some momentous articles on 'Coronary artery disease'

(For Academic Purpose Only)

Dr. DURGA PRASAD KHAITAN

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A hidden shadow narrows the lumen inside
Such danger the heart can no longer hide .....

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A hidden shadow narrows the lumen inside
Such danger the heart can no longer hide ….

Knowledge and skill in the field of electrocardiography are constantly changing with the new researches and understanding.

With humble words I wish to say that some momentous articles related to 'Coronary artery disease' are being covered within this book.. It is only a step towards the vast ocean of knowledge. I may be excused for any error or omission.

With thanks and regards

DEDICATED
TO
FELLOW COLLEAGUES

Let us have smiling hearts and peaceful minds, filled with love and knowledge

Index

STEMI : PATHOPHYSIOLOGICAL BASIS AND EVOLUTION OF ECG CHANGES

ECG

STEMI : PATHOPHYSIOLOGICAL BASIS AND EVOLUTION OF ECG CHANGES

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OUTLINE

Introduction
A STEMI (ST-elevation myocardial infarction) represents the electrical manifestation of acute transmural myocardial injury resulting from abrupt coronary artery occlusion.

Pathophysiological basis

- Shift to anaerobic metabolism → ATP depletion → Failure of ionic pump → Extracellular K+ accumulation → hyperacute T-wave
- Evolving transmural necrosis→ ST-segment elevation
- Completion of transmural necrosis → pathological Q wave

Evolution of ECG changes
Hyperacute T-wave , Coved ST-segment elevation , Pathological Q wave , Symmetrical T-wave inversion

A practical approach to STEMI diagnosis on ECG
In practice , STEMI is fundamentally an ECG diagnosis requiring immediate reperfusion therapy.

Illustration by ECG

Take-Home Message

References

STEMI : Pathophysiological Basis and Evolution of ECG Changes

A Narrative Review

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The battle line between good and evil runs through the heart of every human being. One has to be pure by the heart if the seed of humanity is to be planted and greened on the earth. One has to think over how unpleasant environment to be kicked out, and pleasant environment, thus should be created – it would be a marvellous gift to the suffering humanity.

Similarly the human heart behaves in the same way. If the heart is healthy, the life is comedy and if the heart is unhealthy , the life is tragedy. Since coronary arteries deliver blood to the cardiac muscle, its narrowing can have a catastrophic effect by reducing the flow of oxygen and the nutrients to the heart. Atherosclerosis is the most leading cause of such situation.

- ☐ **A STEMI (ST-elevation myocardial infarction) represents the electrical manifestation of acute transmural myocardial injury resulting from abrupt coronary artery occlusion.**
- ☐ **This is not a static event but a dynamic continuum evolving from ischemia to injury and ultimately to infarction.**

Thus, the ECG becomes more than a tracing on paper—it is the visible electrical expression of an invisible coronary catastrophe, whispering vital clues into the ears of the clinician about the evolving battle within the myocardium.

1. Introduction (Keypoints)

- ○ ST-elevation myocarial infarction (STEMI) is the story of one of the most dramatic electrical manifestations of acute coronary artery occlusion. Traditionally recognized through characteristic ST-segment elevation on the electrocardiogram (ECG), STEMI dictates more than an isolated waveform abnormality; rather, **it is a dynamic electrophysiological foot-steps of evolving myocardial ischemia, injury, and infarction.**
- ○ Acute interruption of coronary blood flow boosts a cascade of metabolic and ionic disturbances resulting from myocardial ischemia. As oxygen deprivation steps ahead, depletion of intracellular ATP, dysfunction of membrane ionic pumps, extracellular potassium accumulation, with altered transmembrane electrical gradients collectively reshape myocardial depolarization and repolarization.
- ○ These evolving cellular events become progressively visible on the ECG as hyperacute T waves, coved ST-segment elevation, pathological Q waves, and symmetrical T-wave inversion.

- Thus , STEMI may be considered not merely as an ECG diagnosis , but as an advancing STEP of visible electrical evolution of an invisible coronary catastrophe. Understanding this dynamic continuum keeps the clinicians in a position to differentiate it from STEMI mimics and also empowers their minds to have a meaningful interpretation of electrocardiographic changes.
- However, STEMI evolution rarely proceeds through rigidity separated stages. Ischemia, injury , and infarction frequently overlap both spatially and temporally, producing dynamic and heterogeneous ECG patterns. In other words, **in rapidly evolving infarctions, multiple electrophysiological phases may coexist within the same lead on ECG.**

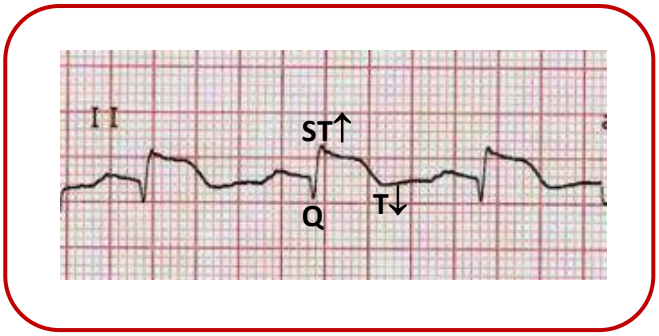


Fig 1.1

2. Pathophysiological basis

Here is a brief description of the pathophysiological changes in ST-elevation myocardial infarction (STEMI), usually atherosclerotic artery is the main victim :

(i) **Plaque Rupture & Thrombosis :** A vulnerable atherosclerotic plaque ruptures, exposing its lipid core. This triggers rapid platelet activation, its aggregation, and fibrin deposition, forming a thrombus that usually occludes the coronary artery completely.

(ii) **Acute Ischemia & Metabolic Shift :** Transmural blood flow ceases. Cardiomyocytes get shifted from aerobic to anaerobic metabolism, depleting ATP , causing a rapid decrease in its contractility, and inducing hyperacute T-waves.

- This phase of Acute ischemia & metabolic shift represents an early hallmark event before cell death becomes permanent – occuring within minutes after abrupt coronary artery occlusion.

(iii) **Irreversible Cell Death :** Persistent ischemia causes irreversible necrosis, starting in the subendocardium and spreading to the epicardium (transmural progression of necrosis). Cardiac troponins start to leak into the bloodstream.

- Key physiological message: The “transmural progression” means time is critical – 50% of the at-risk myocardium is lost within the first hour.

(iv) **Ventricular Remodeling (Days to Months):** Damaged tissue is transformed into granulation tissue, replaced by fibrosis, leaving a permanent scar. The ventricle may remodel (change shape/size) accordingly to compensate for lost myocardium.

Summary sketch of Pathophysiological changes :

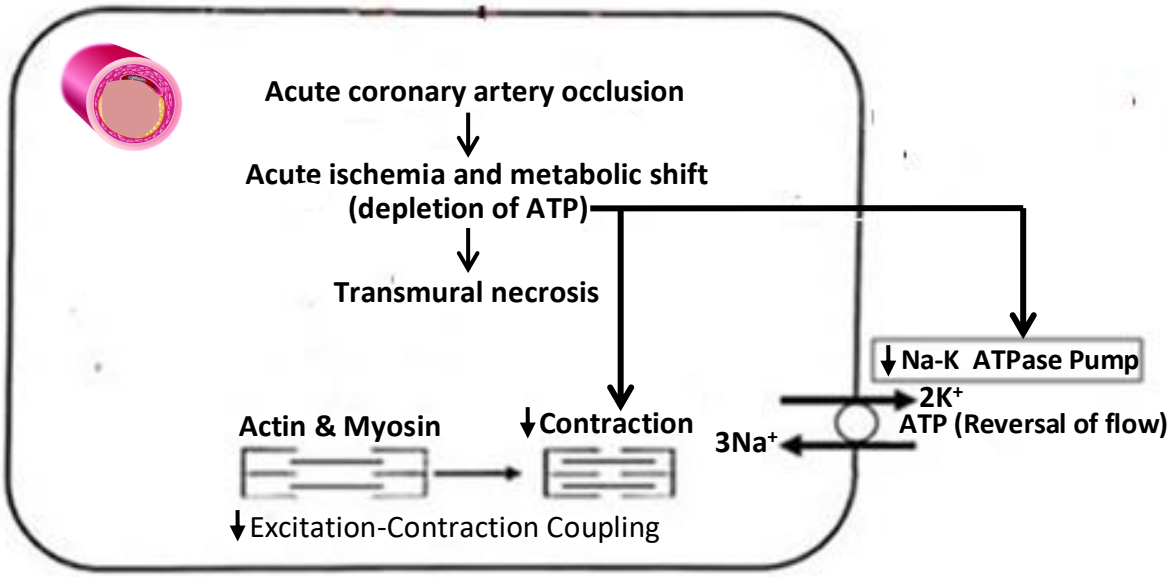


Fig 1.2

Key takeaway : Coronary artery occlusion shows a "wave-front" of myocardial injury that spreads from the subendocardial myocardium to the subepicardial myocardium. The pathophysiological cycle starts with the following changes , without understanding these events the clinician is unable to properly frame the clinical scenario in mind.

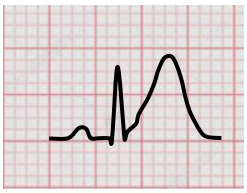
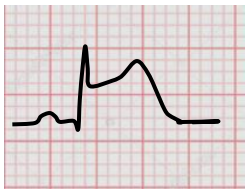
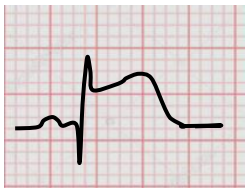
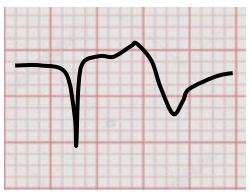
- Shift to anaerobic metabolism → ATP depletion → Failure of ionic pump → Extracellular K+ accumulation → hyperacute T-wave
- Evolving transmural necrosis→ ST-segment elevation
- Completion of transmural necrosis → pathological Q wave

3. Evolution of ECG changes

These evolving cellular events become progressively visible on the ECG as hyperacute T waves, coved ST-segment elevation, pathological Q waves, and symmetrical T-wave inversion. **The authenticity of STEMI lies not in isolated ECG findings, but in the dynamic orchestration of evolving electrical events.** These changes happens to occur rapidly , often within minutes to hours of coronary occlusion. The attending clinician should appreciate the ECG findings that follow the evolution of an STEMI with a sound understanding of the accompanying pathophysiology in the background of clinical scenario.

During acute coronary occlusion, the ischemic myocardium initially attempts to maintain cellular viability by utilizing its limited glycogen reserves through anaerobic glycolysis; however, this compensatory mechanism is short-lived and can sustain myocardial survival only briefly unless sufficient collateral coronary circulation is present.

Evolution of ECG changes and the corresponding contributory factors are enumerated in the following table :

ECG changes	Contributory factors
• Hyperacute T-wave 	Extracellular accumulation of potassium (K+) released from ischemic myocardial cells.
• Coved ST-segment elevation 	There exists voltage gradients between injured and relatively preserved regions, generating injury currents directed toward the ischemic zone, which are manifested on the ECG as coved ST-segment elevation (J-point elevation).
• Pathological Q wave 	Transmural extension of myocardial necrosis progressively abolishes local depolarization vectors, leading to attenuation of the R wave. Ultimately, the infarcted myocardium becomes electrically inert, and the ECG records the reciprocal unopposed forces from the contralateral viable myocardium as a pathological Q wave.
• Symmetrical T-wave inversion 	Surrounding this necrotic core lies a border region of injured and ischemic myocardium with relatively homogeneous repolarization abnormalities, resulting in the inscription of symmetrical T-wave inversion

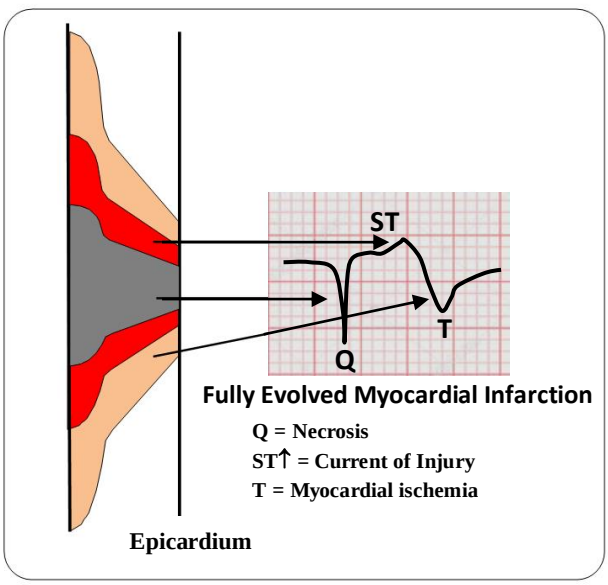


Fig 1.3

The sideway sketch is more or less in pyramidal shape with braod base towards endocardium and narrower base towards epicardium. An exploring lead placed over the infarct area would be the picking lead of such electrocardiographic changes , just underneath myocardium.

Modern criteria define **pathological Q wave** as Q wave $\geq$ 0.03 seconds in duration and / or $\geq$ 0.1 mV in depth in two contiguous leads , with even smaller Q waves in V2-V3 considered abnormal.

NB :

- ❒ Hyperacute T waves announce ischemia, coved ST elevation reflects active injury, pathological Q waves signify irreversible necrosis, and symmetrical T-wave inversion reveals the surviving peri-infarction myocardium.
- ❒ As viable myocardium is progressively lost:
  the R wave diminishes
  while the Q wave deepens/widens
  Thus, there is often an inverse relationship between them.
  The evolving balance between diminishing R-wave forces and expanding Q-wave dominance reflects the electrophysiological progression of transmural myocardial necrosis.
- ❒ In certain STEMI cases, the electrophysiological evolution may proceed with remarkable rapidity, with ischemic, injury, and infarction patterns evolving and overlapping within a span of only a few hours.
  Rapid completion of STEMI evolution occurs in the setting of abrupt total coronary occlusion, minimal collateral circulation, and accelerated transmural necrosis.
- ❒ The observer's mind must translate electrocardiographic patterns into the underlying pathological events occurring within the myocardium. ST depression in leads opposite the affected area further helps in establishing the diagnosis of STEMI.

4. A practical approach to STEMI diagnosis on ECG

- ➢ ECG criteria for STEMI
  ST-segment elevation measured at the J-point in at least two contiguous leads:
  - ≥1 mm (0.1 mV) in leads other than V2–V3
  - In V2–V3:
    - ≥2.5 mm in men <40 years
    - ≥2.0 mm in men ≥40 years
    - ≥1.5 mm in women

- ➢ Additional STEMI equivalents include: (please see the texts for detail)
  - New posterior MI pattern
  - New/presumed new LBBB with ischemic presentation
  - Right ventricular STEMI patterns.

- ➢ The biochemical confirmation of STEMI relies primarily on **high-sensitivity cardiac troponin (hs-cTnI or hs-cTnT)**.
  - Coronary occlusion occurs first , ischemia develops immediately and troponin rises only after myocyte membrane disruption.
  - A **rise and/or fall** of cardiac troponin values, and at least one troponin value above the 99th percentile URL

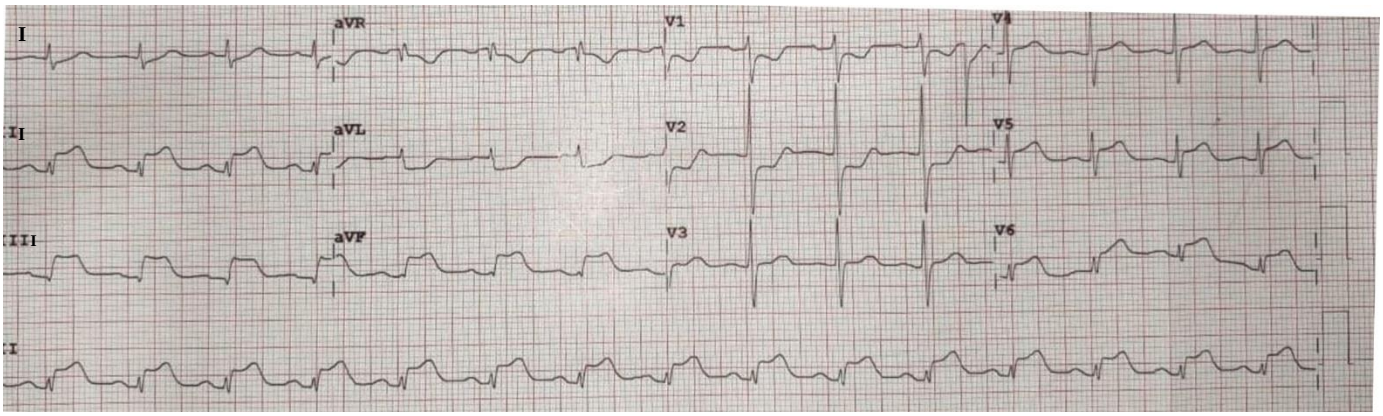
Temporal Profile of Cardiac Biomarkers

Cardiac biomarkers	Rise	Peak	Return to normal
Troponin I/ T	1-3 hours	12-48 hours	7-10 days
CK-MB	3-6 hours	18-24 hours	2-3 days

NB : Different laboratories have different cutoff values of these biomarkers. CK-MB → supplementary or alternative marker when troponin testing is unavailable or when reinfarction is suspected.

- **In practice, STEMI is fundamentally an ECG diagnosis requiring immediate reperfusion therapy. Treatment should not be delayed while waiting for enzyme elevation because coronary occlusion precedes detectable biomarker release.**

5. Illustration by ECG



Source : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Prinicipal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

(This ECG belongs to a middle-aged male presenting with severe chest pain since 3 hours).

Findings on ECG :

- ECG shows ST elevation (>1mm) in V4 - V6 and inferior leads II , III and aVF plus the presence of Q wave in inferior leads.
- Reciprocal ST depression in lead I , aVL and V1-V2.

These findings are suggestive of proximal left circumflex artery occlusion.

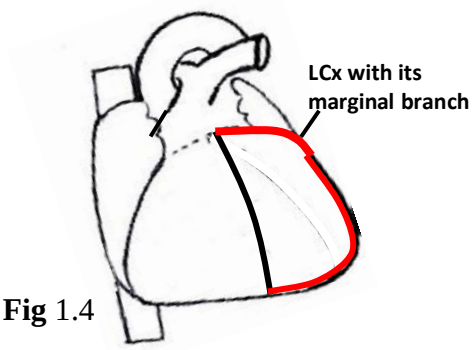


Fig 1.4

Left circumflex artery supplies :

- Inferior wall (leads II , III and aVF)
- Lateral LV territory (V5-V6)

☐ The injury vector (ST vector) is predominantly directed towards the inferior leads , but away from I and aVL. That's why , reciprocal ST depression is noted in leads I and aVL.

☐ The same holds true with V1 and V2 – ST segment quite away from those of V4-V6 leads, and hence , with reciprocal ST depression therein.

6. Take-Home Message

- ☐ A STEMI (ST-elevation myocardial infarction) represents the electrical manifestation of acute transmural myocardial injury resulting from abrupt coronary artery occlusion.
- ☐ ECG changes become progressively visible as hyperacute T wave, coved ST-segment elevation, pathological Q waves, and symmetrical T-wave inversion.
- ☐ Evolution of pathophysiological changes :
  - Shift to anaerobic metabolism → ATP depletion → Failure of ionic pump → Extracellular K+ accumulation → hyperacute T-wave
  - Evolving transmural necrosis→ ST-segment elevation
  - Completion of transmural necrosis → pathological Q wave
- ☐ In certain STEMI cases, the electrophysiological evolution may proceed with remarkable rapidity, with ischemic, injury, and infarction patterns evolving and overlapping within a span of only a few hours.
  Rapid completion of STEMI evolution occurs in the setting of abrupt total coronary occlusion, minimal collateral circulation, and accelerated transmural necrosis.
- ☐ In practice, STEMI is fundamentally an ECG diagnosis requiring immediate reperfusion therapy. Treatment should not be delayed while waiting for enzyme elevation because coronary occlusion precedes detectable biomarker release.

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ELECTROCARDIOGRAPHIC MAPPING OF CORONARY ARTERY TERRITORIES : A LEAD-WISE INTERPRETATION

ECG

ELECTROCARDIOGRAPHIC MAPPING OF CORONARY ARTERY TERRITORIES : A LEAD-WISE INTERPRETATION

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OUTLINE

Electrocardiographic Mapping of Coronary Artery Territories : A Lead-wise Interpretation

A Narrative Review

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A map is a representation of a territory—an invaluable guide that helps us approach a defined destination with purposeful action. It serves as an essential tool for identifying pathways and navigating toward the required intervention.
In a similar manner, electrocardiographic lead patterns provide a non-invasive map of myocardial perfusion, enabling the localization of coronary artery involvement.

- **Every deviation on the ECG reflects a deeper truth—that the vitality of the heart depends on the uninterrupted flow of blood.**
- **Coronary arterial territories define the spatial distribution of its supply, mirrored as lead-wise changes observed on the electrocardiogram.**

The electrocardiogram is not merely an electrical tracing; rather, through the delicate interplay of blood flow and electrical activity, it becomes a functional map of the living heart—guiding the clinician to localize the site of coronary insufficiency and act accordingly.
It is aptly said that each ECG lead observes the heart from a different window, capturing the subtle signatures of perfusion—revealing whether the myocardium is being adequately nourished or deprived.

1. Introduction (Keypoints)

- The electrocardiogram is not merely an electrical tracing but a physiological narrative, where the distribution of coronary blood flow is translated into patterns of depolarization and repolarization. In the delicate interplay of blood and current, the electrocardiogram becomes a canvas of the living heart.
- Since coronary arteries deliver blood to the cardiac muscle , its narrowing can have a catastrophic effect by reducing the flow of oxygen and nutrients to the heart. Atherosclerosis is the most leading cause of such situation.
- The interpretation of ECG in ‘coronary insufficiency spectrum’ is deeply rooted in an understanding of coronary anatomy and myocardial electrophysiology. Each ECG lead records electrical activity as a specific change, reflecting the physiological state of the underlying myocardial region. Since myocardial perfusion is governed by coronary arterial supply, such alterations in electrical activity manifest as region-specific ECG changes.
  Thus, the ECG can be viewed as a surface projection of coronary perfusion dynamics, enabling clinicians to localize the site of coronary insufficiency and infer the likely coronary artery involved.

- Here to mention that the localization of the culprit artery is generally well-correlated with specific coronary vessels ; however , because of vessel heterogeneity , the accurate vessel involvement in such a situation needs to be verified by coronary angiogram or other imaging modalities.
- This article provides an integrated overview of coronary circulation and its reflection on ECG through a lead-wise approach. By correlating myocardial territories with corresponding ECG leads, a systematic framework is presented to aid in accurate localization of coronary artery lesions.

2. Blood supply to the heart (Coronary circulation)

Coronary blood supply shapes the electrical behavior of the myocardium, and the ECG serves as its visible signature.

The concept of coronary circulation is essential to localize the culprit artery of coronary occlusion.

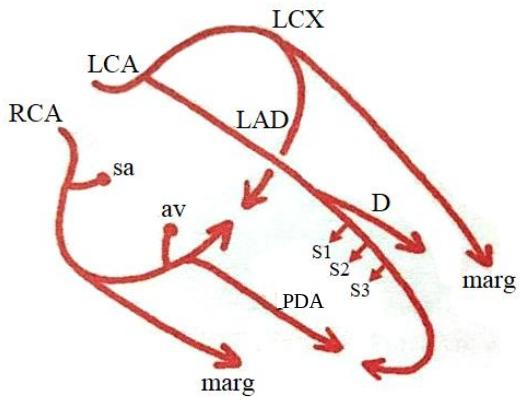


Fig. 1.1 **Coronary Circulation**

- Right coronary artery arises from the right aortic sinus and the left coronary artery from left aortic sinus , just above the aortic valve ring.
- The right coronary artery mainly supplies right atrium , SA node, AV node and right ventricle , including the lateral and inferior walls and the posterior third of the interventricular septum. Left coronary artery supplies mainly the left atrium , left ventricle , including the anterior wall , apex , and anterior interventricular septum. Left circumflex artery assists in this distribution.

- Right coronary artery distribution

Anterior	Posterior
• Right atrium • SA node (in 60%) • Right marginal artery → to the inferior border • Right anterior ventricular branches	• AV node • Right posterior ventricular branches • Posterior interventricular branch (via PDA)

- Left coronary artery distribution

LAD	LCX
• Septal branches - S →anteroseptal region • Diagonal branches - D →anterior/lateral wall • LAD distal → anteroapical	• Left atrium • SA node (40% cases) • Left Marginal artery → the left posterior wall in the direction of apex • Posterolateral branches

☐ If the posterior descending artery is supplied by the right coronary artery (RCA), then the coronary circulation can be classified as **"right-dominant".** (In 70% of cases).
If the posterior descending artery is supplied by the circumflex artery (LCX), then the coronary circulation can be classified as **"left-dominant."** (In 20% of cases).
Rarely by both (10%)
(A precise anatomic definition of dominance would be the artery which gives off supply to the AV node)

3. ECG Lead System and Spatial Orientation

Electrocardiographic leads represent surface projections of specific myocardial regions, enabling localization of coronary insufficiency.

The 12-lead ECG reflects a spatial representation of cardiac electrical activity:

- Limb leads (I, II, III, aVR, aVL, aVF) view the frontal plane
- Precordial leads (V1–V6) view the horizontal plane

Mapping of coronary insufficiency , as per lead-wise / supply-wise understanding :

Leads	Coronary supply
Anterior wall : Leads V1-V4	LAD
Septal region : Leads V1-V3	Septal branches of Left anterior descending artery (LAD)
Lateral wall : Leads I , aVL , V5-V6	Left circumflex artery (LCX)
Inferior wall : Leads II , III , aVF	RCX (commonly) or LCX , depending upon other assoicates
Posterior wall : indirectly assessed via V1-V3	RCA (commoner) or LCX if it is dominant artery
Right ventricle : Lead V1,V2 with inferior leads in case of RV infarction ; V4R (for further confirmation)	RCA (for details see the posted ECG in favour of RCA involvement)

ECG Lead System : It would be easier if zonal orientation of coronary artery territories is identified on 12-lead ECG and for the purpose the corresponding leads are tick-marked accordingly to locate the culprit artery.

I	**aVR**	**V1**	**V4**
II	**aVL**	**V2**	**V5**
III	**aVF**	**V3**	**V6**

4. Further elaboration : Zonal orientation of Lead system

- Leads I , aVL + V5,V6 = Lateral zone → Left circumflex artery
- Leads II , III and aVF = Inferior Zone →
  Right coronary artery (RCA) or the left circumflex artery (LCX)
  ST elevation in III > II → RCA
  ST elevation in II > III → LCX
- Leads V1-3 = Anteroseptal zone
  V1-6 = Anterior zone
  V1-6 + aVL , (I) = Extensive anterior zone
  Extensive anterior involvement is most likely indicative of LAD coronary artery. If lead aVL (sometimes I) is involved in addition it indicates an anterior extensive infarct , the culprit lesion is likely to be proximal to the diagonal branch.
- Posterior zone is not directly recorded over 12 lead ECG. Lead V1-3 oriented to the anterior wall reflects the inverse – mirror image changes of the posterior wall → RCA (commoner) or LCX
- Inferior infarction plus right ventricular infarction (ST elevation in V1 with ST depression in V2 indicates RV infarction) → Proximal RCA
- Lateral zone + inferior + Posterior → LCX , as a dominant artery

What changes to be observed on ECG :

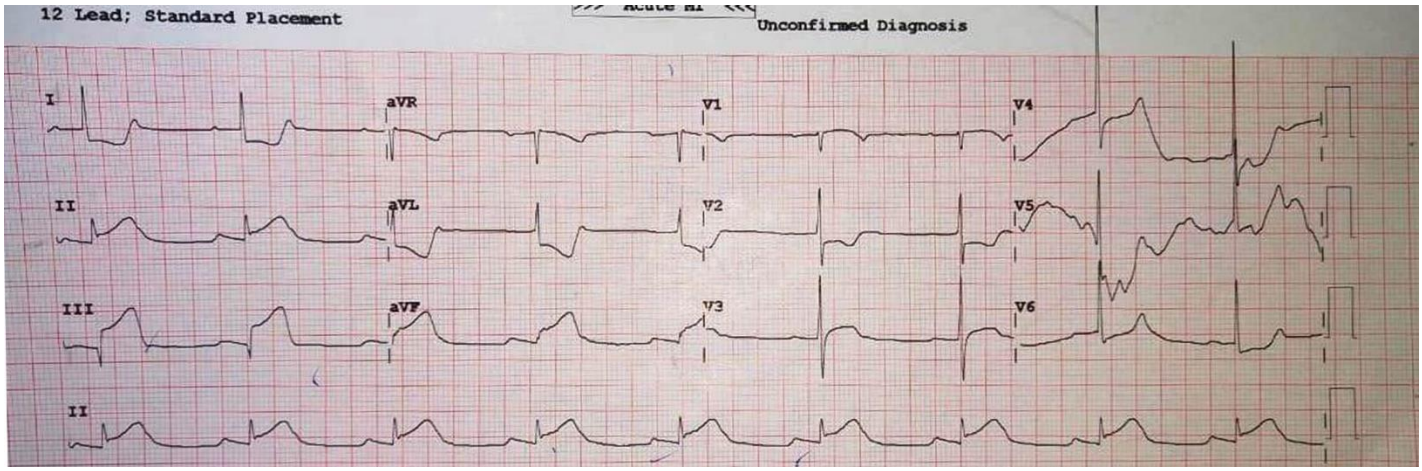
- ☐ ST-segment elevation (transmural injury , as indicative of STEMI)
- ☐ ST-segment depression
  - Diffuse ST depression with aVR elevation suggestive of LMCA (left main coronary artery) lesion , or severe multivessel disease.
  - Unstable angina
- ☐ T-wave inversion (repolarization abnormality)
- ☐ Look for 'Reciprocal changes' defined as ST-segment depression ≥ 1 mm in at least 2 leads in a single anatomic segment , occurring onto the contralateral side , as a mirror-image effect of ST-segment elevation associated with STEMI.
- ☐ Any other ECG finding in favour of coronary insufficiency

5. Limitations of Lead-wise Localization

Despite its utility, ECG localization has limitations :

- Anatomical variations in coronary supply and also coronary dominance (right-dominant or left-dominat)
- Collateral circulation
- Overlapping vascular territories
- Conduction abnormalities affecting repolarization

6. Illustration by ECG



Source : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Prinicipal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

(ECG belongs to Middle aged lady presenting with severe chest pain and features of shock).

Plotting of ECG findings on 12-lead system :

I ST↓ - Reciproc. change	**aVR**	**V1** ST↑	**V4**
II ST↑	**aVL** ST↓ - Reciproc. change	**V2** ST↓	**V5**
III ST↑ + Q	**aVF** ST↑	**V3**	**V6**

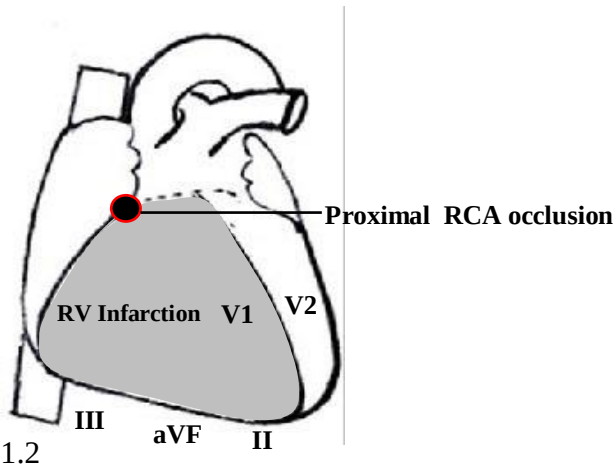


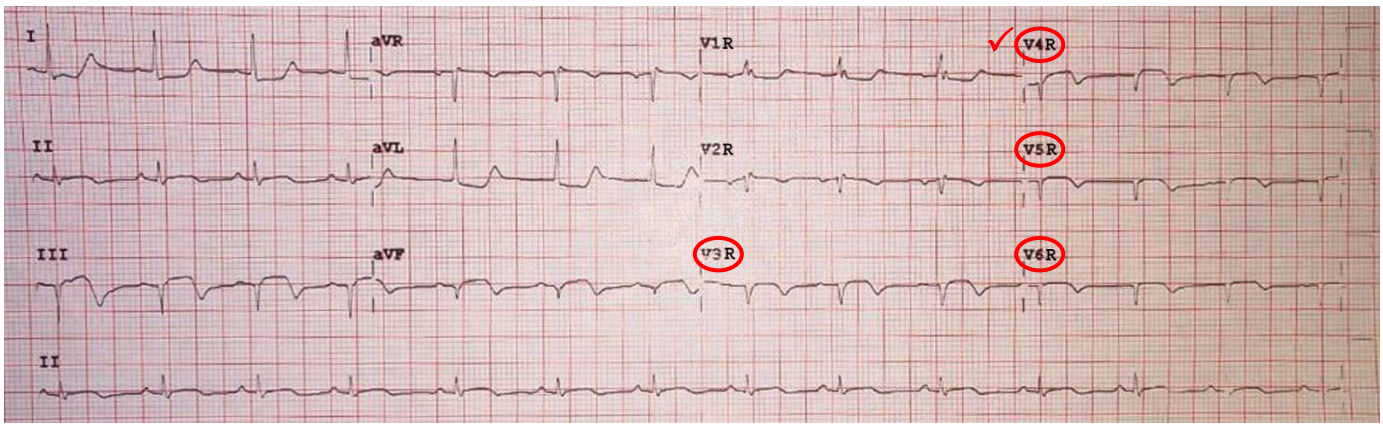
Fig. 1.2

- ST↑ in lead II , III and aVF , maximum in lead III
  (Q in lead III / reciprocal ST changes over leads I , aVL)
- ST↑ in V1 with ST↓ in V2 , being away from V1 , that's why , ST depression with this lead.

  (Extra finding in addition : sinus bradycardia with 1st degree AV block - PR interval 0.23 Sec ; This is to mention here that right coronary artery supplies also SA node).

Acute inferior MI + ST elevation in V1 with ST depression in V2 = **acute right ventricular infarction due to proximal RCA occlusion** , to be further confirmed by exploring through right sided chest leads (V1 is the only standard ECG lead that peeps directly through the right ventricle)

By placing the precordial leads over the right chest



Comments :
Diagnosis is confirmed by the presence of ST elevation over the right-sided chest lead V3R-V6R. The most useful information is ST elevation over V4R.

ST elevation in V4R has a sensitivity of 88% , specificity of 78% and diagnostic accuracy of 83% in the diagnosis of RVMI

7. Take-Home Message

This article provides an integrated overview of coronary circulation and its reflection on ECG through a lead-wise approach. By correlating myocardial territories with corresponding ECG leads, a systematic framework is presented to aid in accurate localization of coronary artery lesions.

8. References

All articles cited below are accessible at the indicated links.

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   Mike Cadogan , Jan 30, 2022
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   By Alex Yartsev - 29/06/2015
   Last updated 03/06/2024 - 23:42
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BY **The Editors of Encyclopaedia Britannica** View Edit History
https://www.britannica.com/science/coronary-circulation

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LAD OCCLUSION STEMI : THE MOST DEVASTATING CORONARY EVENT ON ECG

ECG

LAD OCCLUSION STEMI : THE MOST DEVASTATING CORONARY EVENT ON ECG

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OUTLINE

Introduction
Occlusion of the Left Anterior Descending Artery (LAD) yields the most extensive electrophysiological disruption because this arterial part supplies a vast and functionally critical myocardial territory.

The overall electrophysiological reflection of LAD occlusion

Blood supply by LAD to the ventricle

- Septal perforator branches
- Diagonal branches

NB : Wrap-Around LAD

Determining the sites of LAD occlusion
Septal perforators compromise and intensify septal injury ± conduction abnormalities , whereas diagonal branch involvement accentuates high lateral electrical forces.

Illustration by ECG
ECG 1 : Extensive anterolateral wall MI (LAD occlusion before both septal and diagonal)
ECG 2 : Proximal / Early LAD distal to the diagonal branch
ECG 3 : Proximal LAD occlusion with wrap artery around the apex of the left

Take-Home Message

References

LAD Occlusion STEMI : The most devastating coronary event on ECG

A Narrative Review

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When there is a plumbing emergency in a large house, one seeks a reliable, skilled, and efficient plumber whose team can quickly identify the site of the defect and provide prompt repair.

A similar principle applies to occlusion of the left anterior descending (LAD) coronary artery—the major supplier of blood to a large portion of the myocardium. Rapid recognition of LAD occlusion on the ECG is essential so that the clinician, like an expert plumber locating a critical defect, can intervene urgently to preserve myocardial function and save life.

- ☐ **Among all coronary artery occlusions, LAD obstruction produces the most extensive electrophysiological, mechanical, and prognostic consequences because of the large myocardial territory it supplies.**
- ☐ **Consequently, LAD occlusion is associated with the greatest infarct burden, severe left ventricular pump failure, malignant arrhythmias, and major electrophysiological disturbances.**

Truly, the LAD artery nourishes the very frontlines of the heart; therefore, its sudden occlusion produces dramatic ECG manifestations—the cry of a struggling myocardium. Like a skilled plumber identifying and correcting a critical defect, the attending clinician must promptly recognize the abnormality and initiate immediate reperfusion therapy to minimize myocardial damage with improved survival.

1. Introduction (Keypoints)

- Occlusion of the Left Anterior Descending Artery (LAD) yields the most extensive electrophysiological disruption because this arterial part supplies a vast and functionally critical myocardial territory. This includes :
  - Left Ventricle: the anterior, lateral, and apical walls.
  - Interventricular Septum: the anterior two-thirds of the septum.
  - A considerable portion of the ventricular conduction system: the right bundle branch and the anterior fascicle of the left bundle branch.
  - And the anterior papillary muscle of the mitral valve.
- The resultant acute infarct with a considerable involvement of anatomical location , as supplied by LAD may also cause conduction system involvement with propensity to arrhythmic risk. There is associated hemodynamic compromise with its overall surviving prognosis.

- Consequently, LAD occlusion is associated with the greatest infarct burden, severe left ventricular pump failure, malignant arrhythmias, and major electrophysiological disturbances.

2. The overall electrophysiological reflection of LAD occlusion

- Acute interruption of blood flow within this arterial territory rapidly initiates a cascade of biochemical events occurring in the myocardium.
  - **Ischemic ionic disturbances :** Depletion of intracellular ATP impairs membrane ionic pump (Na-K ATPase pump), causing extracellular accumulation of potassium ions and intracellular accumulation of sodium ions. This surplus of intracellular sodium forces the Na-Ca exchanger mechanism to operate in reverse, resulting in intracellular calcium overload.

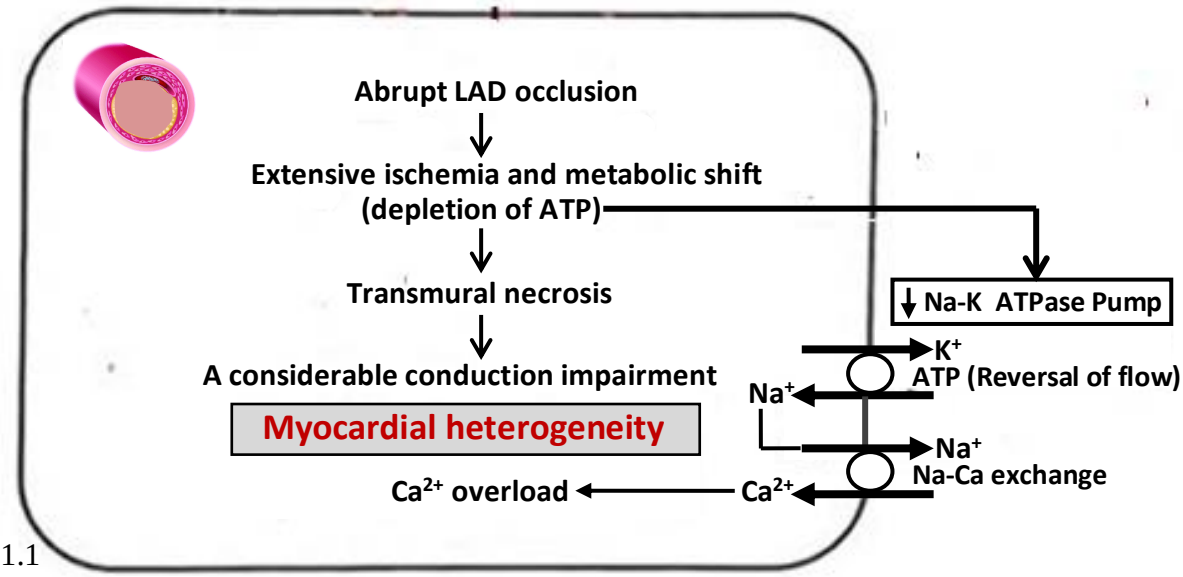


Fig 1.1

  - **Myocardial heterogeneity :** Ionic disturbances with accompanying extensive transmural necrosis cause electrical structure remodelling. This creates profound myocardial heterogeneity , resulting in abnormal action potential propagation and dispersion of repolarization.

- LAD occlusion shows an extensive "wave-front" of myocardial injury that spreads from subendocardial myocardium to subepicardial myocardium , causing a pathophysiological cycle to set in with the following changes :
  - Shift to anaerobic metabolism → ATP depletion → Failure of ionic pump → Extracellular K+ accumulation → hyperacute T-wave
  - Evolving transmural necrosis→ ST-segment elevation
  - Completion of transmural necrosis → pathological Q wave

Because of the large myocardial area involved, reciprocal ST depression may simultaneously appear in electrically opposite leads.

- To be noted, LAD occlusion also carries a critical arrhythmogenic potential. Ischemic heterogeneity, conduction slowing, dispersion of refractoriness, Purkinje system involvement, and calcium-mediated triggered activity collectively create an unstable electrophysiological substrate capable of generating malignant ventricular arrhythmias including ventricular tachycardia and ventricular fibrillation.
- Proximal LAD occlusion may additionally disrupt septal conduction pathways, producing:
  - Right bundle branch block,
  - Left anterior fascicular block,
  - Or varying degrees of atrioventricular conduction disturbance.
- It is worthwhile to mention here that mitral regurgitation (MR) with Left Anterior Descending (LAD) occlusion may also create a critical cardiac complication known as ischemic mitral regurgitation. This condition often presents as a medical emergency , associated with pulmonary oedema of variable degree.
- Therefore , the dynamic ECG evolution in LAD occlusion reflects not merely localized myocardial injury, but widespread disturbance of ventricular electrical organization. The extensive electrophysiological consequences directly parallel the large anatomical territory supplied by the LAD artery and explain the severe hemodynamic and prognostic impact associated with anterior STEMI.

3. Blood supply by LAD to the ventricle

This seems essential first to discuss the blood supply by LAD to the concerned ventricular myocardium. This is subserved by its two main branches : septal perforators and diagonal branches :

- **Septal perforator branches** :
  - The proximal septal perforator branches of the LAD, particularly the first few septal branches (mainly the proximal 2–4 septal perforators), supply most of the anterior two-thirds of the interventricular septum, including important components of the conduction system such as the right bundle branch and the left anterior fascicle.
  - The more distal septal perforator branches course deeper along the interventricular septum toward the apical septal region and contribute to the perfusion of the adjacent septal myocardium of the left ventricle.

The septal perforator branches of the LAD are represented on ECG predominantly by the anterior–septal leads V1–V4, but depending on the extent and level of LAD involvement, the electrical manifestation may extend across V1–V6 and occasionally to the high lateral leads I and aVL.

- **Diagonal branches** of the left anterior descending (LAD) artery primarily supply the anterolateral wall of the left ventricle, which is represented on the ECG mainly by leads I, aVL, V5, and V6. The obtuse marginal branches of the left circumflex (LCx) artery also contribute to the perfusion of this lateral

myocardial territory, resulting in a variable overlap of blood supply between the diagonal branches of the LAD and the marginal branches of the LCx.

NB : Wrap-Around LAD (Left Anterior Descending) artery may have an anatomical variation where the LAD extends past the apex of the heart to supply a significant portion of the inferior wall. This differs from standard anatomy , where the artery terminates at the apex without wrapping around.

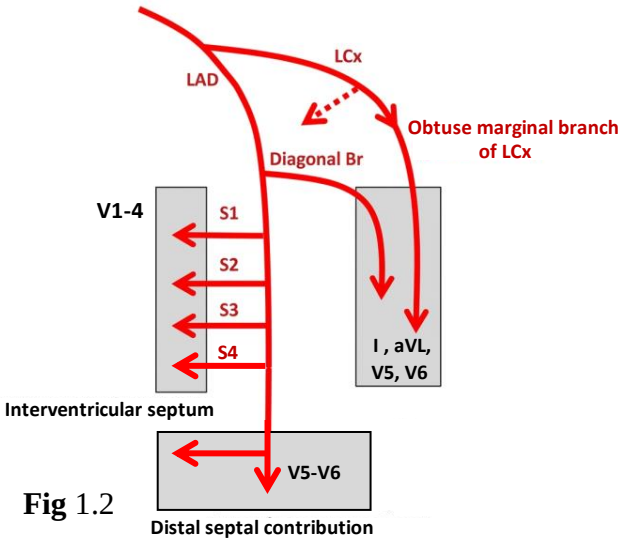


Fig 1.2

- Diagonal branches from LAD exist in variable number , often 2 to 9. They run somewhat obliquely along its course but here in this sketch the diagonal branch is shown to be arising as a single oblique branch (for the purpose of simplicity).
- The left circumflex (LCx) artery typically has 1 to 3 obtuse marginal (OM) branches. However, human coronary anatomy varies widely, meaning some individuals may have no prominent OM branches, while others can have up to 4 or more.
  Here obtuse marginal branches are also represented by a single one – for the purpose of simplicity.
- The distal lateral territory viewed by leads V5-V6 is supplied by both the terminal septal branches and the diagonal branches of LAD.
- Amongst all the septal branches the first one (S1) is having comparatively wider lumen , which gradually narrows while descending downward.

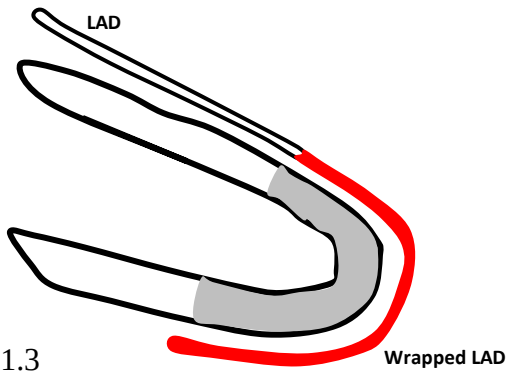


Fig 1.3

- The incidence of wrapped LAD is typically reported in between 26% to 35%. In this variant , the LAD wraps around the significant portion of the apex.

4. Determining the sites of LAD occlusion

It would be wiser and practical to determine the sites of LAD occlusion by noting changes over the concerned leads including I, aVL and V5-V6. The selection and extent of involvement of these leads would dictate towards the precise site of LAD occlusion.

☐ **Septal perforator branches** primarily supply the interventricular septum, the resulting electrical effects of septal ischemia may extend across the entire anterior precordial lead system depending upon the extent of myocardial involvement.

- Occlusion Proximal to Septal and Diagonal Branches ("Very proximal LAD occlusion") :
  This includes septum , anterior wall , apex and diagonal territory. And ECG Features include marked ST elevation in leads V1-V6 , I and aVL with reciprocal changes in inferior leads II, III and aVF.
  ST elevation in I and aVL during this extensive LAD occlusion, the principal mechanism endures either of :
  superior-leftward injury vector orientation, overall directed towards leads I and aVL
  OR
  anterolateral extension to high lateral leads.
  Conduction disturbances are also possible to occur involving Right bundle branch , left anterior fascicle and portion of HIS-Purkinje system.
- Proximal / Early LAD : When LAD occlusion occurs distal to the diagonal branch origin (around proximal septal supply) , preservation of the anterolateral myocardium by the simultaneous running of diagonal branches of LAD may limit leftward injury-vector extension, thereby restricting ST elevation predominantly to V1–V4.
- Mid LAD Occlusion (just below S1) : predominantly affects V2-V4 , ± V5 territory.
- Distal LAD occlusion (just below S2) : It is reflected through leads V3-V6 signifying apical and anterior distal territory.
- With wrap-LAD: additional involvement of apical-inferior surface with inferiorly directed vector component.

Sites	Leads wise involvement	Anatomical territory
Very Proximal LAD	V1–V6, I, aVL	Septal + high lateral involvement
Proximal/Early LAD (around proximal septal supply)	V1-V4	Proximal anteroseptal
Mid LAD	V2–V4	Predominantly anterior
Distal LAD	V3–V6	Apical/anterior distal

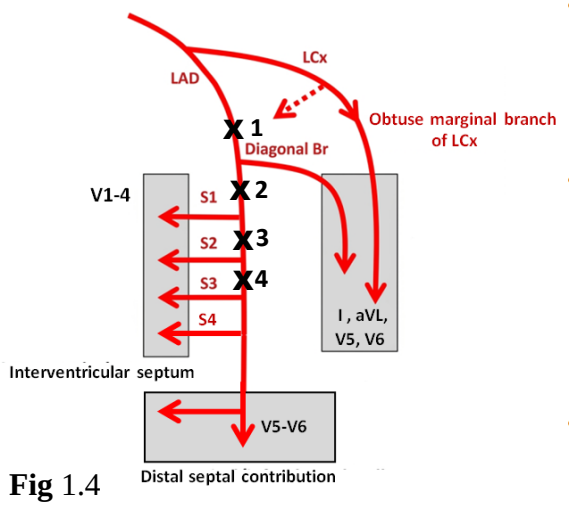


Fig 1.4

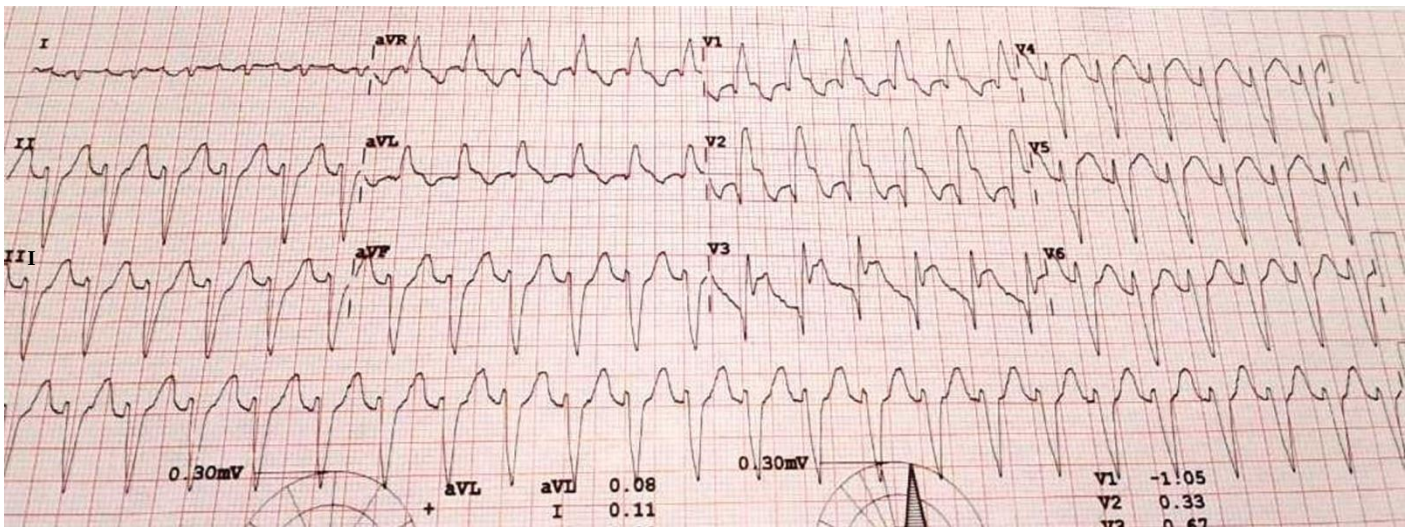
1 = Very proximal LAD occlusion (Proximal to septal and diagonal branches)
2 = Distal to the diagonal branch origin , mainly limited to V1-V4
3/4 = Mid-LAD / Distal-LAD occlusion depending upon the site of involvement

Broadly to say :

- ✓ Septal perforators compromise and intensify septal injury ± conduction abnormalities , whereas diagonal branch involvement accentuates high lateral electrical forces.
- ✓ V1 involvement suggests septal jeopardy, while aVL involvement suggests superior-leftward extension of the injury vector. This is to be noted that V1 faces the basal septum most directly , thus , marked V1 involvement often suggests proximal septal branch compromise. Exclusion of leads I and aVL involvement signifies the occlusion being just after such diagonal branch (proximal/early LAD occlusion).
- ✓ Mid and distal LAD localization on ECG is governed not only by the site of obstruction but also by the extent to which diagonal branch perfusion continues to preserve adjacent myocardium.

5. Illustration by ECGs

ECG 1 : Extensive anterolateral wall MI (LAD occlusion before both septal and diagonal)



Source : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Prinicipal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

Findings :

- RBBB pattern with QR over V1-3 with coved ST elevation over these leads.
- Also coved ST elevation over V4-5
- Atrial flutter with 2:1 AV conduction (as per Bix rule) , in association with bifasicular block (C RBBB + high LAFB)

Comments :

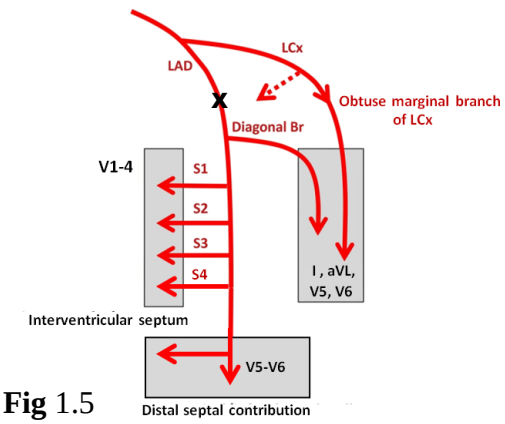
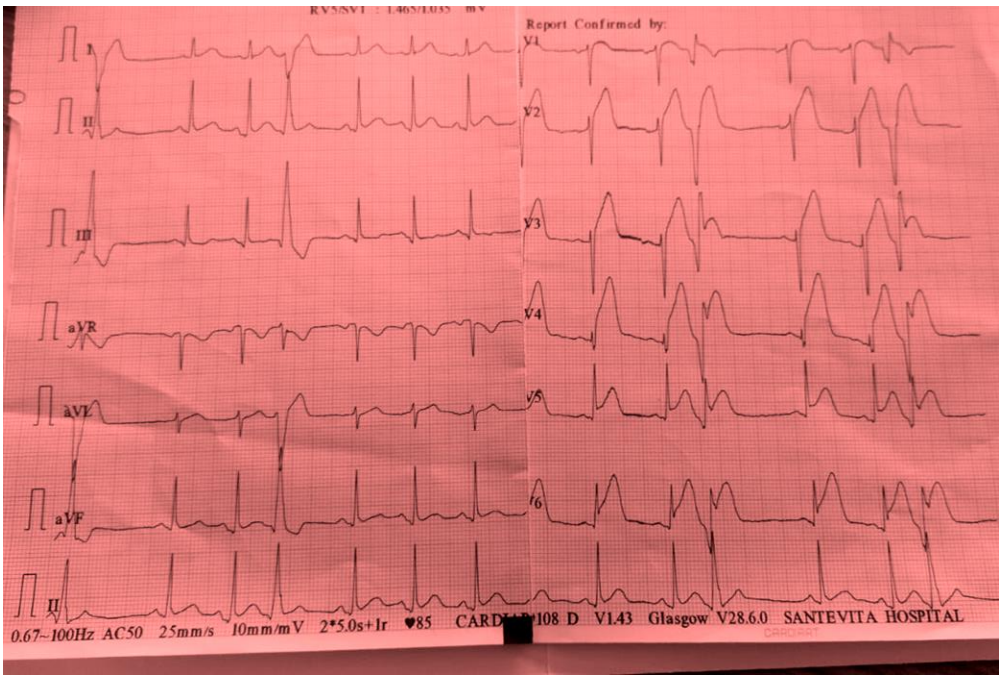


Fig 1.5

- Involvement of leads V1-V6 , I and aVL point towards very proximal LAD occlusion
- Additional conduction disturbances involving right bundle branch and left anterior fascicle (bifascicular block) also point towards the same site of LAD occlusion.

ECG 2 : Proximal / Early LAD distal to the diagonal branch



Source : : Dr. Varun kumar , DM Card. , Orchid , Ranchi (Jharkhand Doctor's Forum) on 17.05.2026

(This patient presented with typical chest pain of a few hour duration and is subsequently underwent successful Primary PTCA of LAD)

Findings :

- Coved ST segment elevation in leads V1-V6 , these are more obvious upon the simultaneous running extrasystoles (not so broad pointing towards paraseptal PVC) – please see leads V3-V4 for the purpose. This is ECG dictum that ventricular extrasystoles makes the pre-existing ST elevation more prominent , as in this case.
- Poor R-wave progression in association

NB : If the PVC has a negative QRS , say in precordial leads , its native , built-in tendency to elevate the ST segment adding directly to the existing ischemic STEMI elevation , making it appear drastically exaggerated and 'more obvious' – as per "electrophysiological rule of discordance".

Comments :

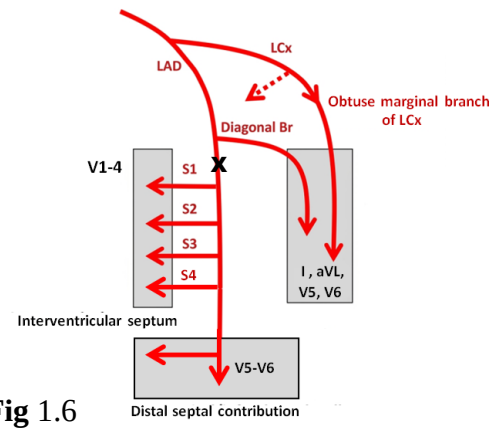
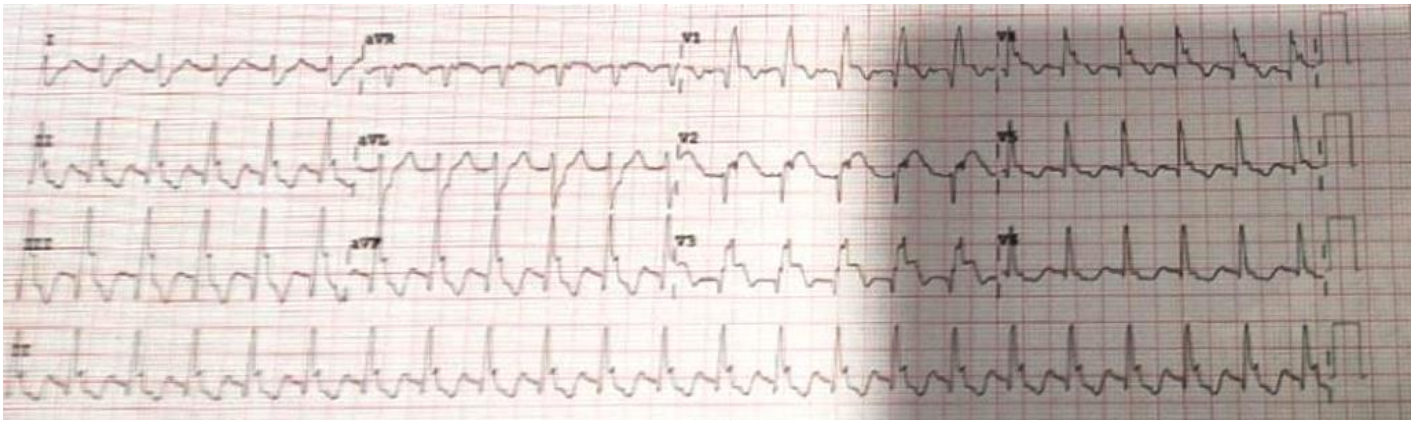


Fig 1.6

- As there is no ST elevation in leads I and aVL , the LAD occlusion is considered just after the diagonal branches.

ECG 3 : Proximal LAD occlusion with wrap artery around the apex of the left ventricle



Source : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Prinicipal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

(Middle aged man with severe chest pain since 2 hours)

Findings :..

- ECG shows anterior STEMI with RBBB pattern and associated with concomitant inferior STEMI
  (On frontal plane the vector is directed towards inferior leads)
- Atrial flutter with 2:1 AV conduction as per Bix Rule

Comments :

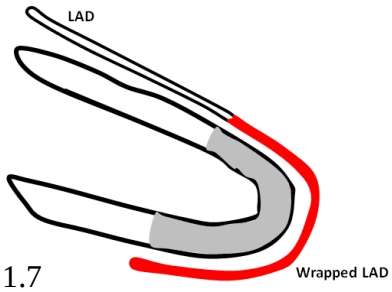


Fig 1.7

- With wrap-LAD: additional involvement of apical-inferior surface with inferiorly directed vector component.

6. Take-Home Message

- ☐ Among all coronary artery occlusions, LAD obstruction produces the most extensive electrophysiological, mechanical, and prognostic consequences because of the large myocardial territory it supplies.
- ☐ Consequently, LAD occlusion is associated with the greatest infarct burden, severe left ventricular pump failure, malignant arrhythmias, and major electrophysiological disturbances.
- ☐ LAD occlusion also carries a critical arrhythmogenic potential. Ischemic heterogeneity, conduction slowing, dispersion of refractoriness, Purkinje system involvement, and calcium-mediated triggered activity collectively create an unstable electrophysiological substrate capable of generating malignant ventricular arrhythmias including ventricular tachycardia and ventricular fibrillation.

☐ Sites of LAD occlusion

- Occlusion Proximal to Septal and Diagonal Branches : Involvement extending to V1-V6 , I and aVL .
- When LAD occlusion occurs distal to the diagonal branch origin, preservation of the anterolateral myocardium limits leftward injury-vector extension, thereby restricting ST elevation predominantly to V1–V4.
- Mid LAD Occlusion : predominantly affects V2-V4 , ± V5 territory.
- Distal LAD involvement is reflected through leads V3-V6 signifying apical and anterior distal territory.
- With wrap-LAD: additional involvement of apical-inferior surface with inferiorly directed vector component.

NB : Mid and distal LAD localization on ECG is governed not only by the site of obstruction but also by the extent to which diagonal branch perfusion continues to preserve adjacent myocardium.

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PERSISTENT HYPERACUTE T-WAVE PATTERN ON ECG : A SIGN OF ACUTE TRANSMURAL MYOCARDIAL ISCHEMIA

ECG

PERSISTENT HYPERACUTE T-WAVE PATTERN ON ECG : A SIGN OF ACUTE TRANSMURAL MYOCARDIAL ISCHEMIA

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OUTLINE

Persistent Hyperacute T-wave Pattern on ECG : A Sign of Acute Transmural Myocardial Ischmeia

A Narrative Review

© **DR. D.P. KHAITAN**
MD (MEDICINE) FCGP (IND) FIAMS (MEDICINE) FICP FICCMD FIACM

A medical professional's mind is struck with astonishment when it encounters an unusual yet genuine clinical problem. How to arrive at the correct answer to such a puzzle continues to haunt the mind until a satisfactory explanation is met with.

The same holds true when a clinician finds a persistent pattern of hyperacute T waves on the ECG without the obvious ongoing evidence of acute myocardial infarction. He focuses his mind on the issue by reviewing the available literatures so that a 'Eureka' moment may arise by resolving the problem into a clear, elegant truth with the ecstatic, breathless certainty of Archimedes.

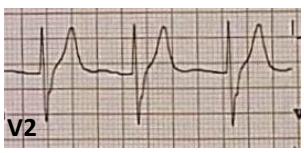
- ☐ **In patients with coronary artery occlusion, hyperacute T waves are usually transient and rapidly evolve into ST-segment elevation.**
- ☐ **A look of utmost surprise appears on the face of a clinician when he documents persistent hyperacute T waves lasting several hours in the setting of acute coronary occlusion.**

In answer to this puzzle, the clinician may propose relative preservation of epicardial perfusion, either by collateral blood flow or by a subtotal coronary occlusion, preventing the development of classic ST-segment elevation while allowing the hyperacute T-wave pattern to persist beyond its usual duration.

1. Introduction (Keypoints)

- A hyperacute T wave is the earliest ECG sign of acute coronary occlusion, appearing as a tall, broad, and symmetric T wave before classical ST-segment elevation develops. This is the signature of transmural myocardial insult which tends to be a short-lived morphological change that evolves rapidly into ST segment elevation. In this way hyperactute T-wave (HATW) signifies the ECG event of ongoing transmural acute myocardial infarction.

- Dr. Stephen Smith and colleagues have emphasized that the proportionality of the T wave to the QRS complex is often more important than the absolute T-wave height.
  The principle is a T wave should be judged in relation to the size of the preceding QRS complex.
  As per Dr. Smith T-wave amplitude / QRS amplitude > 0.36 in at least one anterior precordial lead (especially V2–V4) strongly suggests a hyperacute T wave and possible acute LAD occlusion.



Broadly to say , here T-waves are tall , broad-based , symmetrical with this disproportionately large relative to the QRS complex. **(T-wave amplitude / QRS amplitude > 0.36)**

Fig 1.1

The diagnosis of hyperacute T waves depends more on its proportionality to the QRS complex than on its absolute height. A T/QRS ratio exceeding approximately 0.36 in an anterior precordial lead is a useful clue to acute coronary occlusion.

✓ This diagnosis is strongly suspected in the presence of the clinical history suggestive of acute coronary artery disease.

- Sometime in practice hyperacute T-wave (HATW) may persist for surprisingly longer period without being followed by ST segment elevation. Here this sort of hyperacute T-wave indicates acute myocardial ischemia rather than myocardial infarction. The duration of hyperacute T waves depends mainly on the dynamic balance between ischemia, injury, and reperfusion.
- The larger is the ischemic territory , the greater is the electrical gradient with more prominent and sometimes persistent hyperacute T-waves. This is often seen with proximal LAD occlusion.
- In nutshell to say, Hyperacute T waves persist as long as a significant ischemic electrical gradient exists; their duration is determined by the interplay of coronary occlusion, collateral flow, and the quantum of reperfusion.
- In acute coronary occlusion, time is myocardium; the physician who recognizes the hyperacute T wave hears the warning that it may be soon followed by ST segment elevation if there is ongoing myocardial infarction.

2. The Basic Electrophysiology

Acute subtotal occlusion within the involved arterial territory (say proximal LAD) rapidly initiates a cascade of biochemical events occurring on the concerned myocardium.

❒ **Ischemic ionic disturbances :** Depletion of intracellular ATP impairs membrane ionic pump (Na-K ATPase pump), causing extracellular accumulation of potassium ions and intracellular accumulation of sodium ions. This surplus of intracellular sodium forces the Na-Ca exchanger mechanism to operate in reverse, resulting in intracellular calcium overload.

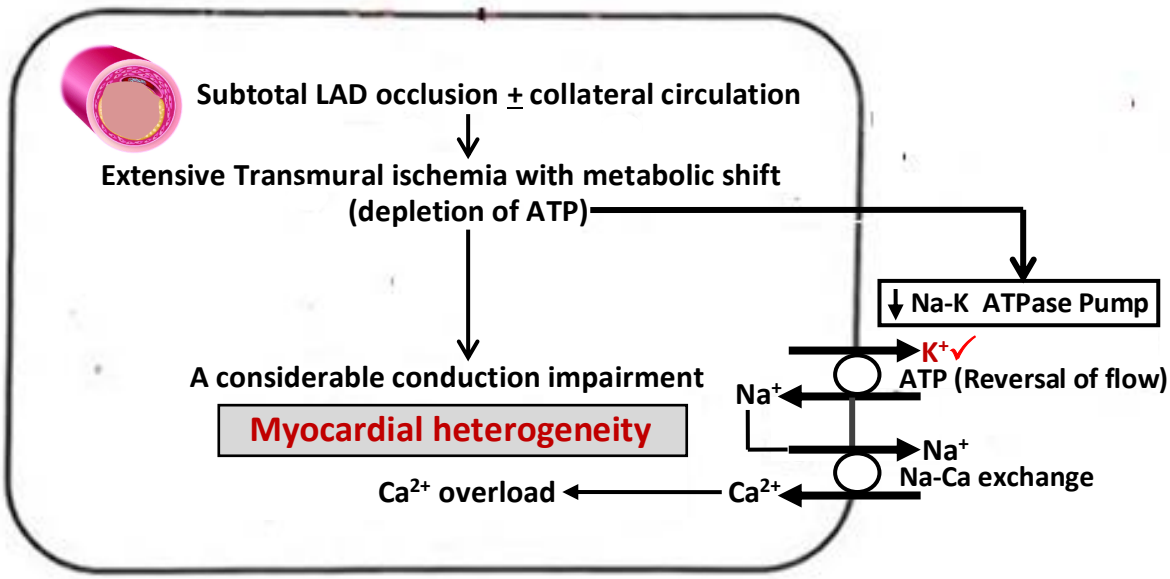


Fig 1.2

Here, ischemic myocardium remains electrically active with local extracellular potassium accumulation which may result in persistant hyperacute T-waves.

Metabolic shift to anaerobic cycle → ATP depletion → Failure of ionic pump → Extracellular K+ accumulation → hyperacute T-wave

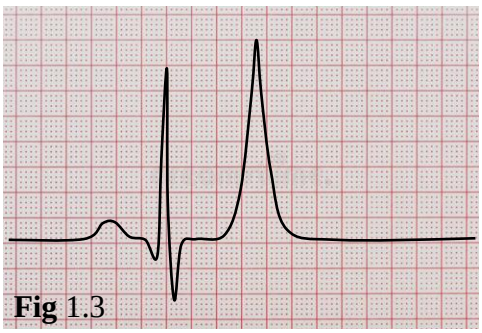
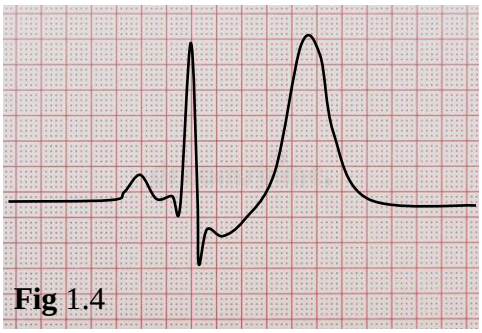
The ischemic process is continuing, and the T wave remains "trapped" in its hyperacute phase.

- **Myocardial heterogeneity :** Ionic disturbances with accompanying extensive transmural ischemia create a profound myocardial heterogeneity , resulting in abnormal action potential propagation with dispersion of repolarization. This carries a critical arrhythmogenic potential → PVCs → Ventricular arrhythmias.

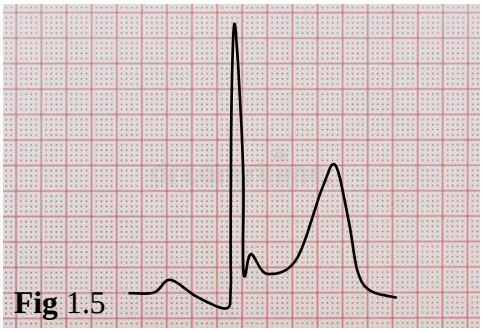
Here exists a profound possibility of relative preservation of epicardial perfusion, either by collateral blood flow or by a subtotal coronary occlusion, preventing the development of classic ST-segment elevation while allowing the hyperacute T-wave pattern to persist beyond its usual duration.

3. Differential Diagnosis

There are a few important 'exceptions and cautions' in context with tall T-waves (T/QRS ratio > 0.36 concept).

ECG entity	Characteristics
➢ **Hyperkalemia**  **Fig** 1.3	T waves are usually narrow-based, sharp, and tented, unlike the broad-based hyperacute T wave.
➢ **De Winter T-wave (STEMI Equivalent LAD Occlusion)**  **Fig** 1.4	Upsloping ST depression preceding tall T-wave in precordial leads. Often mild STE in aVR.

Contd.

➢ **Early repolarization**  **Fig** 1.5	J-point elevation revealed either as terminal QRS slurring or notching associated with upward concave ST segment elevation and prominent T-waves in at least two contiguous leads.

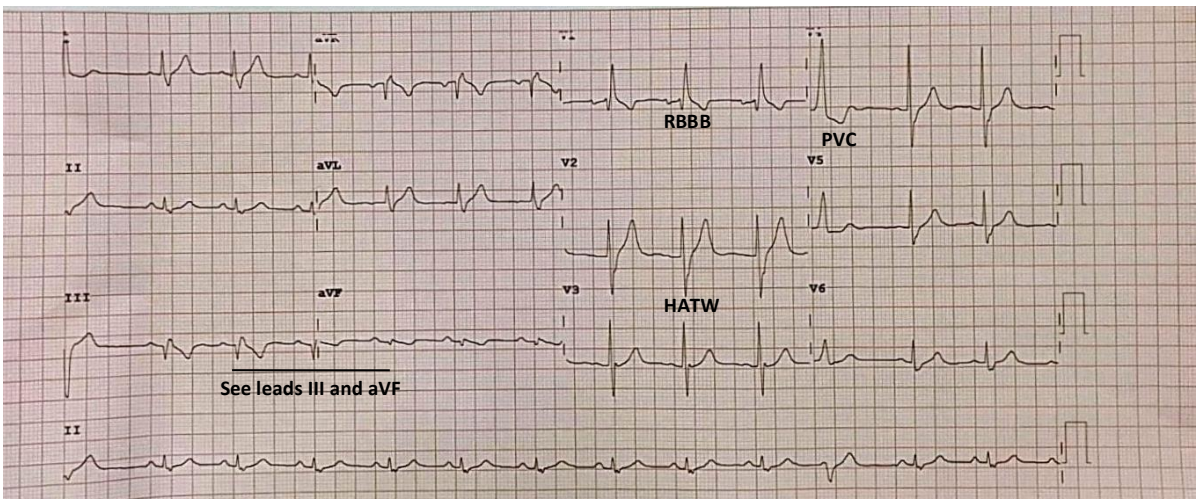
4. Illustration by ECGs

RBBB with anterior hyperacute T-wave (HATW) associated with 'Dual Proximal LAD and RCA occlusion' on ECG

History : 75 years male, Diabetic, history of retrosternal heaviness sometimes radiating to lower jaw while doing morning walks for a week, sometimes even at rest for previous couple of days reported to OPD. His Pulse was 80pm, regular with Intermittent extrasystoles, JVP - N, BP 130/80, CVS and Chest examination – Normal.
Troponin I , serum lipids , and kidney function test : All Normal
Cardiac Echo screen looked perfect with no RWMA or apparent LV / RV dysfunction.
Keeping him as a case of ACS - Unstable Angina he was admitted and put on DAPT/ Statin / Betablocker/ Nitrate and LMWH by Dr. Satish Kumar.

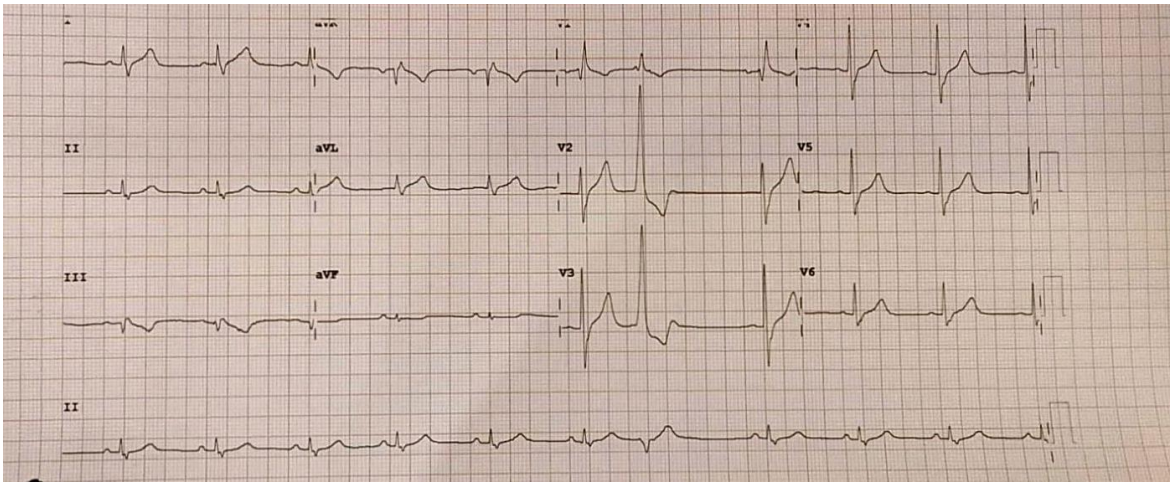
By the next morning , he had much relief in his symptoms.

This is the first ECG taken at the time of admission when angina symptoms were prominent :



Source : CME INDIA on 21.06.2026 by **Dr. Satish kumar**, Senior Consultant Physician and Cardiologist , Wellmark Hospital , Bokaro **(with his kind permission).** Additional labeling done to highlight the changes on ECG.

The next ECG was taken on the next morning , when the patient was relatively symptom free



The next morning ECG of the same patient :

☐ His CAG shows the following findings :

- Left Anterior Descending Artery (LAD) – Ostial to proximal 80-90% stenosis, mid to distal normal (Diagonal : Small vessel)
- Left circumflex (LCX) :- Ostial to proximal 40-50% , LCX distal after omm 70-80% diagnosis (OMM : Normal)
- Right coronarhy artery (RCA) – Proximal total occlusion.

Final Diagnosis : Triple vessel disease

In my opinion, this case represents an ECG illustration par excellence put by Dr. Satish Kumar.

Key Findings :

First ECG at the time of admission shows :

- RBBB pattern restricted to lead V1
- Hyperacute T-wave prominent in lead V2
- Occasional PVCs (its morphology suggestive of left ventricular origin)
- Looks to be ? pathological Q with inverted T in leads III and aVF (changes are more prominent over lead III)

Second ECG (when the patient was relatively symptom free) :

- RBBB pattern persisting as such
- Hyperacute T-wave is still present in V2
- PVCs seen with more clarity (its morphology suggestive of left ventricular origin).
- rS pattern with T inversion (clearly seen on zooming) , being more prominent in lead III compared to aVF.

Corresponding coronary angiogram (CAG) showing 80-90% proximal LAD sub-occlusion and 100% proximal RCA occlusion.

Discussion :

- ☐ The LAD-RBBB Connection: The proximal right bundle branch receives its primary blood supply from the septal perforators of the proximal LAD. A sudden, critical 80-90% narrowing can cause conduction system ischemia, creating RBBB pattern, restricted to lead V1. Here to say that the first perforator branch is having the wider bore compared to the rest perfortors, that's why , lead V1 is only showing the conduction abnormality (RBBB) rather than anterior ischemic changes.

  KEY POINTS

  - The RBB courses through the upper part of the interventricular septum, immediately below the membranous septum.
  - It is a subendocardial structure.
  - Because of end-arterial (terminal) supply, it is vulnerable to ischemia.
  - Occlusion of septal perforators (from LAD) can cause RBBB.

- ☐ In normal RBBB, leads V1–V3 should display discordant ST-depression and T-wave inversion. The presence of upright T-waves in V2–V3 is a major red flag—it means the ischemic vector is completely overpowering the expected repolarization forces of right bundle branch block.

- ☐ Hyperacute T-wave is predominantly seen in lead V2. As per Dr. Smith T-wave amplitude / QRS amplitude > 0.36 in at least one anterior precordial lead (especially V2–V4) strongly suggests a hyperacute T wave and possible acute LAD occlusion.

- ☐ The left-ventricular PVCs as suggested by its morphology are a direct warning sign of a highly irritable, hypoxic myocardium resulting from the critical narrowing of the proximal LAD. As the patient's symptom improve by the following morning , the occurrence of these PVCs may also represent reperfusion ventricular extrasystoles , suggestive spontaneous reperfusion of the ischemic myocardium.

- ☐ Inferior leads with T-inversions , being more marked over lead III point toward RCA involvement as well. Here to be noted that pseudo pathological Q as seen on the first ECG disappeared from leads III and aVF both and is replaced by rS pattern and T inversion on the next ECG (these changes are more prominent over lead III). All these findings might indicate ? old RCA infarction or its present conversion from subtotal to complete occlusion.

Final impression :

The ECG findings discussed so far are predominantly explained by the combined effects of critical proximal LAD stenosis and proximal RCA occlusion. Although coronary angiography also demonstrated moderate ostial-to-proximal LCX stenosis and a significant distal LCX lesion (70–80%), no definite ECG changes attributable to the LCX are apparent. This likely reflects the distal location of the severe LCX lesion with preserved flow through the proximal LCX and obtuse marginal branch, resulting in insufficient ischemic burden to produce characteristic ECG abnormalities.

5. Take-Home Message

- The diagnosis of hyperacute T waves depends more on its proportionality to the QRS complex than on its absolute height. A T/QRS ratio exceeding approximately 0.36 in an anterior precordial lead is a useful clue to acute coronary occlusion.
  (This diagnosis is strongly suspected in the presence of the clinical history suggestive of acute coronary artery disease).
- Sometime in practice hyperacute T-wave (HATW) may persist for surprisingly longer period without being followed by ST segment elevation. Here this sort of hyperacute T-wave indicates acute myocardial ischemia rather than myocardial infarction. The duration of hyperacute T waves depends mainly on the dynamic balance between ischemia, injury, and reperfusion.
- The ischemic myocardium remains electrically active, and local extracellular potassium accumulation may contribute to persistent hyperacute T-wave morphology. In the absence of ongoing myocardial infarction, these changes may persist without evolving into overt ST-segment elevation.
- Extensive transmural ischemia resulting from a subtotal proximal LAD occlusion may also involve the interventricular septum and the right bundle branch, leading to the development of right bundle branch block (RBBB).
- The resultant electrical heterogeneity of the extensively ischemic myocardium creates a highly arrhythmogenic substrate, predisposing to ventricular ectopy and other ventricular arrhythmias. Furthermore, partial restoration of coronary blood flow, whether spontaneous or therapeutic, may precipitate reperfusion arrhythmias, including frequent ventricular premature complexes (PVCs).

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RECIPROCAL ST DEPRESSION ON ECG : REVEALING THE STORY OF UNDERNEATH STEMI

ECG

RECIPROCAL ST DEPRESSION ON ECG : REVEALING THE STORY OF UNDERNEATH STEMI

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OUTLINE

Introduction
In a patient with acute ST segment elevation with no otherwise cause for this, the presence of simultaneous reciprocal ST segment depression has a specificity and positive predictive value of approx. 93% for the diagnosis of acute myocardial infarction.

Reciprocal changes supports STEMI diagnosis

- ☐ Subtle ST elevation with dominant reciprocal ST depression
- ☐ Indicating high risk patient
- ☐ Reciprocal ST segment depression as the only predictive sign

Mechanism

- ➢ A mirror like reflection of STEMI (most likely accepted mechanism)
- ➢ Concomitant subendocardial ischemia at a contralateral distance (with less plausible evidence)

Lead-wise Patterns of Reciprocal Changes

- Leads facing the injured myocardium record ST elevation
- Leads facing away record ST depression (reciprocal change)

Limitations of such reciprocal ST changes

Illustration by ECG

Take-Home Message

References

Reciprocal ST depression on ECG : Revealing the story of underneath STEMI

A Narrative Review

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I look into the mirror, and the mirror looks back at me—one is the true image, the other a reflected projection.

O Heart! Thou, like a mirror, reflect the imprint of thy injury upon the contralateral side—both seem to weep together: one with true tears, the other only echoing their reflection.

Reciprocal changes are not incidental; they are the mirror through which the full geometry of myocardial injury is revealed.

- ☐ **ST segment elevation in one myocardial territory is often accompanied by reciprocal ST-segment depression in electrically opposite (contralateral) leads, typically observed in at least two anatomically contiguous leads.**
- ☐ **Reciprocal changes are an important electrocardiographic finding that enhance the diagnostic specificity of ST-elevation myocardial infarction (STEMI), with a high positive predictive value (approximately 93% in appropriate clinical settings)**

This concept has broadened the clinical perspective in the evaluation of STEMI. Recognition of reciprocal changes allows more precise localization of the infarct-related artery and provides insight into the extent and severity of myocardial injury.

1. Introduction (Keypoints)

- Reciprocal ST-segment changes are a fundamental yet often underappreciated component of electrocardiographic interpretation in ST-elevation myocardial infarction (STEMI). Traditionally viewed as secondary phenomena, these changes are now recognized as integral reflections of the vectorial nature of myocardial injury.
- Acute injury pattern is an electrocardiographic finding that commonly reflected as an abnormal ST segment elevation. The most common cause of such injury current is acute myocardial infarction. This very important hallmark of ST segment elevation in acute MI may be accompanied with reciprocal ST segment depression onto the contralateral side , which supports STEMI diagnosis as an extra criterion.
- Reciprocal change is defined as **ST-segment depression $\geq$ 1 mm in at least two anatomically contiguous leads**, **occurring onto the contralateral side , as a mirror-image reflection of ST-segment elevation associated with STEMI.**
- The recognition of such reciprocal ECG change in combination with the classical ST segment elevation can supplement the diagnosis of coronary artery occlusion. This would be worthwhile to mention here that **the concept of reciprocal change cannot be entertained in a patient who is exhibiting the following patterns on ECG** :

left bundle-branch block, right bundle-branch block, left ventricular hypertrophy with strain or right ventricular paced rhythm from an implanted pacemaker, etc. – resulting in ST depression due to secondary repolarization abnormalities.

- In a patient with acute ST segment elevation with no otherwise cause for this , the presence of simultaneous reciprocal ST segment depression has a specificity and positive predictive value of 93% for the diagnosis of acute myocardial infarction.
- This article explores the electrophysiological basis, diagnostic significance, and clinical implications of reciprocal changes, emphasizing their role in confirming infarction, enhancing localization, and assessing the severity of coronary artery involvement. Understanding these mirrored electrical patterns allows clinicians to uncover the full extent of myocardial injury beyond the primary ST elevation.

2. Reciprocal change supports STEMI diagnosis

☐ Sometimes subtle ST elevation might be the only initial ECG finding in acute occlusion MI. Under such circumstances reciprocal ST depression may remain as a dominant sign pointing to subtle ST elevation.

‘ As two recent studies found, minor ST elevation and reciprocal ST depression or T-wave inversion were the most helpful signs in identifying occlusion MI that do not meet STEMI criteria, or STEMI(-)OMI. ’
Ref : ECG Cases 24 Reciprocal Change and Occlusion MI
Written by Jesse McLaren; Peer Reviewed and edited by Anton Helman. August 2021
https://emergencymedicinecases.com/ecg-cases-reciprocal-change-occlusion-mi/

☐ Indicating high risk patient : reciprocal ST segment depression occurs more frequently in patients having a larger MI or a more proximal coronary occlusion. It reflects greater injury vector magnitude. Thus, reciprocal change in the setting of STEMI may identify the patient having an increased possibility of cardiovascular complications such as heart block , malignant ventricular arrhythmias , cardiogenic shock – with poor prognostic outcome , even including death.

☐ Reciprocal ST segment depression may the only predictive sign on ECG as a diagnostic pointer to STEMI.

The 12-lead surface ECG does not represent the entire electrocardiographic field , there are certain grey areas which are not covered by any lead placed over the body surface – so the initial ST segment elevation in acute occlusion MI might be missed , only reciprocal ST depression may be the only initial finding to be observed , say , in posterior STEMI.

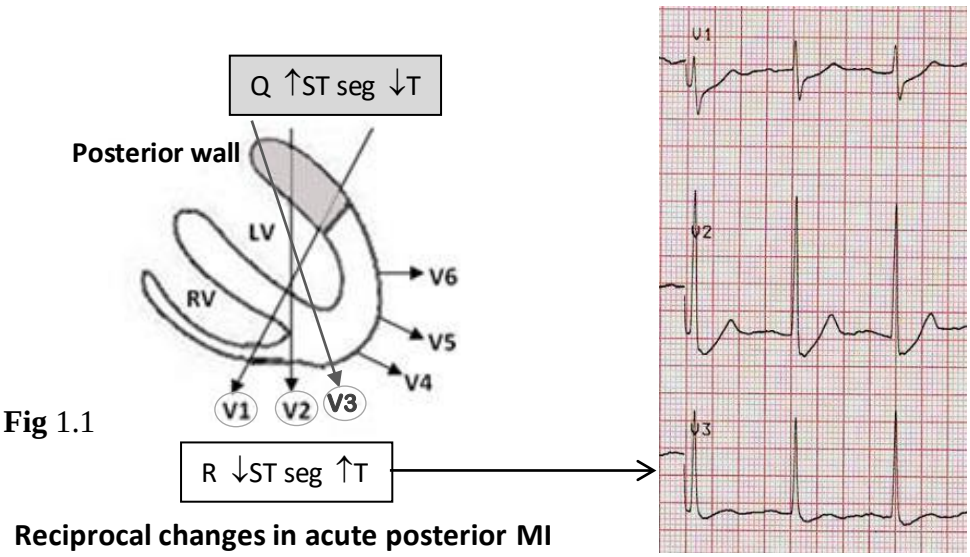


Fig 1.1

Reciprocal changes in acute posterior MI on V1 V2 V3

3. Mechanism

Two major mechanisms have been recognized to explain the reciprocal ST segment change on ECG.

- A mirror like reflection of STEMI as ST segment depression onto the contralateral side (most likely accepted mechanism).
- Concomitant subendocardial ischemia at a contralateral distance created by diverting its blood as a donor to the suffering myocardium with transmural injury. (with less plausible evidence).

A mirror like reflection of STEMI as ST segment depression onto the contralateral side

Myocardial injury generates electrical gradients between ischemic and non-ischemic tissue, producing so-called "current of injury". This current creates vector directed toward the affected region.

- Leads facing the injured myocardium record ST elevation
- Leads facing away record ST depression (reciprocal change)

Therefore , it can be said that reciprocal changes are a natural consequence of the spatial orientation of injury vector rather than independent pathological event.

Experimental evidence

This study demonstrated that in a **pig heart model** without associated subendocardial ischemia, acute coronary artery occlusion produced reciprocal ST-segment depression and deepening of T waves in ECG leads facing distant nonischemic regions. However, these reciprocal changes were not observed in the local extracellular electrograms directly recorded from the nonischemic myocardial areas.

This is obvious by this pig heart model that reciprocal ST segment depression is recorded in those ECG leads which overlie distant nonischemic zone. This is further strengthened by the

failure to demonstrate this phenomenon of reciprocal change if the leads are directly based over the extracellular zone without having any evidence of myocardial injury.

Ref : Mechanism and diagnostic potential of reciprocal ECG changes induced by acute coronary artery occlusion in pigs
Francisco J.Norieg MD Esther Jorge DVM, PhD Dabit Arzamendi MD Juan Cinca MD, PhD
Heart Rhythm Volume 10, Issue 6, June 2013, Pages 883-890
https://www.sciencedirect.com/science/article/abs/pii/S1547527113001653

Concomitant subendocardial ischemia at a contralateral distance created by diverting its blood as a donor to the suffering myocardium with transmural injury

Another possible mechanism (with less plausible evidence) for such ST segment depression might be attributed to subendocardial ischemia by diverting its blood through collaterals to the infarct zone - as a safeguard mechanism. This occurs more likely with multivessel disease.

4. Lead-wise Patterns of Reciprocal Changes

In patient with acute ST segment elevation with no otherwise cause for this , ST segment depression has a specificity and positive predictive value of approx. 93% for the diagnosis of acute myocardial infarction.
While explaining the diagnosis of STEMI one should always keep the following sketch in mind and its sideway written wordings for the purpose :

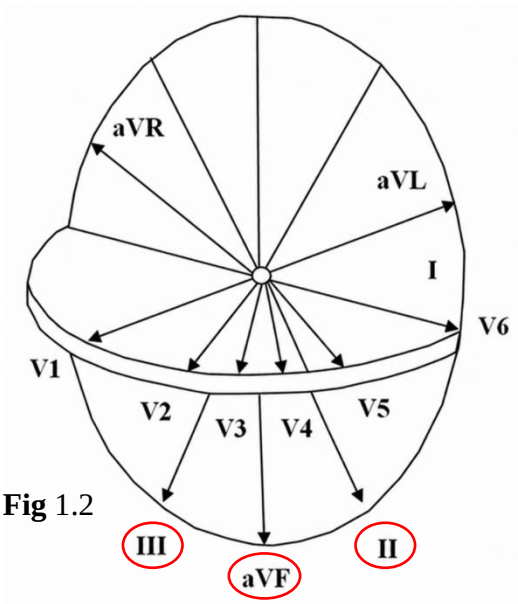


Fig 1.2

- ☐ Vertical and horizontal planes are almost placed at right angle to each other.
- ☐ Lead aVL and inferior leads (II , III and aVF) are placed at more than 90^0 to each other.
- ☐ V1-V4 are also situated reciprocally to inferior leads (II, III and aVF)

This is now obvious that the changes over inferior leads (II, III and aVF) are the most important to be observed herein. That's why , inferior leads are kept within red marked circles in the above sketch.

The coupling of ST elevation vs reciprocal depression in the concerened leads in realtion with the site of STEMI is illustrated as below :

- ST elevation: II, III, aVF
  Reciprocal depression: I, aVL ⟶ Inferior STEMI
- ST elevation: V1–V4
  Reciprocal depression: II, III, aVF ⟶ Anterior STEMI
- ST elevation: I, aVL, V5–V6
  Reciprocal depression: inferior leads ⟶ Lateral STEMI
- Posterior STEMI
  Primary event not directly visualized
  Reciprocal changes:

  ST depression in V1–V3 with Tall R waves

Clinical Implications

Recognition of reciprocal changes aids in:

Prompt diagnosis of acute coronary occlusion
Risk stratification
Decision-making regarding urgent reperfusion therapy

In some cases, reciprocal changes may be the only early clue, especially in subtle or evolving infarction.

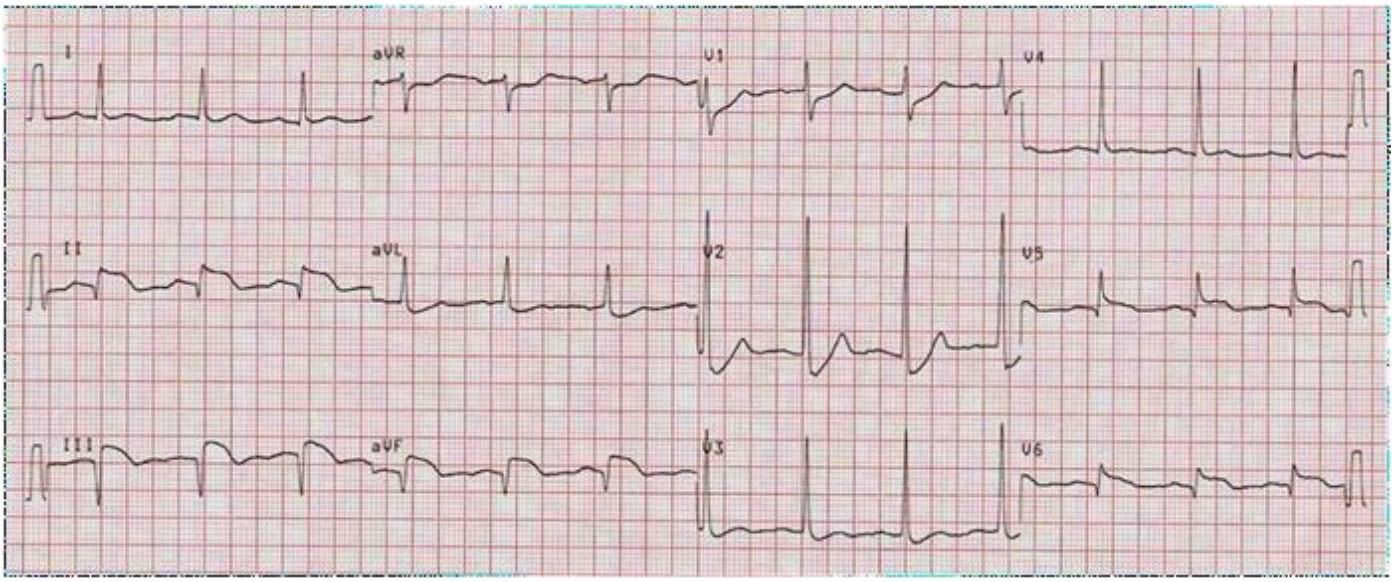
5. Limitations of such reciprocal ST changes

Reciprocal changes embody the fundamental principle that the ECG reflects vectorial summation of myocardial electrical activity. But there are certain limitations with this principle :

- ❒ Not always present in all STEMI cases
- ❒ May be subtle or transient
- ❒ Interpretation may be affected by the associated secondary changes. But rather than being dismissed as secondary findings , they should be actively sought and interpreted in the light of reciprocal ST changes as a consequence of STEMI.

Primary ST depression	**Secondary ST depression**
• Causal factor : coronary insufficiency	• Causal factor : Abnormal depolarization examples: LVH , Bundle branch block , Ventricular pacing
• Features Reciprocal ST depression Dynamic changes Associated symptom	• Features Appropriate discordance Fixed pattern No dynamic evolution

6. Illustration by ECG



Source : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Prinicipal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

(This ECG belongs to middle aged man presenting as sudden severe chest pain since 4 hours)

Findings on ECG :

- ECG shows Q waves with ST elevation in II , III , aVF (ST in II > III) and also over V5-6. **Reciprocal ST depression** in aVL. These findings are suggestive of proximal left circumflex artery occlusion.
- **Reciprocal ST depression** in V1-V3 with tall R and upright T suggests acute posterior infarction , which in this case suggestive of PDA involvement as a branch of 'left dominant LCx.

  All these features suggest Dominant LCX lesion.

  If the posterior descending artery is supplied by the circumflex artery (LCX), then the coronary circulation can be classified as **"left-dominant"** (In 20% of cases).

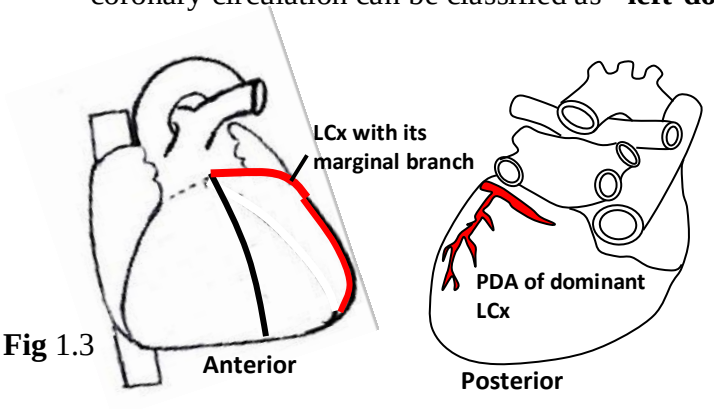


Fig 1.3

The dominant LCx supplies the following cardiac surfaces through its different branches :

- Inferior wall (ST elevation in leads II , III and aVF with reciprocal ST depression in aVL)
- Posterior wall event mirrors its image through V1-V3 (tall R-wave , ST depression and upright tall T-wave)
- Lateral LV territory is reflected as ST elevation over V5-V6

Short conceptual Summary :

- ☐ In dominant LCx occlusion , the injury vector is predominantly directed inferiorly and posteriorly rather than towards the high lateral wall.
- ☐ Therefore, the ECG may show inferior STEMI with posterior reciprocal changes and low lateral ST elevation in V5-V6, even in the absence of ST elevation in lead I and aVL.

7. Take-Home Message

- ☐ Reciprocal ST depression is an essential component of ECG interpretation in STEMI. They enhance diagnostic confidence, improve localization, and provide insight into the extent of myocardial injury.
- ☐ Every ST elevation casts a reciprocal shadow, and within that shadow lies the full story of myocardial infarction.
- ☐ Key Clinical Pearls
  - Always look for reciprocal changes in suspected STEMI
  - Their presence increases diagnostic specificity
  - They often indicate more severe or proximal lesions
  - Absence does not exclude infarction
- ☐ Reciprocal ST depression is a mirror image, not a separate disease. Each ECG lead views the heart from a unique angle; opposing leads record opposite manifestations of the same event.
- ☐ Reciprocal changes may precede, accompany, or evolve with primary ST elevation always consider serial ECGs.
- ☐ Reciprocal ST changes gain meaning only when interpreted in the clinical context.
- ☐ Understanding these principles allows the clinician to move beyond pattern recognition and appreciate the ECG as a dynamic reflection of myocardial injury.

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Heart Rhythm Volume 10, Issue 6, June 2013, Pages 883-890
https://www.sciencedirect.com/science/article/abs/pii/S1547527113001653

REGRESSION OF ECG CHANGES IN ACUTE CORONARY OCCLUSION : MIRACLE OF EARLY ANTITHROMBOTIC THERAPY OF ENDOGENOUS FIBRINOLYSIS ?

ECG

REGRESSION OF ECG CHANGES IN ACUTE CORONARY OCCLUSION : MIRACLE OF EARLY ANTITHROMBOTIC THERAPY OR ENDOGENOUS FIBRINOLYSIS ?

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OUTLINE

Regression of ECG Changes in Acute Coronary Occlusion : Miracle of Early Antithrombotic Therapy or Endogenous Fibrinolysis ?

A Narrative Review

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A patient with acute true posterior occlusion MI (in association with inferior STEMI) received a loading dose of DAPT (aspirin + Ticagrelor) , high-dose atorvastatin, and low-molecular-weight heparin (Enoxaparin) immediately after the diagnosis. He had presented with the sudden onset of epigastric pain , nausea and sweating lasting for about half an hour. Within a short period, the symptoms subsided, and the characteristic ECG changes regressed markedly. A colleague posed a simple yet profound question : Was this dramatic improvement attributable to the pharmacological effects of the administered drugs, or had Nature accomplished spontaneous fibrinolysis ?

- ☐ **Modern therapy does not compete with endogenous fibrinolysis—it creates the conditions under which endogenous fibrinolysis can succeed.**
- ☐ **When an occluded coronary artery reopens after early antithrombotic therapy, the question is not whether drugs or spontaneous fibrinolysis deserve the credit, but how each contributed to the restoration of coronary flow.**

A useful analogue : Imagine a river blocked by a landslide. Endogenous fibrinolysis is the river's own current trying to clear the obstruction. Antithrombotic drugs do not remove the rocks, but they prevent new rocks from being added. Consequently, the river's current has a greater chance of reopening the channel.

1. Introduction (Keypoints)

- ○ A true posterior MI (Occlusion Myocardial Infarction or OMI) typically presents on the standard 12-lead ECG as horizontal ST-segment depression with tall, prominent R waves and upright T waves in leads V1-3. This is the "mirror image" of what is happening on the posterior wall.

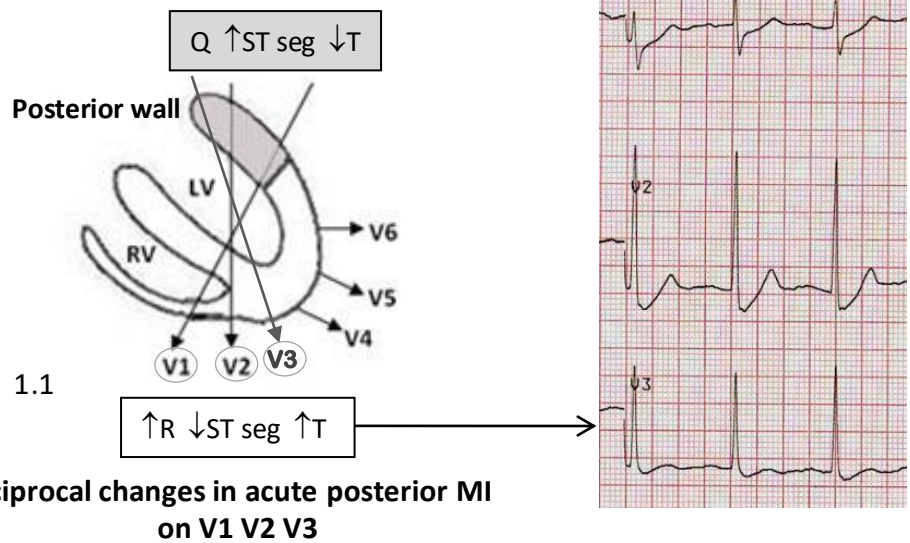


Fig. 1.1

Reciprocal changes in acute posterior MI on V1 V2 V3

NB : If True Posterior MI occurs with inferior STEMI → think RCA -PDA as the culprit.

- The Biology of the Culprit Thrombus

An acute coronary thrombus is in a constant state of:

- Platelet aggregation
- Fibrin deposition
- Endogenous fibrinolysis
- Fragmentation
- Re-thrombosis

> **The clinician may think of these infallible sequences as a "battlefield" where thrombosis and fibrinolysis coexist.**

- In the initial stage of an acute myocardial infarction (AMI), the concerned clinician administers dual antiplatelet therapy (DAPT) combined with an anticoagulant , e.g. , Low Molecular Hiparin (Enoxaparin) and High-dose Atorvastatin. This strategy instantly halts clot propagation, prevents re-occlusion, and stablizes the ruptured plaque. Each drug has a group of beneficial effects on the existing thrombus :

DAPT • Inhibits further platelet activation, • Prevents propagation of the thrombus, • Reduces recurrent platelet aggregation. Thus, it shifts the balance away from continued thrombosis.	**Enoxaparin** It inhibits factor Xa (and to a lesser extent thrombin). Therefore it: • suppresses new fibrin formation, • prevents enlargement of the thrombus, • stabilizes the coronary lesion. Again, it is antithrombotic rather than thrombolytic.
High-dose Atorvastatin Beyond lipid lowering, statins exert rapid pleiotropic effects Within hours they may: • improve endothelial nitric oxide production, • reduce vascular inflammation, • decrease tissue-factor expression, • improve endothelial function, • stabilize the ruptured plaque. Although statins are not thrombolytic agents, they create a vascular environment that is less conducive to ongoing thrombosis.	

- The role of spontaneous reperfusion by endogenous fibrinolysis.
Autothrombolysis is mediated principally by the body's endogenous fibrinolytic system:

Steps :

Endogenous plasminogen activation → plasmin → fibrin degradation → thrombolysis

- Medical therapy does not directly produce autothrombolysis, but it can facilitate successful endogenous fibrinolysis by preventing fresh platelet and fibrin deposition over the thrombus.

  It would be worthwhile to mention a useful analogue in this context :

  Imagine a river blocked by a landslide. Endogenous fibrinolysis is the river's own current trying to clear the obstruction. DAPT and enoxaparin do not remove the rocks, but they prevent new rocks from being added. Consequently, the river's current has a greater chance of reopening the channel.

- **A profound and pertinent question may arise here** – whether this scenario addresses the dynamic biology of coronary thrombosis, or the beneficial impact of opening of the thrombus by endogenous fibrinolysis.

  The answer lies in understanding that the culprit thrombus is not a static structure but a living , evolving entity. This perspective leads to a better understanding of the underlying process.

  This article attempts to explore the biological basis of this intriguing clinical observation.

2. Biological Basis – Conceptual Framework

'It takes the heart's grief and transmutes it into strength;
thus , it fills the heart once more with the joy of relief.'

Prompt administration of dual antiplatelet therapy and anticoagulation favours endogenous coronary reperfusion by preventing further thrombus propagation and allowing intrinsic fibrinolytic mechanisms to restore coronary blood flow. An important point here is at any moment , ECG reflects the balance between :

- Thrombus Propagation
- Endogenous fibrinolysis (Autothrombolysis)

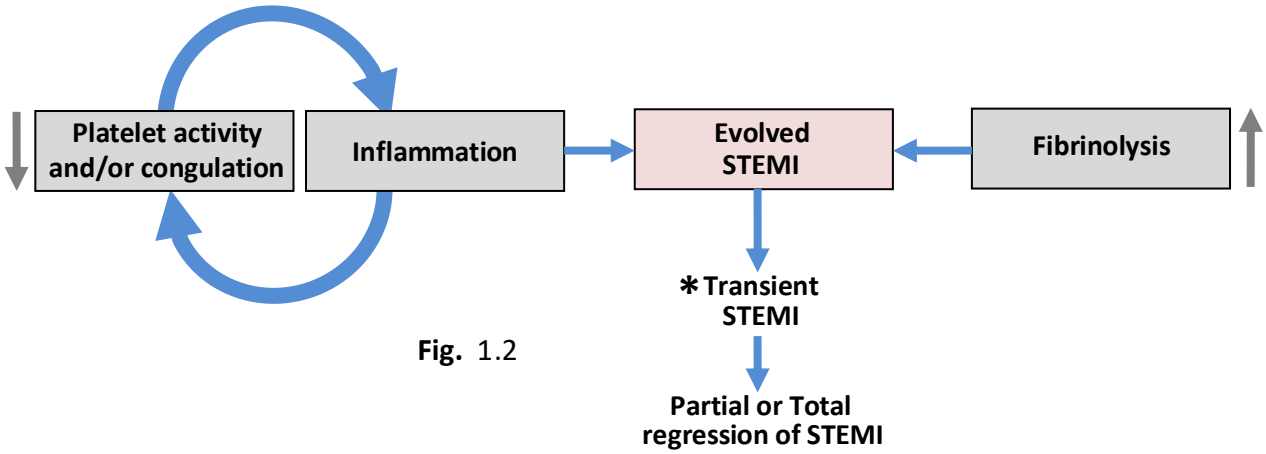


Fig. 1.2

∗ **Transient STEMI** (t-STEMI) or spontaneous reperfusion, which occurs in approximately 20% of patients with STEMI , is generally defined as spontaneous ST-segment improvement on electrocardiogram and / or the achievement of initial thrombolysis in myocardial infarction grade 2-3 flow in the infarct-related artery (IRA) before primary percutaneous coronary intervention (PPCI) and typically coincides with resolution or improvement of symptoms.

Ref : Spontaenous reperfusion in STEMI : Its mechanisms and possible modulation ,
Author : Joshua H Leader, Rahim Kanji, Diana A Gorog.
https://journals.viamedica.pl/polish_heart_journal/article/view/99737

NB : Thrombolysis in Myocardial Infarction (TIMI) flow grade criteria are as follows : grade 0 indicates no blood flow beyond the occlusion, grade 1 indicates incomplete filling of the distal vessel beyond the occlusion, grade 2 indicates partial filling of the distal vessel beyond the occlusion within 3 cardiac cycles, and grade 3 indicates complete filling of the distal vessel beyond the occlusion within 3 cardiac cycle. [Ref. 2, p 86]

The evolution of the ECG in acute occlusion myocardial infarction may be viewed as the surface manifestation of a dynamic equilibrium between ongoing coronary thrombosis and endogenous fibrinolysis. Modern antithrombotic therapy does not directly lyse the thrombus; rather, it suppresses further thrombus propagation, permitting intrinsic fibrinolytic mechanisms to restore coronary perfusion whenever possible

Evidence based points :

- ❒ The mainstay of pharmacological treatment of ACS patients is dual antiplatelet therapy. Anticoagulants (including vitamin K antagonists, fondaparinux, and heparin) have been shown to alter fibrin clot structure by increasing clot permeability and altering clot density. [Ref. 1 , p 370]
- ❒ In patient with ischemia time < 4 hours , the thrombi are mainly composed of white thrombi, which are predominantly platelet-based, while in patients with ischemia time of 4-7 hours , the thrombi are mainly composed of mixed thrombi. White thrombi are mainly composed of platelets, so early antiplatelet therapy has a significant effect on thrombolysis and SR. [Ref. 2 , p 92]
  SR = Spontaneous reperfusion

  SR is associated with early medical contact , early use of antiplatelet drugs, and the time of action after taking antiplatelet drugs. Early use of antiplatelet drugs can increase the incidence of SR. Patient should seek medical attention as soon as possible and receive antiplatelet therapy after experiencing myocardial infarction symptoms. [Ref. 2 , p 92]
- ❒ Among patients with spontaneous reperfusion, those with complete ST-segment resolution had more rapid endogenous fibrinolysis times and had reduced platelet reactivity than those with only partial ST-segment resolution [Ref. 1 , p 366]
  Endogenous fibrinolysis : Despite optimal contemporary treatments with PPCI and antithrombotic medications, studies have shown that impaired endogenous fibrinolyis is an independent predictor of adverse cardiovascular outcomes in patients with ACS. [Ref. 1 , p 368] PPCI = Primary Percutaneous Coronary Intervention.

The Dynamic Equilibrium of Coronary Thrombosis

- Plaque rupture initiates thrombosis.
- Endogenous fibrinolysis begins simultaneously.
- DAPT, anticoagulation, and high-intensity statins suppress further thrombus propagation and stabilize the culprit lesion.
  The clinical outcome depends on the balance between thrombosis and endogenous fibrinolysis.

It would be also worthwhile to mention here that restoration of myocardial perfusion permits gradual recovery of ATP production, thereby facilitating normalization of ATP-dependent ionic transport, including Na^+/K^+-ATPase activity, with consequent improvement in transmembrane ionic gradients and electrical stability.

Early myocardial salvage is achieved not by a single miracle but by a trilogy of biological events: arrest of thrombus propagation, endogenous thrombus resolution, and restoration of myocardial ionic homeostasis.

3. An Interesting Case

Source : Dr. Satish kumar , Senior Consultant Physician and Cardiologist , Wellmark Hospital , Bokaro Steel City. The case was shared on the CME INDIA on 05.07.2026. It is discussed here with his kind permission.

History : 76 years male, non-smoker , non-diabetic with history of hypertension (well controlled on Amlodipin 5 mg OD), reported to the casualty in the early morning with complaints of sudden onset epigastric pain, nausea and sweating for about half an hour.

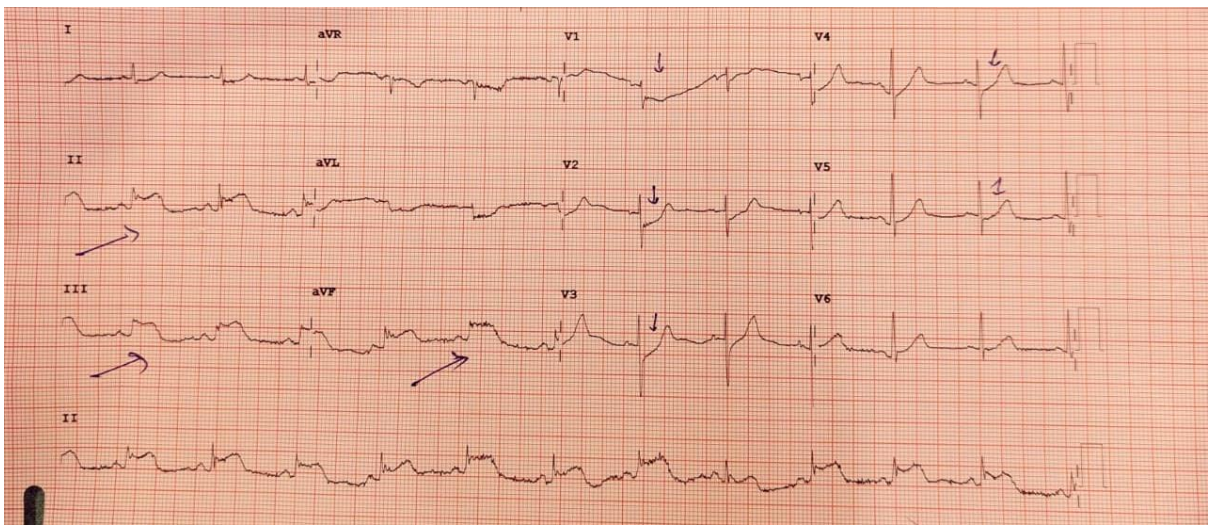
On examination , he was found restless with a low volume pulse of 70pm, regular, b/l symmetrical , BP of 90/70 and SPO2 at room air was 96%. There was no S3, Bruit, Murmur or Rub. Chest was clear.

As his ECG (ECG 1) was showing inferior STEMI + True Posterior MI, he was immediately given loading doses of 4 tablets of ASA (each 81 mg) , 2 tablets of Ticagrelor (180 mg) and 2 tablets of Atorvastatin (80 mg). His BP improved with IV fluids (about 450 ml given over 20 mins). The patient was shifted to the CCU and Cath Lab was activated.

His Coronary Angio was almost normal with a minor 30-40% residual plaque in Proximal RCA with good flow. He didn't receive any thrombolytic therapy at PCI centre.

ECG 1 **(Immdediately after reporting to the casuality)**

Acute posterior MI with inferior STEMI→ RCA-PDA involvement



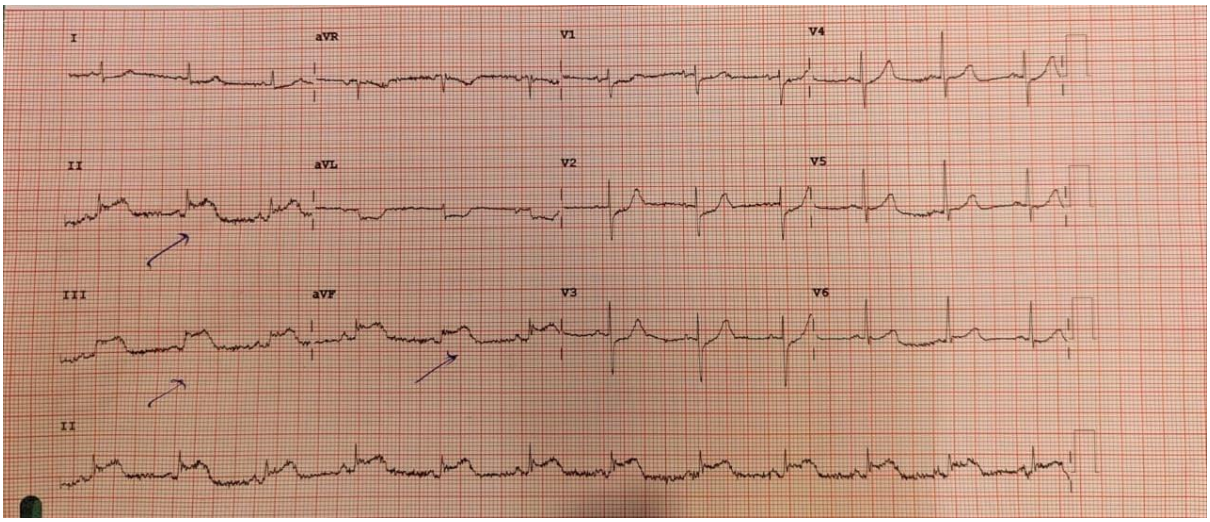
Inferior STEMI

ST elevation in leads II, III , aVF with reciprocal ST depression in leads I and aVL

True Posterior MI

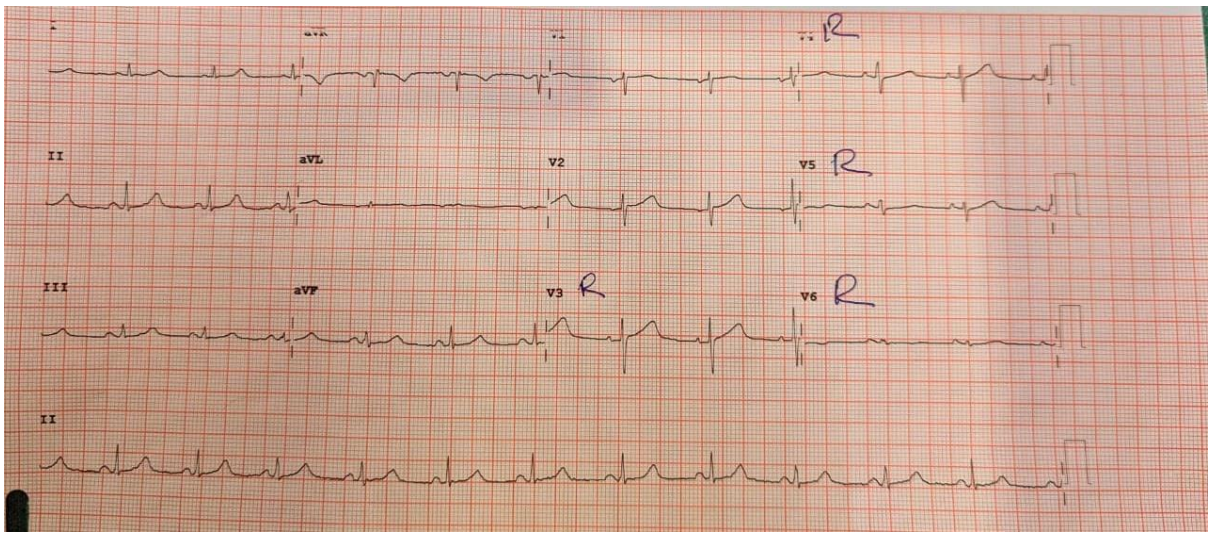
- ST depression in leads V1-V3 with corresponding upright T-waves.
- Here the absence of concurrent tall and prominent R-wave is most likely due to slow PMI evolution.

ECG 2 **(Just after coming to the CCU) Findings still persisting but with somewhat resolution of ST depression in leads V1-V3**



The patient was shifted immediately to the CCU. His repeated ECG was showing the findings noted as above. His Trop-T was negative. Bed side Echo however suggested subtle RMWA in inferior and Posterior Walls of LV with normal LVEF and RV dilatation. Expecting RC occlusion , Cath Lab was activated , he was given Enoxaparin (60 mg) IV bolus.

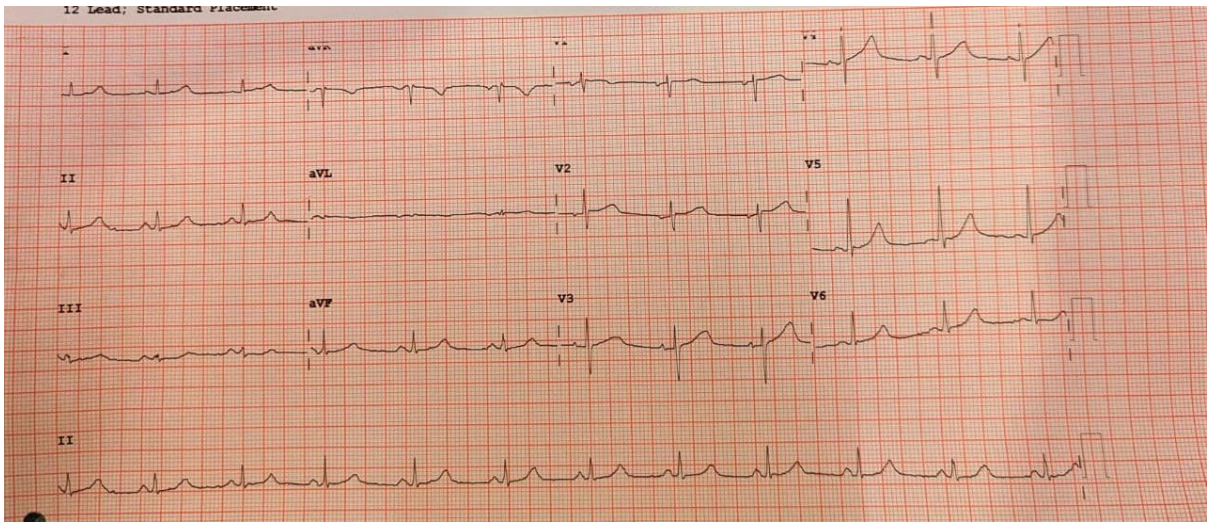
ECG 3 : **Before being taken to the Cath Lab , his ECG was repeated with Right Chest leads to look ST elevation in V4R (~ 1 hour)**



ST Elevation in V4R was not so there and to our surprise ST segment elevation in II, III and aVF and depression in V1 and V2 had significantly normalised to baseline.

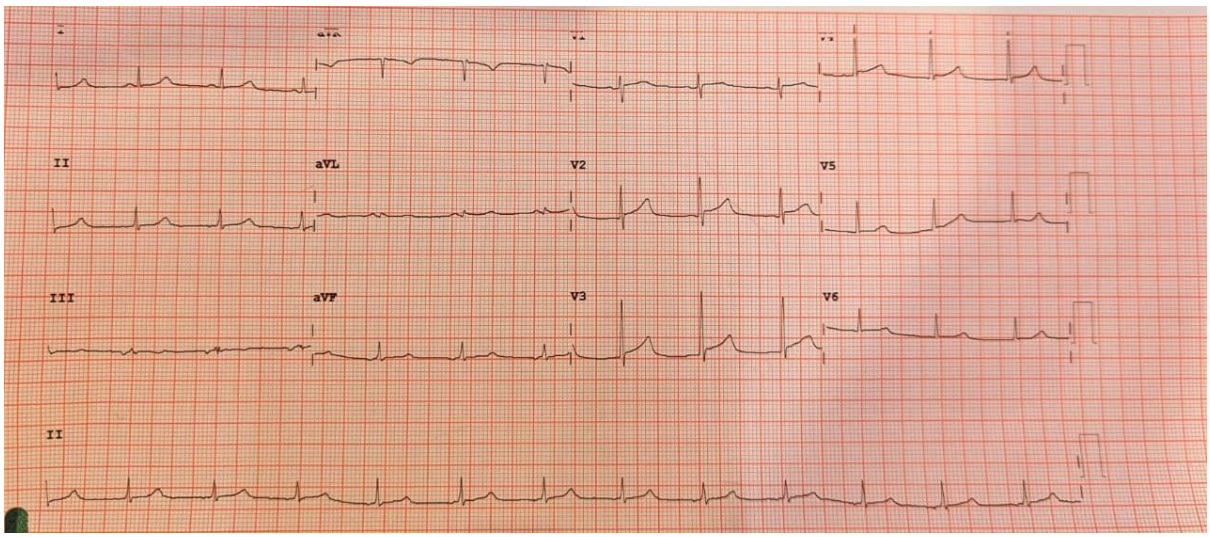
As discussed earlier, his coronary angiogram was essentially normal, thereby obviating the need for primary PCI (He didn't receive any thrombolytic therapy).

ECG 4 : **Approximately immediate after 3rd ECG**



Normalization of ST elevation in inferior leads maintained , but just a mildly ST elevation was noted in leads V2 and V3.

ECG 5 **(6 hours later)**



Complete normalization of inferior STEMI continued , but with somewhat more mild ST elevation was noted in leads V1-3 with the appearance of prominent R in leads V2-3.

- This mild ST elevation is known as ‘overshoot’ phenomenon. This indicates successful restoration of blood flow over the posterior wall. As the posterior wall recovers from the impact of injury current , posteriorly placed leads often show a slight ST depression and consequently , as a mirror image mild ST segment elevation is seen in anteroseptal leads (V1-V3).
- STEMI evolution rarely proceeds through rigidly separated stages. No surprise to see the appearance of prominent R-wave so late in V2-V3 as a representative of posterior Q wave.

NB : His subsequent ECGs and Echo were normal over the next 3 days. Even his Quantitative Troponin I levels remained within normal.

4. Discussion

Based on the clinical history, serial ECG findings, and ancillary investigations including echocardiography and coronary angiography (CAG), this case appears to represent **spontaneous coronary reperfusion**. The findings supporting this interpretation are as follows:

- The patient's symptoms of epigastric pain, nausea, and vomiting improved rapidly after the administration of loading doses of dual antiplatelet therapy and anticoagulation
- Complete resolution of the inferior STEMI pattern on serial ECGs, along with near-complete resolution of the reciprocal ST-segment depression in leads V1–V3 (in the setting of inferior STEMI with true posterior myocardial infarction).
- Mild residual ST-segment elevation ("overshoot" phenomenon), most evident on ECG 5, suggesting successful restoration of blood flow to the posterior left ventricular wall.

In summary, these serial ECG changes are highly suggestive of **transient STEMI (t-STEMI)**. Transient STEMI is reported in approximately 20% of patients presenting with STEMI and is generally characterized by spontaneous improvement of ST-segment deviation before primary percutaneous coronary intervention (PPCI), accompanied by restoration of good TIMI flow in the infarct-related artery (IRA) and, typically, resolution or marked improvement of ischemic symptoms.

As discussed previously, the most plausible mechanism in this case appears to be the prompt administration of dual antiplatelet therapy together with anticoagulation, which prevented further thrombus propagation while facilitating endogenous fibrinolysis (autothrombolysis), thereby restoring coronary blood flow.

An important concept illustrated by this case is that, at any given moment, the ECG reflects the dynamic balance between two opposing processes:

- **Thrombus propagation**
- **Endogenous fibrinolysis (autothrombolysis)**

The evolving ECG pattern represents the net effect of these competing mechanisms.

In my opinion, this case represents a **par excellence** illustration of spontaneous coronary reperfusion, as demonstrated by the sequential ECG changes , **excellently put by Dr. Satish kumar.** The serial ECGs provide a rare and compelling visual documentation of the transition from acute coronary occlusion to successful spontaneous reperfusion.

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5. Take-Home Message

The prompt regression of ischemic ECG changes following administration of a loading dose of antithrombotic should not be interpreted as proof of direct pharmacological thrombolysis. Rather, these therapies inhibit further thrombus propagation and stabilize

the culprit lesion, thereby creating favourable conditions for endogenous fibrinolysis to restore coronary perfusion. The observed reperfusion is therefore best understood as the result of a biological partnership between Nature's intrinsic fibrinolytic system and timely administered pharmacotherapy

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NON-ST-ELEVATION MYOCARDIAL INFARCTION : ECG REFLECTION OF SUBENDOCARDIAL ISCHEMEIC INSULT

ECG

NON-ST-ELEVATION MYOCARDIAL INFARCTION : ECG REFLECTION OF SUBENDOCARIDAL ISCHEMIC INSULT

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OUTLINE

Non-ST-Elevation Myocardial Infarction : ECG Reflection of Subendocardial Ischemic Insult

A Narrative Review

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When a catastrophe strikes in life, an individual instinctively attempts to escape through the wisest possible pathway, trying to minimize the extent of injury as much as possible; however, despite such efforts, the damage may still progress with more harmful consequences.

Non-ST-segment elevation myocardial infarction (NSTEMI) behaves in a somewhat similar manner. Initially, the ischemic insult may remain confined to the subendocardial region, but if left untreated or neglected, it may extend across the entire myocardial wall, ultimately evolving into a STEMI.

- ☐ **NSTEMI reflects subendocardial myocardial necrosis occurring without full-thickness transmural electrical injury.**
- ☐ **This entity demands careful and thoughtful ECG interpretation to identify the underlying myocardial jeopardy.**

This remains a continuous clinical challenge , as progressive worsening of coronary low compromise or thrombotic propagation may ultimately transform subendocardial necrosis into transmural myocardial injury, culminating in STEMI. In other words , the transition from NSTEMI to STEMI reflects the progressive failure of residual myocardial perfusion.

1. Introduction (Keypoints)

- ○ Non-ST-elevation myocardial infarction (NSTEMI) represents subendocardial myocardial necrosis resulting from an acute ischemic insult without persistent transmural ST-segment elevation on the ECG. However, if the ischemia progresses or remains untreated, the subendocardial injury may extend transmurally and evolve into ST-elevation myocardial infarction (STEMI). Therefore, NSTEMI occupies a distinctive position within the spectrum of acute coronary syndromes.
- ○ Here ischemic injury predominantly affects the vulnerable subendocardial layers, which are exposed to:
  - higher intramural pressure,
  - greater metabolic demand,
  - and relatively compromised perfusion reserve.

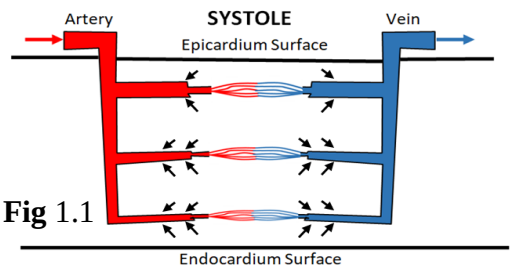


Fig 1.1

Further to say , this subendocardium is also more vulnerable to ischemic injury because of the greater systolic compressive forces exerted by its close proximity to the high-pressure left ventricular (LV) cavity. In addition, the epicardial coronary arteries must traverse a longer intramyocardial course to supply the subendocardial region, making this territory more susceptible to reduced perfusion.

- As a result, injury vector becomes directed toward the subendocardial territory, producing diffuse ST-segment depression and other repolarization abnormalities. In many patients, reciprocal ST elevation appears in lead aVR due to the global orientation of these injury currents. In other words, diffuse ST depression with reciprocal aVR elevation often reflects extensive subendocardial ischemic burden.
- Importantly , NSTEMI is not merely a subendocardial form of infarction , rather it often reflects a dynamic electro-pathological state wherein loss of residual perfusion can subsequently extend this ischemic insult transmurally, ultimately cultiminating in STEMI. Thus , this clinical entity is a moving electrophysiological scenario , not frozen ECG photographs.
- The definition of NSTEMI integrates biochemical evidence of myocardial necrosis with clinical and electrophysiological evidence of acute ischemia. Therefore , Suspect NSTEMI in patients with ischemic symptoms, dynamic ischemic ECG changes without classical STEMI criteria, and supportive troponin rise/fall (a rise and/or fall in cardiac troponin values, with at least one value above the 99th percentile URL).

2. Electrophysiological Basis of Non-STEMI

Understanding the concept of subendocardial necrosis in NSTEMI allows a better correlation between coronary anatomy , ischemic progression , and myocardial electrical behaviour.

NSTEMI most commonly results from:

- subtotal coronary artery occlusion,
- unstable atherosclerotic plaque,
- platelet-rich thrombus formation,
- or severe diffuse coronary insufficiency.

During subendocardial ischemia, the injury current is directed inward toward the ischemic subendocardial region. This occurs because electrical current flows from relatively healthy myocardium with a higher resting membrane potential toward the ischemic myocardium, which has a lower membrane potential. **Similarly in subendocardial infarction, the injured subendocardial zone is relatively more depolarized and electrically more negative than the overlying subepicardial myocardium, thereby inducing inwardly directed injury current typically reflected on ECG as ST-segment depression.**

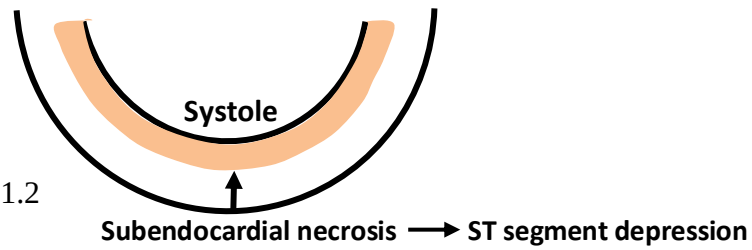


Fig 1.2

The ECG dynamically mirrors the anatomical depth and directional progression of ischemic insult.

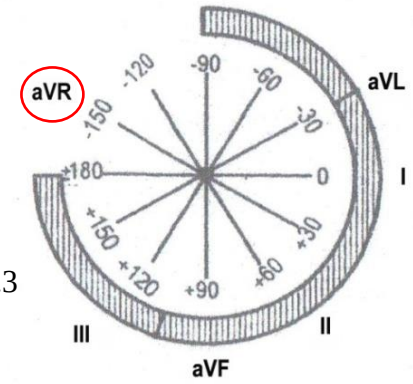


Fig 1.3

NSTEMI commonly manifests as ST-segment depression in multiple ECG leads, often accompanied by ST-segment elevation in lead aVR. **Electrophysiologically** , diffuse ST depression generates reciprocal injury-vector orientation toward rightward, superior and basal electrical fields. Lead aVR directly faces this vector and therefore records reciprocal ST elevation. This factual concept is the very important part of electrophysiological changes in NSTEMI . Apart from subendocardial necrosis, this diffuse ST depression with reciprocal ST elevation in lead aVR may also occur with the following conditions and therefore it needs D/D :

- extensive subendocardial ischemia,
- severe multivessel coronary disease,
- left main coronary artery compromise,
- proximal LAD disease,
- or severe global oxygen supply-demand mismatch.

In nutshell to say : in NSTEMI residual coronary flow partially limits subendocardial necrosis depth , thereby , restricting injury predominantly to the subendocardial region.

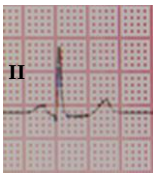
That's why, a separate terminology NSTEMI was coined by electrophysiologists. Factually no single cardiologist is credited with inventing the term NSTEMI. The terminology emerged collectively from guideline-based cardiology practice during the late 1990s. The ACC/AHA 2000 guidelines popularized and standardized the terms STEMI and NSTEMI worldwide.

3. Corresponding ECG changes

Non-ST-Elevation Myocardial Infarction can become more meaningful when the corresponding ECG changes are delineated. The ECG dynamically mirrors the anatomical depth and directional spread of myocardial ischemic injury. Subendocardial necrosis depresses the ST segment inwardly.

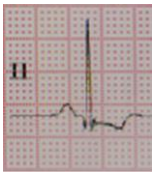
- ☐ **Horizontal or downsloping ST segment depression $\geq$ 0.5 mm at the J-point $\geq$ 2 contiguous lead points towards myocardial ischemia , usually of subendocardial origin.**
- ☐ ST depression $\geq$ 1 mm is rather more specific having a worse prognosis.
- ✓☐ ST depression $\geq$ 2 mm is usually associated with a higher possibility of Non-ST elevation myocardial infarction. ST depressions in NSTEMI are frequently accompanied by T inversions or flat T waves , but the primary ECG finding is the ST segment depression.
- ☐ Upsloping ST segment (non-specific) is rarely caused by ischemic insult. It is usually physiological in nature , seen in normal persons usually during physical exercise and resolves rapidly once the exercise is stopped.

Fig 1.4



Horizontal ST depression
with a sharp angle in between ST segment and T wave. This is the earliest expression of ST segment depression in NSTEMI.

Fig 1.5



Downsloping ST segment depression
The ST segment is depressed below the isoelectric line at its origin and then it slops further downwards

In nutshell to say :

- NSTEMI represents the electrophysiological expression of predominantly subendocardial myocardial injury occurring under critically compromised coronary perfusion.
- Diffuse ST depression with ST elevation in aVR often reflects extensive global subendocardial ischemic insult.

4. NSTEMI in the Modern Era of Occlusion MI (OMI)

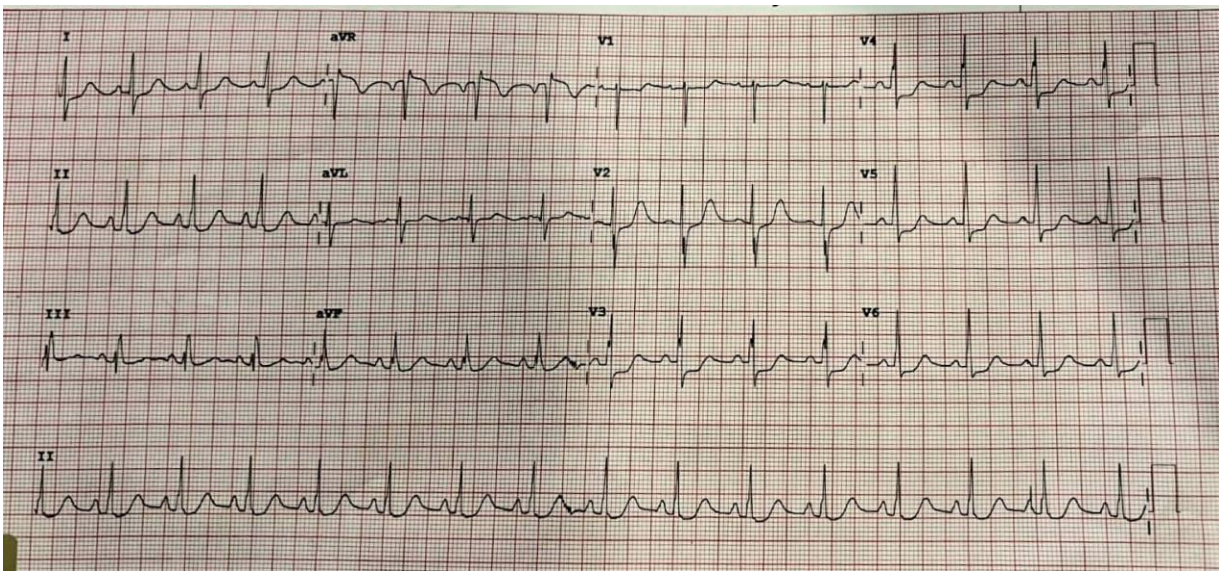
Traditionally, NSTEMI has been regarded as the electrophysiological reflection of predominantly subendocardial infarction usually resulting from subtotal coronary obstruction. However, contemporary understanding through the OMI paradigm has demonstrated that certain patients categorized clinically as NSTEMI may actually harbour acute coronary occlusion despite absence of classical STEMI criteria on ECG. But NSTEMI should not always be considered synonymous with occlusive MI , rather should be considered as a dynamic ischemic spectrum requiring deeper electrophysiological interpretation.
The following concepts are important:

- Although NSTEMI classically reflects subendocardial infarction, the OMI concept highlights that it may sometimes remain electrically concealed within non-ST-elevation ECG patterns. One of the best examples is the **de Winter ECG pattern**, an STEMI equivalent typically associated with acute proximal LAD occlusion. It is characterized by upsloping ST-segment depression at the J point in the precordial leads, accompanied by tall, symmetrical T waves, often with slight ST-segment elevation in lead aVR.
- The OMI paradigm does not negate the concept of NSTEMI; rather, it reveals the hidden complexity within the NSTEMI spectrum.
- **OMI generally represents a more advanced stage of ischemic progression with a critical transmural myocardial involvement extending beyond the subendocardial layer.** Thus, the distinction between NSTEMI and OMI may sometimes reflect the timing and electrical expression of ischemia rather than an absolute anatomical separation.

In this context, NSTEMI has been discussed here from both its traditional and contemporary perspectives. Early recognition and deeper understanding of NSTEMI , compared to its counterpart OMI may contribute to improved patient outcomes.

5. Illustration by an interesting ECG

A 53-year-old woman with diabetes presented with classical angina for 3 hours, restlessness and sweating. Troponin was still negative. Echo showed subtle global hypokinesia with LVEF around 50%.



Source : CME INDIA on 24.05.2026 by **Dr. Satish Kumar** , Senior Consultant Physician and Cardiologist , Wellmark Hospital , Bokaro (with his kind permission)

Findings :

- Diffuse ST depression (1-3 mm) with reciprocal ST elevation in lead aVR
  (hallmark of subendocardial ischemic insult)
  This is to be mentioned here that NSTEMI most commonly results from subtotal coronary artery occlusion And the patient was taken directly to the Cath lab.

"Angiography revealed near-subtotal ostial LMCA occlusion with a critical mid-RCA lesion. During the procedure, she developed repeated VT episodes and required CPR support. In a very high-risk setting, with no CTVS backup, urgent PCI was performed. Both LMCA and RCA were stented in the same sitting. She was stabilized and discharged well, and later had preserved myocardium with LVEF around 60%". The case was well discussed by Dr. Satish Kumar in CME INDIA on 24.05.2026 and was highlighted further by Dr. N.K. Singh, Editor, www.cmeindia.in

Discussion :

Three key elements must be integrated in such cases: the clinical history, ECG findings (supported by echocardiography), and cardiac troponin levels. An initially negative troponin level in a diabetic woman with classical angina should not delay cath when clinical suspicion is strong enough.

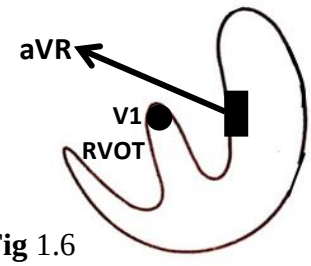


Fig 1.6

Electrophysiologically , diffuse ST depression on ECG generates reciprocal injury-vector orientation toward rightward, superior and basal electrical fields. Lead aVR directly faces this vector and therefore records reciprocal ST elevation.

6. Take Home Message

- ☐ Non-STEMI represents a dynamic manifestation of coronary insufficiency characterized predominantly by subendocardial ischemic insult.
- ☐ The ECG findings of diffuse ST depression with reciprocal ST elevation in aVR provide important insight into the vectorial orientation of widespread subendocardial injury currents and frequently indicate severe coronary pathology.
- ☐ Because coronary perfusion in NSTEMI remains unstable and dynamically evolving, progression toward STEMI may occur when ischemic insult extends transmurally. Thus, careful electrophysiological interpretation of NSTEMI allows the clinician to recognize not merely the presence of ischemic insult, but also its evolving anatomical depth, vectorial orientation, and potential future progression.
- ☐ NSTEMI associated subendocardial infarction may be either diffuse or regionally localized depending upon the underlying coronary pathology and ischemic burden.
- ☐ Contemporary understanding through the OMI paradigm has demonstrated that certain patients categorized clinically as NSTEMI may actually harbour acute coronary occlusion despite absence of classical STEMI criteria on ECG. But NSTEMI should not always be considered synonymous with occlusive MI , rather should be considered as a dynamic ischemic spectrum requiring deeper electrophysiological interpretation.
- ☐ Unlike STEMI, NSTEMI frequently demands:
  - deeper ECG interpretation,
  - integration with troponin kinetics,
  - and understanding of ischemic vectors.

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ST DEPRESSION ON ECG AS A FOOTPRINT OF SUBENDOCARIAL ISCHEMIA

ECG

ST DEPRESSION ON ECG AS A FOOTPRINT OF SUBENDOCARDIAL ISCHEMIA

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OUTLINE

Introduction
Subendocardial ischemia is one of the most fundamental manifestations of an imbalance between myocardial oxygen supply and demand.

ST segment in a normal person
An isoelectric line on the ECG between the end of S wave (the J point) and the beginning of the T wave.

Pathophysiological basis of subendocardial ischemia

- ❒ Anatomic and hemodynamic vulnerability
- ❒ Supply-demand disparity in the subendocardium
- ❒ Transmural flow heterogeneity and vascular compliance
- ❒ Microvascular and enothelial dysfunction

Classification
Classification based on mechanism
Classification based on clinical setting

ECG changes during subendocardial ischemia
Horizontal / downsloping ST segment ± T-wave inversion

Case Study with accompanying ECG

Take-Home Message

References

ST depression on ECG as a footprint of subendocardial ischemia

A Narrative Review

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Just as the sun dips below the horizon, marking the end of the day's warmth, ST-segment depression reflects the waning endurance of the myocardium. It represents the "dusk" of coronary perfusion, forewarning a phase of subendocardial drowning if the supply of oxygenated blood is not restored.

- ☐ **Subendocardial ischemia is one of the most fundamental manifestations of an imbalance between myocardial oxygen supply and demand.**
- ☐ **Horizontal or downsloping ST-segment depression is a well-recognized electrocardiographic pointer of subendocardial ischemia.**

In the setting of coronary insufficiency, reduced perfusion of the subendocardium alters the transmembrane action potential gradient, generating current of injury that is directed away from the overlying recording leads. This results in the characteristic ST-segment depression on the surface electrocardiogram.

Thus, ST-segment depression serves as both a pathological signal and a red flag warning — indicating that coronary perfusion has become inadequate to meet myocardial metabolic demand.

1. Introduction (Keypoints)

- Subendocardial ischemia is a condition in which myocardial oxygen deprivation selectively involves the subendocardial layers of the ventricular wall due to inadequate coronary perfusion, without full-thickness involvement.
- The subendocardium is particularly susceptible due to:
  - Highest oxygen demand
  - Greatest wall stress
  - Reduced perfusion during systole
  - Dependence on diastolic coronary flow

  Hence , it is first region to suffer when coronary perfusion declines.
- In the setting of coronary insufficiency reduced perfusion of the subendcardium generates current of injury which is directed away from the recording electrodes inscribing ST depression on the ECG. Thus , ST depression is the electrocardiographic signature of subendocardial ischemia.
- Subendocardial ischemia is commonly seen in stable angina , unstable angina , non-ST elevation myocardial infarction.
- This sort of ischemia represents a gradient phenomenon, not a sharply localized event. It begins in inner layers but may extend outward if severe.

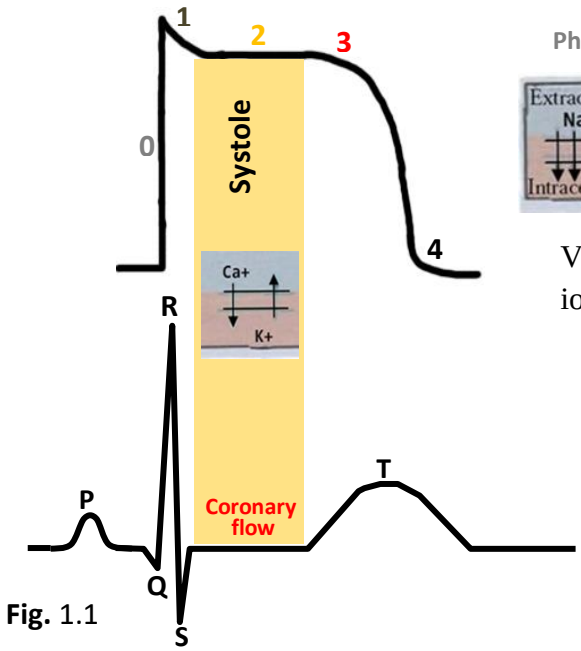
2. ST segment in a normal person

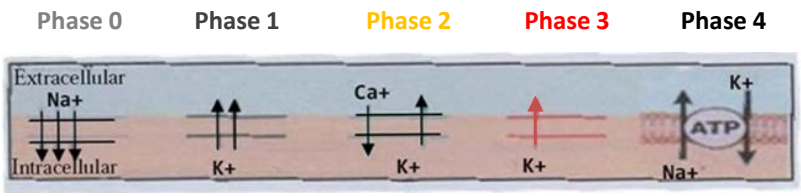
Normally , ST segment is an isoelectric line on the ECG between the end of S wave (the J point) and the beginning of the T wave .

It includes the following steps :

- The inward Ca^{2+} flow and outward K^{+} flow currents are approximately equal but with reverse polarity on either side of the cardiac membrane during phase 2.
- The exchange in between Ca^{2+} and K^{+} brings 'excitation-contraction coupling'occuring during the systolic phase.

The entire concept is illustrated with the following sketch :





Ventricular action potential has 5 phases (0–4) driven by ion channel changes.

Fig. 1.1

3. Pathophysiological basis of subendocardial ischemia

Subendocardial ischemia arises because the subendocardium is the most vulnerable segment of the myocardial wall. It has the highest metabolic demand and experiences significantly higher compressive pressures during systole, attenuating its blood supply. Coronary filling occurs almost exclusively during diastole—most easily outstripped by oxygen demand, especially under conditions of reduced perfusion pressure, tachycardia, or increased systolic wall stress.

Let us peep through its details :

❒ Anatomic and hemodynamic vulnerability

- The subendocardium is supplied by the most distal branches of the coronary arteries , which are having the lowest perfusion pressure.
- Under coronary narrowing or reduced aortic pressure , flow is preferentially redistributed to the zone of subepicardium, further depriving the subendocardium from its blood supply.

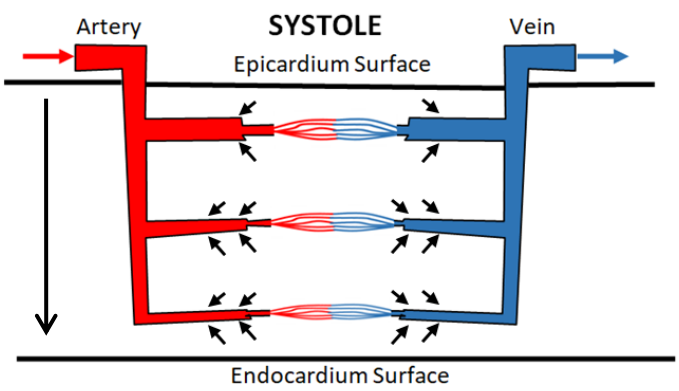


Fig. 1.2

To further elaborate with the following scenarios , one should know how the subendocardium reaches its ischemic threshold. These are key settings that favour subendocardial involvement.

❒ **Supply–demand disparity in the subendocardium**

Metabolic demand is on the higher scale here because this layer contracts against the highest intraventricular pressure during systole and short-axis myocardial fibers in the subendocardium also contribute towards this.

❒ **Transmural flow heterogeneity and vascular compliance**

Myocardial contraction implements stronger extravascular loading on subendocardial vessels, lowering the transmural pressure difference across these vessels compared with those of epicardial ones.

❒ **Microvascular and endothelial dysfunction**

Microvascular dysfunction (as occuring in hypertensive LVH, diabetes or cardiomyopathy) compromises the subendocardium's ability to dilate and recruit flow when demand rises

In nutshell Pathophysiologic Basis :

Subendocardial ischemia occurs when coronary blood flow is reduced but not completely occluded OR oxygen demand exceeds supply leading to ischemia confined to inner myocardial layers.

The subendocardium is the first to suffer and last to recover when coronary perfusion declines.

4. Classification

Classification based on mechanism :

Demand ischemia (Type-2 physiology-dependent mechanism)	**Supply Ischemia (Coronary insufficiency)**
• Oxygen demand exceeds supply • Causes : Tachycardia Hypertension Severe anemia Sepsis • Coronary arteries may be: normal or mildly diseased	• Reduced coronary flow • Causes : Coronary artery stenosis Plaque rupture (non-occlusive) Hypotension • Usually abnormal coronary artery

NB : Mixed mechanism (both reduced supply plus increased demand) very common in real clinical practice.

Classification based on clinical setting :

A. Acute Subendocardial Ischemia
   - Unstable angina / NSTEMI

B. Chronic Subendocardial Ischemia
   - Stable angina
   - Effort-induced ischemia

Rather than viewing acute and chronic forms of subendocardial ischemia as separate entities , they represent different clinical expressions of the same underlying substrate , modulated by the severity, duration and the dynamic nature of coronary flow. In essence, the subendocardium tells the story of ischemia in two voices—a sudden cry in the acute setting, and a predictable whisper in the chronic state—yet both arise from the same underlying imbalance.

5. ECG changes during subendocardial ischemia

➢ In the setting of subendocardial ischemia, reduced perfusion of the subendocardium alters the transmembrane action potential gradient, generating current of injury that is directed away from the overlying recording leads. This results in the characteristic ST-segment depression on the surface electrocardiogram.

> Subendocardial ischemia is the earliest and most sensitive manifestation of coronary insufficiency, where the vulnerable inner myocardium reflects its distress through subtle yet significant changes in ventricular repolarization.

➢ **Detailing :** The current of injury is produced at the level of susceptible subendocardium , the reasons already explained earlier. In the setting of subendocardial ischemia , this is the segment which suffers most as a diffuse involvement during phase 2 of ventricular action potential , so there is the maximum negative charges over the subendocardial zone rather than over the subepicardium.
Hence , the current flows from subepicardium towards subendocardium. (positive pole → negative pole) , i.e. away from the overlying recording leads.

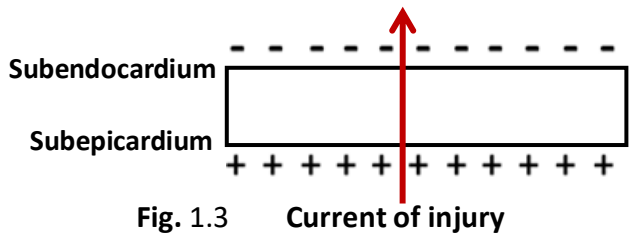
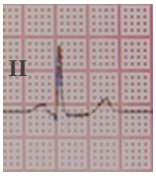


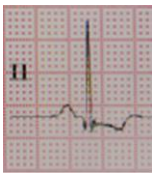
Fig. 1.3 **Current of injury**

➢ This subendocardial ischemia is detected on ECG as diffuse ST segment depression , (often in inferior leads II , III , aVF and V3-V6) and sometimes reciprocal ST elevation in lead aVR , reflecting ST-vector as directed towards this lead rather than to a specific wall. Its prompt recognition is critical, as it can progress to transmural involvement.

➢ The resultant ST depression in subendocardial ischemia is basically characterized by a horizontal / downsloping ST segment.



Horizontal ST depression
with a sharp angle in between ST segment and T wave. This is the earliest expression of ST segment in myocardial ischemia.



Downsloping ST segment depression
The ST segment depressed below the isoelectric line at its origin and then it slops further downwards. This indicates severe coronary insufficiency.

➢ Morphometry of ST depression

- ☐ **Horizontal or downsloping ST segment depression ≥ 0.5 mm at the J-point ≥ 2 contiguous lead points towards myocardial ischemia , usually of subendocardial origin.**
- ☐ ST depression ≥ 1 mm is rather more specific having a worse prognosis.
- ☐ ST depression ≥ 2 mm in ≥ 3 leads is usually associated with a higher possibility of Non-ST elevation acute coronary syndrome. ST depressions in NSTEMI are frequently accompanied by T inversions or flat T waves , but the primary ECG finding is the ST segment depression.

NB : Upsloping ST segment (non-specific) is rarely caused by ischemia. It is usually physiological in nature , seen in normal persons usually during physical exercise and resolves rapidly once the exercise is stopped.

➢ **Associated T-wave inversion associated with subendocardial ischemia :**

T-wave inversion may be considered to be evidence of myocardial ischemia if :

- At least 1 mm deep present in ≥ 2 continuous leads that have dominant R waves (R/S ratio > 1)
- Dynamic — not present on old ECG or changing over time

NB. T wave inversion is only significant if seen in leads with upright QRS complexes (dominant R waves). T wave inversion is a normal variant in leads III, aVR and V1.

➢ If one sees focal , non-diffuse ST depression , it should raise the suspicion of ST depression brought by associated secondary factors.

It become essential here to discriminate in between primary (ischemia) ST depression from that of secondary ST depression.

Primary ST depression	**Secondary ST depression**
• Causal factor : coronary insufficiency • Features Horizontal / downsloping Dynamic changes Associated symptom	• Causal factor : Abnormal depolarization examples: LVH , Bundle branch block , Ventricular pacing • Features Appropriate discordance Fixed pattern No dynamic evolution

- Dynamic changes are the strongest clue for subendocardial ischemia , expressed as ST depression that appears , evolves and resolves.
- Discordant ST-T changes are those occurring in the opposite direction to the main QRS complex having abnormal morphology (abnormal depolarization).

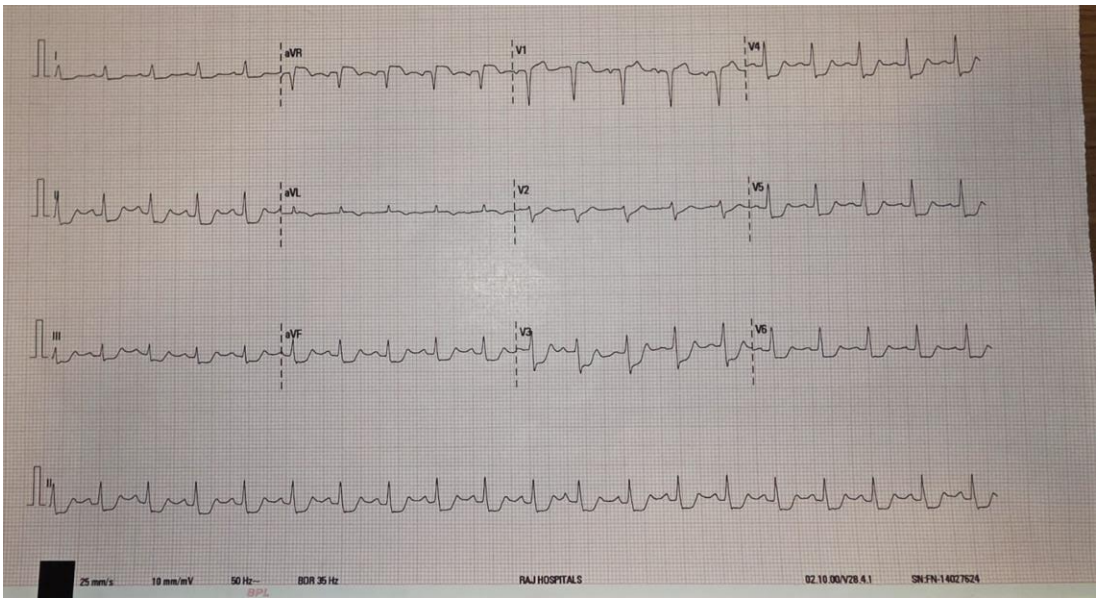
NB : While diffuse ST depression is more typical of global subendocardial ischemia, localized ST depression should be carefully evaluated, as it may represent either secondary repolarization changes or regional ischemia depending on the clinical and electrocardiographic context.

➢ For subendocardial-type ischemia, ECG is very useful for diagnosing ischemia and risk-stratifying (e.g., in NSTE-ACS), but localization to a specific coronary artery usually requires additional data (symptom pattern, echo wall-motion abnormalities, or coronary angiography).

6. Case study with accompanying ECG

56 yrs old male, diabetic with acute chest discomfort over the background of long standing low effort exertional dyspnoea.

This case illustrates how long standing coronary artery disease (TVD) may be associated with diffuse ST depression + aVR elevation (supply dependent ischemia).



Source : Dr. Varun Kumar, DM (Card) , Senior Consultant in Cardiology , Ranchi (Jharkhand)

Findings :

- Regular sinus rhythm with Heart rate = 115 bpm
- Diffuse ST depression with ST elevation in aVR suggesting either critical LMCA disease , ostio-proximal LAD lesion or TVD.

Discussion :

This diabetic patient is having the history of acute chest discomfort but not typical pain, though gives the h/o low effort exertional dyspnoea for long time but never evaluated for that.... his coronary angiography revealed extensive TVD with chronically totally occluded

LAD & LCX with critical lesion in RCA , suggesting patient might had coronary insufficiency previously also but not appreciated by the patient. Patients with long standing diabetes many a times do not feel chest pain but may have chest discomfort, dyspnoea, excessive fatigue or fainting.... what is called as anginal equivalents....

Early evaluation in a diabetic patient for CAD is a 'must' , since at later stages he may develop extensive CAD as this patient had.... this patient was referred for CABG.

7. Take-Home Message

- ☐ Subendocardial ischemia is a condition in which myocardial oxygen deprivation selectively involves the subendocardial layers of the ventricular wall due to inadequate coronary perfusion, without full-thickness involvement.
- ☐ In such setting of coronary insufficiency reduced perfusion of the subendcardium generates current of injury which is directed away from the recording electrodes inscribing ST depression on the ECG. Thus , ST depression is the electrocardiographic signature of subendocardial ischemia.
- ☐ Decision making steps :
    - Normal QRS + horizontal /downsloping ST depression (dynamic in nature) = Subendcoardial ischemia
    - Abnormal QRS + discordant ST = Secondary changes
    - Diffuse ST depression + aVR elevation = Severe global ischemia (LMCA lesion , triple vessel disease , at times LAD involvement).
- ☐ For subendocardial-type ischemia, ECG is very useful for diagnosing ischemia and risk-stratifying (e.g., in NSTE-ACS), but localization to a specific coronary artery usually requires additional data (symptom pattern, echo wall-motion abnormalities, or coronary angiography).

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MYOCARDIAL BRIDGING : UNDERSTANDING ITS CONSEQUENCES ON THE ECG

ECG

MYOCARDIAL BRIDGING :
UNDERSTANDING ITS CONSEQUENCES ON THE ECG

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OUTLINE

Introduction
A myocardial bridge is a congenital coronary anomaly in which a segment of an epicardial coronary artery traverses intramyocardially beneath a band of cardiac muscle.

Pathophysiological basis : Haemodynamic consequences
Greater systolic compression + Less available diastolic time = reduced coronary perfusion → This disturbs the hemodynamic harmony of coronary circulation.

Anatomical Consideration
Approximately 70–90% of clinically significant bridges involve left Anterior Descending Artery particularly the mid-LAD segment.

ECG manifestations
- ☐ The ECG is normal at rest.
- ☐ Dynamic transient ST segment depression
- ☐ Other repolarization abnormalities: T-wave inversion /T-wave flattening.

Discussion with differential diagnosis of Myocardial Bridging
Primarily aims at differentiating in between obstructive and non-obstructive coronary artery involvement (Main D/D are myocardial bridging , microvascular angina (Syndrome X), coronary vasospasm , and obstructive CAD.

An interesting case study

Take-Home Message

References

Myocardial Bridging : Understanding its Consequences on the ECG

A Narrative Review

© **DR. D.P. KHAITAN**
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"Rather than focusing on the obstacle in your path, focus on the bridge over the obstacle." – a famous metaphor by Mary Lou Retton.

Possibly, this statement highlights the significance of overcoming an obstacle by building a bridge across it. Interestingly, the same metaphor finds a unique parallel in myocardial bridging—a condition in which an epicardial coronary artery takes a tunneled course beneath the myocardium.

- **A myocardial bridge is a congenital coronary anomaly in which a segment of an epicardial coronary artery traverses intramyocardially beneath a band of cardiac muscle.**
- **Unlike fixed atherosclerotic narrowing, myocardial bridging represents a dynamic coronary phenomenon, with transient systolic compression of the tunneled arterial segment.**

This myocardial bridging may appear anatomically innocuous, yet it can become physiologically significant under conditions of stress, tachycardia, or increased myocardial contractility, thereby concerning both the patient and the physician. In such circumstances, the ECG becomes a messenger, revealing the silent dialogue between the bridged coronary artery and the overlying myocardium.

1. Introduction (Keypoints)

- **Myocardial bridging** is a congenital anomaly where a segment of an epicardial coronary artery dives into the heart muscle (myocardium), travels intramurally as a 'tunneled artery', and re-emerges onto the epicardial surface. This represents a dynamic coronary compression in which anatomy and physiology converge to produce transient myocardial ischmemia.

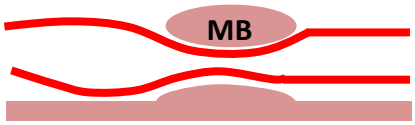


Fig 1.1

Myocardial bridging **(MB)** compressing intramyocardial segment of coronary artery

- Recognition of this entity is essential in patients presenting with unexplained ischemic symptoms or dynamic ECG abnormalities. Under such situation no fixed coronary narrowing is observed on coronary angiography. Such cases alert the concerned clinician towards the consideration of myocardial bridging. This scenario represents hierarchy of recurrent ischemic episodes.
- Commonly , the mid LAD segment is the preferred site of such myocardial bridging.

- One should always suspect myocardial bridging when ischemic symptoms or dynamic ECG changes appear despite the absence of significant fixed coronary artery disease (CAD), particularly when the LAD is involved and symptoms are brought on the surface by exercise or tachycardia. It should be kept in mind that the ECG is frequently normal at rest in patient with myocardial bridging. The hallmark of this condition is not fixed obstruction, but rather a dynamic compression. Recognition of this dynamic behaviour often provides an important clue to the diagnosis of myocardial bridging.
- This article focuses on "Myocardial Bridging : Understanding its Consequences on the ECG". It is a very relevant topic in cardiology. Every possible effort is made here to lay down a clear cut concept – why a normal epicardial coroanary artery becomes a victim when it takes an intramyocardial course by passing through it. This article also deals with the other relevant facts associated with this bridging.

2. Pathophysiological basis : Haemodynamic consequences

The hallmark of myocardial bridging is dynamic systolic compression of the tunneled coronary segment.

A. During ventricular systole:
   - The overlying myocardial fibers contract,
   - The arterial lumen narrows,
   - Coronary flow transiently decreases.

B. Although coronary perfusion predominantly occurs during diastole, compression may persist into early diastole, especially during tachycardia and exercise. This results in:
   - Reduced coronary flow reserve,
   - Impaired myocardial perfusion,
   - Subendocardial ischemia.

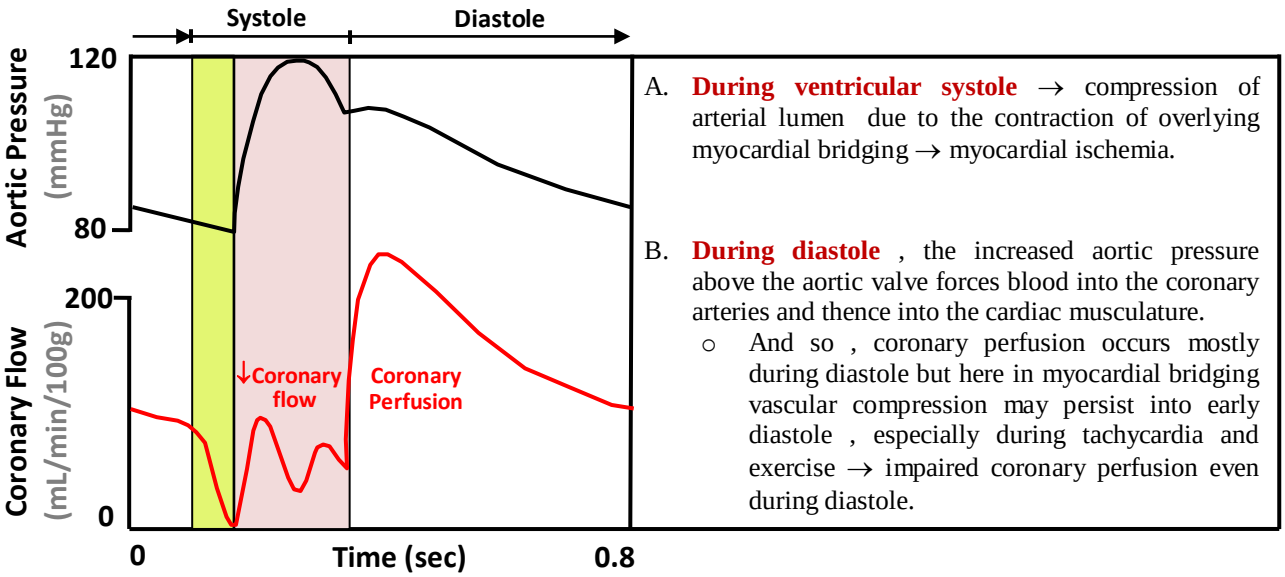


Fig 1.2

In nutshell to say , since coronary perfusion occurs mainly during diastole →
Less diastolic time + greater systolic compression = reduced coronary perfusion →
This disturbs the hemodynamic harmony of coronary circulation.

The hemodynamic significance of myocardial bridging often becomes apparent only during exertion, when enhanced myocardial contractility and shortened diastolic filling time synergistically increase compression of the tunneled coronary segment.
Thus, myocardial bridging should be regarded as a dynamic physiological lesion rather than a fixed anatomical obstruction.

3. Anatomical consideration

- ❒ Approximately 70–90% of clinically significant bridges involve left Anterior Descending Artery particularly the mid-LAD segment.
- ❒ The reasoning behind the mid LAD involvement :
  - Mid-LAD traverses through the anterior interventricular groove, where myocardial fibres may create a compressive loop over the underneath arterial segment.
  - This arterial territory often lies deeper within the myocardium than many other epicardial coronary segments
  - Further to say , the LAD is intimately related to the powerful contractile mass of the left ventricle, making it particularly susceptible to systolic compression when tunneled.
- ❒ The most commonly involved vessel is the mid-segment of the left anterior descending (mid-LAD) coronary artery. Less frequently, diagonal branches, ramus intermedius, circumflex artery, or right coronary artery may be involved.
  It seems essential here to say some words about ramus intermedius. This artery is an anatomical variant situated at where the LMCA divides into three branches instead of the usual two, seen in about 15% to 30% of people. It typically supplies blood to the high lateral wall of the left ventricle.
- ❒ Length of the Bridge may range from a few millimeter to several centimeters. Generally longer and deeper bridges are more likely to produce hemodynamic consequences.

A. Less Lengthened Myocardial Bridging

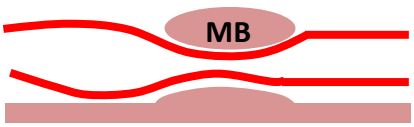


Fig 1.3

B. More Lengthened Myocardial Bridging with deeper encroachment

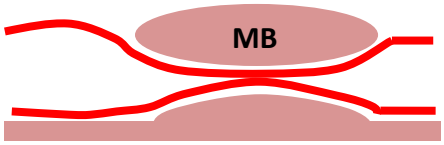


Fig 1.4

4. ECG Manifestations

Myocardial bridging represents a dynamic coronary compression entity in which anatomy and physiology converge to produce transient myocardial ischemia , reflected through the language of the ECG.

- ☐ The ECG is normal at rest.
- ☐ When ischemia develops, the following abnormalities may be witnessed :
  - The commonest ECG manifestation is transient ST-segment depression, particularly during exercise or Treadmill test (TMT). This reflects subendocardial ischemia caused by compressed coronary perfusion.
- ☐ Other repolarization abnormalities may be found in addition : T-wave inversion / T-wave flattening.
- ☐ These changes are dynamic in appearance and disappearance of these changes after the event of exercise or stress support a functional rather than fixed coronary lesion. ST depression is usually either horizontal or downsloping. These changes are often most prominent in anterior precordial leads due to the frequent involvement of the LAD.
- ☐ Arrhythmogenic ECG Manifestations
  Some patients may demonstrate:
  - Ventricular premature complexes,
  - Supraventricular tachyarrhythmias,
  - Ventricular tachycardia.

  These likely result from ischemia-induced electrical instability.

In nutshell to say , significant myocardial bridging may produce distal subendocardial ischemia, particularly during exertion. The ECG commonly demonstrates regional ischemic changes, although extensive ischemia may occasionally manifest as widespread ST-segment depression with reciprocal ST elevation in aVR, mimicking diffuse subendocardial ischemia.

5. Discussion with differential diagnosis of Myocardial Bridging

The differential diagnosis of myocardial bridging depends upon whether it is obstructive or non-obstructive coronary artery lesion.
The differential diagnosis of myocardial bridging mainly includes microvascular angina, coronary vasospasm, and obstructive coronary artery disease. Demonstration of a tunneled coronary segment with dynamic systolic compression favours myocardial bridging, whereas normal epicardial coronaries in the presence of ischemic symptoms and stress-induced ischemia suggest coronary microvascular dysfunction.
While formulating the differential diagnosis of myocardial bridging, one should think in a pathophysiology-based hierarchy. Myocardial bridging itself represents a dynamic coronary compression phenomenon, whereas microvascular angina results from impaired vasodilatory function of the subendocardial microcirculation, and coronary spasm reflects transient vasoconstriction of an epicardial coronary artery.

Differential diagnosis :

Clinical entity (non-obstructive)	Diagnostic consideration
⇒ **Myocardial Bridging**	• Usually younger patients • Ischemic symptoms are precipitated by the events brought by sympathetic activity , like stress or exercise. • The findings on ECG are dynamic in nature , so they disappear at rest. • Angiography demonstrates systolic narrowing of the coronary artery. This is the hallmark of angiographic finding , known as the 'milking effect' on the coronary artery.
⇒ **Microvascular Angina (Syndrome X)**	• Often middle-aged/postmenopausal women • Also an exertional or emotion stress-related ,so reversible as well • Coronary angiography normal • Abnormal coronary flow reserve testing may aid in the diagnosis.
⇒ **Coronary vasospasm (Variant angina)**	• Episodic chest pain often at rest • Early morning occurrence • ECG changes : Transient ST elevation
⇒ **Hypertrophic Cardiomyopathy** Interestingly, myocardial bridging is more frequently encountered in patients with hypertrophic cardiomyopathy.	• Left ventricular hypertrophy on ECG, with 'dagger-like' Q wave and deep T-wave inversions. • Family history. • Echocardiographic abnormalities
⇒ **Supply-Demand Ischemia** Severe anemia,Tachyarrhythmias, Thyrotoxicosis, Sepsis, Hypotension.	• No primary coronary abnormality • Associated clinical findings are present on the clincal examination of the patient

Coronary flow reserved testing in microvascular angina (CFR testing) : A Concept

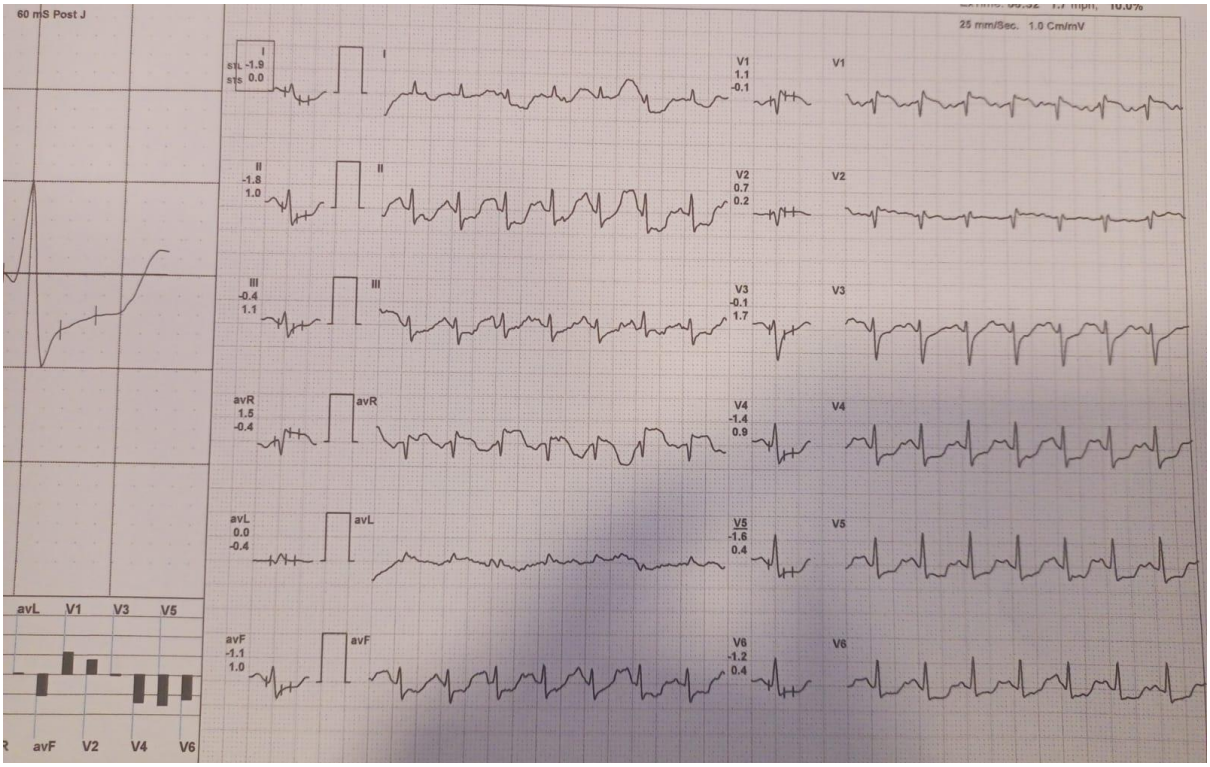
It diagnoses coronary microvascular dysfunction by relieving blunted , inadequate blood flow. A healthy heart can increase its blood flow 3 to 4 times over the baseline. In microvascular angina its reserve is impaired , and the ratio typically drops below 2.0 or 2.5 due to poor micro circulation (please refer to the text for knowing the details of procedure) .

Clinical entity (Obstructive)	Diagnostic consideration
⇒ **Obstructive CAD**	• Multiple risk factors • Fixed exertional angina • Significant stenosis on angiography. • Typical ischemic territory involvement corresponding to a definite coronary domain • Troponin rise and/or fall.

6. An interesting case study

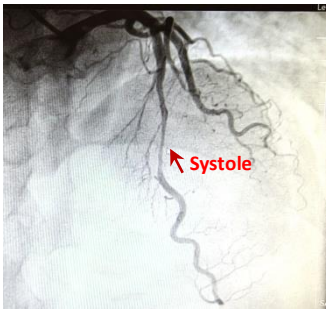
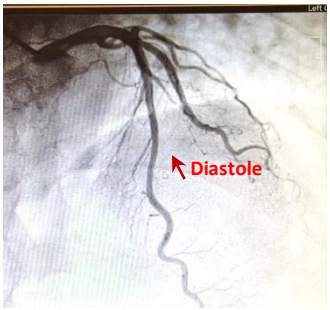
47 years old female presented with exertional chest pain and dyspnea with normal basal ECG.

She was subjected to TMT testing. The concerned tracings at peak exercise show diffuse ST depression with reciprocal ST elevation > 1 mm in aVR.



Source : Dr. Varun kumar , Senior Consultant Cardiologist , DM Card , Sante Vita Hospital , Ranchi (Jharkhand) (reproduced with his kind permission)

The patient further underwent coronary angiography (CAG). The angiogram demonstrated a myocardial bridge located within the distal LAD; the substantial length of this bridging segment resulted in greater systolic compression. Her angiogram shows the following findings :

During systole	During diastole
	
• 'Milking effect' induced by myocardial bridging , where the artery is visibly compressed (squeezed) during the systole.	• The concerned coronary artery opens and returns almost to its normal diameter during the diastole.

Discussion :

- A more lengthened LAD myocardial bridge may not only produce marked systolic compression but may also delay complete luminal recovery during early diastole. This combination can reduce coronary flow reserve and potentially provoke subendocardial ischemia.
- The following factors are acting simultaneously here :
    - Increased myocardial contractility → stronger systolic squeezing of the tunneled segment.
    - Delayed vessel relaxation in early diastole →reduced effective coronary perfusion time.

  The total impact is reduced coronary perfusion which has disturbed hemodynamic harmony of coronary circulation.
- Female sex , exertional symptoms , normal rest ECG with diffuse ischemic changes during TMT – all these point towards concomitant coronary microvascular dysfunction in addition to myocardial bridging.
- In nutshell to say , A long myocardial bridge contributed to TMT-induced ischemia, while concurrent coronary microvascular dysfunction may have further amplified the total ischemic burden. Consequently, the resulting ST-segment changes on the ECG are more extensive than would be expected from the myocardial bridge alone.

7. Take Home Message

- ☐ Myocardial bridging represents an intramyocardial course of a normally epicardial coronary artery, most commonly involving the LAD. Although frequently asymptomatic, significant bridges may produce dynamic coronary compression leading to ischemia, arrhythmias, and characteristic ECG manifestations.
- ☐ The clinical significance of myocardial bridging frequently emerges during periods of enhanced sympathetic activity, which simultaneously increases heart rate, myocardial contractility, oxygen demand, and systolic compression of the tunneled coronary segment.
  (Enhanced sympathetic drive converts an anatomically bridged artery into a physiologically significant obstruction).
- ☐ The ECG often serves as the first electrophysiological witness to the hemodynamic consequences of myocardial bridging. Recognition of this dynamic coronary phenomenon enables accurate diagnosis and appropriate management while preventing misclassification as fixed obstructive coronary artery disease.
- ☐ When ischemia is present without a fixed coronary obstruction, the clinician must think beyond the angiogram—toward myocardial bridging, coronary microcirculation dysfunction , and vasospasm , etc.

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