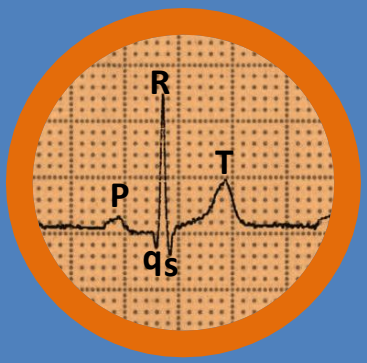

ECG Review : Occlusion MI - A Paradigm Shift

(For Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

MD (MEDICINE) FCGP (IND) FIAMS (MEDICINE) FICP FICCMD FIACM

Occlusion MI in disguise
A hidden thread with surprise …..

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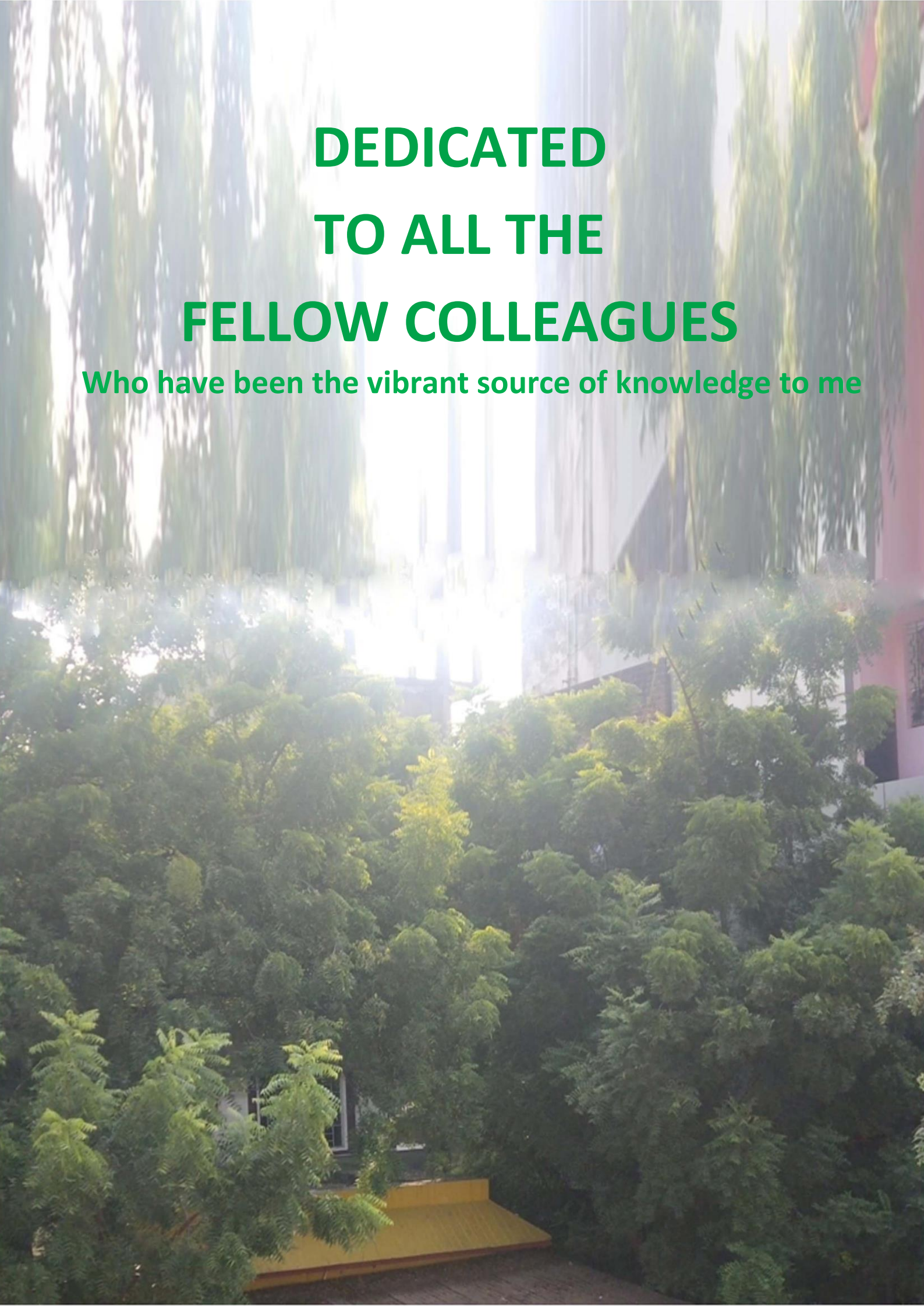
Occlusion MI in disguise
A hidden thread with surprise …..

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 this ECG review contains some of my wirte-up on Occlusion MI. 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 ALL THE
FELLOW COLLEAGUES

Who have been the vibrant source of knowledge to me

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ECG

LET US WAKE UP TO THIS NEW POSSIBILITY - OCCLUSION MI (OMI) IN DISGUISE : A PARADIGM SHIFT

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OUTLINE

Let us wake up to this new possibility – Occlusion MI (OMI) in disguise : A paradigm shift

A Narrative Review

© **DR. D.P. KHAITAN**
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In life , both knowing and experiencing are equally important – one supplements the other. One may take any number of wrong turnings ; but by keeping eyes open one can not move far beyond – one revises within oneself to adopt the correct paradigm to recognize such a wrong turning.
This wrong turning may happen with the diagnosis of certain occlusion myocardial infarction patterns on ECG which don't show the classical pattern of STEMI.

- ☐ **These are actually 'occlusion MI' in disguise. Missing of such alarming pathological entities means depriving the patient from the life saving procedure like PCI.**
- ☐ **Unfortunately , approximately half of high-risk patients with occlusion myocardial infarction fail to meet STEMI criteria.**

Clinicians apply their knowledge and experience to their best to address such occlusion MI on ECG.

1. Introduction (keynotes)

- Electrocardiogram (ECG) is considered as one of the most useful diagnostic tools for the identification of ST-segment elevation myocardial infarction. Traditionally STEMI is diagnosed as a group of symptoms consistent with acute coronary syndrome in the presence of new ST-segment elevation at J-point in at least two anatomical continuous leads of $\geq$ 2 mm in a male or $\geq$ 1.5 mm in a female over leads V2-V3 and/or at least 1 mm in other continuous leads or limb leads , in the absence of bundle branch block , LVH , or other non acute MI ST-segment elevation situations.
- There exists some STEMI equivalent patterns but in disguise on ECG – a challenging situation to be diagnosed , otherwise a significant part of the left ventricle may be jeopardized with a poor outcome.
- The following pitfalls have been observed by restricting the term to 'STEMI' :
  - The chance of missing the diagnosis of acute coronary occlusion remains upto 30% .
  - Accordingly , there is a lapse of time in salvaging the involved injured tissue by reperfusion therapy and so making this situation further grave by increased morbidity and mortality.
  - Sometimes benign ST elevation on ECG is wrongly diagnosed as STEMI with unwarranted catheterization , exposing the concerned patients to the associated risks of coronary dissection $\pm$ perforation , arterial punctures with bleeding and contrast associated nephropathy.

- **In 2018 , Meyers , Weingart and Smith** put a new concept of 'occlusion myocardial infarction' (OMI) in the place of STEMI equivalent – a more reliable paradigm for detecting acute coronary occlusion.
- **Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation , leading to imminent infarction of the affected myocardium - to be diagnosed in the absence of traditional ST elevation on ECG remains a 'Herculean task'.**
- With much unfamiliarity with this new occlusion MI one may take wrong turnings and so it becomes essential to have its conceptual knowledge so that one may not miss its diagnosis.

2. A basic concept while interpreting 'occlusion myocardial infarction'

The following pertinent points are to be considered in this context :

- **Prediction of the anatomical site of occlusion MI** : Most of the cases of occlusion MI occur in the proximal territory of LAD. Therefore , the site of occlusion can be inferred from the pattern of ST changes in leads corresponding to the two most proximal branches of the LAD : the **first septal branch** (S1) and the **first diagonal branch** (D1)
  - S1 being the first and the largest septal branch supplies the basal part of the interventricular septum.
  - D1-LAD supplies the high lateral region of the heart (leads I and aVL)
- Repolarizing abnormalities (ST segment ± T-wave abnormalities) occur with this new occlusion MI.

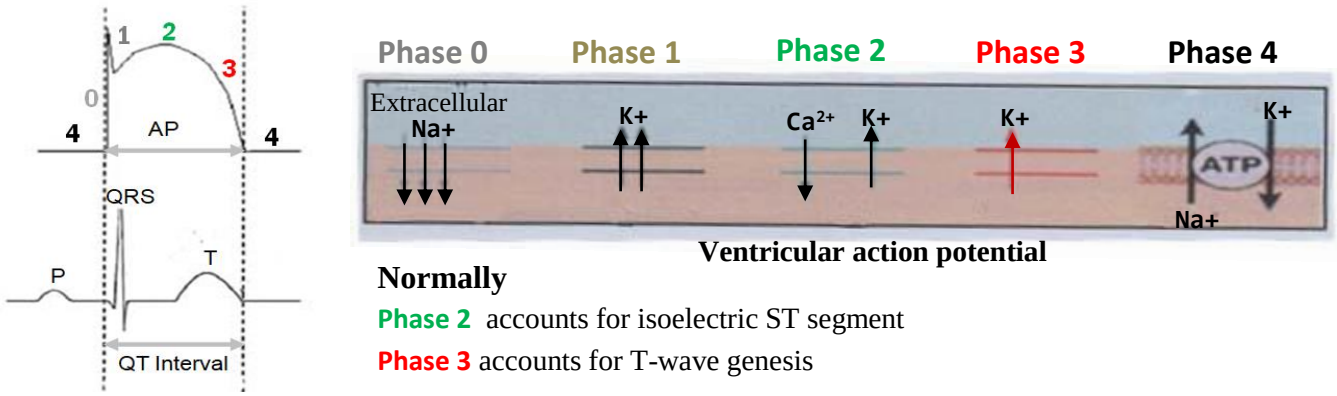


Fig 1.1

- **ST segment elevation** : Normally the inflow of Ca^{2+} with simultaneous outflow of K^{+} during **phase 2** accounts for isoelectric ST segment (plateau phase).
  - The ST segment elevation occurs with epicardial injury during occlusion MI ; since the current of injury so produced is directed towards the concerned exploring electrodes.

- **ST segment depression** : equally is the importance of understanding the concept of ST depression in this context.
  - If the ST segment depression is localized and limited to certain leads , especially opposite to leads with clear ST elevation , it is likely reciprocal – a phenomenon mostly observed in occlusion MI.
  - If ST depression is diffuse , horizontal/downslowing , think of associated subendocardial ischemia in the presence of concomitant ST elevation (as seen in **Aslanger pattern OMI** , discussed in page 7) . Here the current of injury is subendocardial and so its flow is away from the concerned exploring electrodes → ST depression.
- **Predictive value of ST segment elevation in aVR vs V1** : Since in proximal LAD occlusion , as with precordial swirl pattern , ST elevation is seen in both the leads V1 and aVR with reciprocal ST depression in leads V5 and V6 , it becomes essential here to get it excluded from that of proximal LAD / LMCA occlusion or severe 3VD.
  - ST segment elevation in aVR greater than in V1 indicates proximal LAD / LMCA occlusion or severe 3VD and it is in association with widespread ST depression.
  - ST elevation in V1 > aVR favours proximal LAD occlusion with anterior transmural injury, as seen in anterior STEMI or Precordial Swirl Sign (PSS) , distinguished by LAD occlusion - ST elevation extending across V2–V4, whereas in PSS, it remains isolated to V1 and aVR with reciprocal ST depression in lateral leads V5-V6.

- **T-wave** : **Normally** , there is a outflow of K+ ions from intracellular compartment to extracellular compartment during the phase 3 , interrupting the isoelectric ST segment due to the newly created potential difference in between intracelluar and extracellular compartments – the resultant situation is the genesis of upright T-wave.
  In certain occlusion MI , there is alteration in T-wave morphology in consistent with proximal LAD occlusion.
  - Hyperacute T-wave (HATW) : Taller and broad-based , often generally more symmetrical than the normal T-wave.
  - Peaked tall T-wave in ‘de Winter pattern’ (dWp)
  - Transmural ischemia-reperfusion injury with change in T-morphology in Wellens’ syndrome
    - **Type A Wellens’ syndrome** (Biphasic T-wave : the initial positivity with terminal negativity)
    - **Type B Wellens’ syndrome** (Deeply inverted and symmetrically T-wave)

3. A working classification of 'occlusion MI' based on ECG changes

☐ **Alteration in T-wave**
- Hyperacute T-wave (HATW)
- Peaked tall T-wave (de Winter pattern)
- T-wave morphology in Wellens' syndrome

☐ **Alteration in ST segment (Atypical pattern)**
- Precordial Swirl Sign
- South African Flag Sign
- Posterior OMI
- Aslanger pattern
- Northern OMI

☐ **Miscellaneous group**
- Modified Sgarbossa-Smith Criteria (occlusion MI in the presence of LBBB)
- Terminal QRS distortion
- New-onset RBBB and LAFB

4. Electrocardiographic changes – a spy-key to open the door of 'occlusion MI'

☐ Alteration in T-wave

👉 **Hyperacute T-wave (HATW)** : This is postulated that there is hyperpermeability related K+ ions transfer from intracellular compartment to extracellular compartment over the infarct area resulting in hyperacute T wave. Hyperacute T-wave with ≥ in two contiguous leads may be the first sign of a developing infarct, often preceding any STE. Hyperacute T-waves appear broad-based , often generally more symmetrical than the normal T-wave.

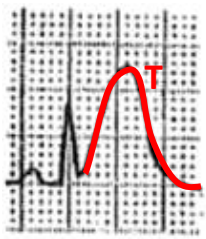


Fig 1.2

Dr Stephen W. Smith suggests that if T wave /QRS complex ratio in any V1-4 is greater than 0.36, it represents acute MI, not subacute or old.
(A new upright T-wave in V1>V6 is considered as loss of precordial T-wave balance especially in favour of coronary insufficiency).

👉 **Peaked tall T-wave in 'de Winter pattern' (dWp)**

A regional subendocardial ischemia with preservation of a small ring of subepicardial tissue with alteration in T-wave morphology (tight prox. LAD occlusion)

Peaked tall T-wave is the expression of the shorter time duration between subendocardial and subepicardial repolarization. There is a shorter distance in between the subendcoardial zone of ischemia and preserved peripheral rim of subepicardial healthy tissue with a positive voltage gradient between endo-epicardium.

- Tall prominent symmetrical T waves in the precordial leads
- Upsloping ST segment depression > 1mm at the J point in the precordial leads
- Absence of ST elevation in the precordial leads
- Reciprocal ST segment elevation (0.5-1mm) in aVR

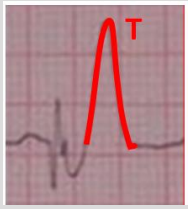


Fig 1.3

👉 **Transmural ischemia-reperfusion injury with change in T-morphology in Wellens' syndrome**

- A temporary but significant obstruction of the proximal LAD coronary artery , commonly caused by the rupture of an atherosclerosis plaque with the subsequent thrombolysis before complete myocardial infarct sets in.
- The next phase is transmural ischemia-reperfusion injury leading to myocardial oedema.

Biphasic T-wave (with initial postivity and terminal negativity) is followed by deeply inverted T-wave , mainly in chest leads V2 and V3.

Reasoning :

- Myocardial ischemia – reperfusion injury results in local myocardial oedema which can change the direction of ongoing repolarization current. Due to the delay in passing the current through this oedematous myocardium – the current flows uniformly through the opposite direction i.e. from subendocardium to subepicardium. This produces symmetrical deep T-wave inversion.

- In the initial stage of partial oedematous myocardium the first half of the wave with initial positivity and the terminal part with negativity.

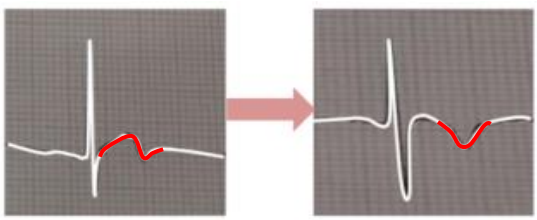


Fig 1.4

Biphasic T-wave (the initial positivity and terminal negativity)

Type A Wellens' Syndrome

Deeply inverted and symmetrical T-wave

Type B Wellens' Syndrome

☐ Alteration in ST-segment

👉 Precordial Swirl Sign

As per Smith and Meyers et al. the precordial swirl sign illustrates the following facts :

- ☐ This occlusion MI is the resultant of **septal ischemia** , including the corresponding anterior wall (LAD occlusion proximal to S1)
- ☐ This entity is recognized on ECG by ST elevation in leads V1 and aVR. There is associated reciprocal ST depression in leads V5 and V6.

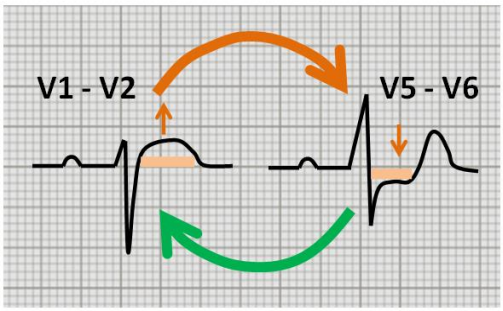


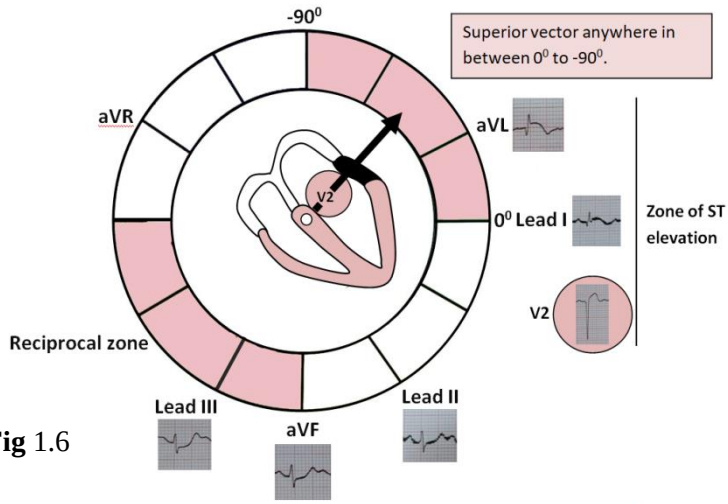
Fig 1.5

- ☐ Since precordial swirl sign is dynamic in nature , evolving hyperacute T-wave changes are also included therein :
  'ST-segment elevation and /or hyperacute T-waves in V1-V2 and reciprocal ST depression and /or T-wave inversion in V5-V6'.

👉 South African Flag Sign (High lateral MI)

The following points are to be considered to have a clear concept of high lateral MI on ECG :

- High lateral STEMI occurs usually due to occlusion of the first diagonal branch of LAD (LAD-D1) – involving the superior and lateral portion of left ventricle.



ST vector passing through leads I , aVL and V2
→ ST↑ in these leads

Fig 1.6

Reciprocal ST depression in inferior leads , most prominent in lead III

A model to understand ST vector in high lateral MI with its changes on ECG

- The ECG findings are plotted over 'South African Flag' as a very specific sign , hence the nomenclature.

Fig 1.7

ST elevation in leads I , aVL , V2 and ST depression in lead III are plotted over SOUTH AFRICAN FLAG , as illustrated therein.

👉 Posterior OMI

Reciprocal ECG changes (mirror image changes of MI of the posterior wall) : ST depression , tall R-waves and prominent positive T-waves (V1-3) should be considered a posterior OMI until proven otherwise.

Posterior OMI is a result of a critical reduction in blood flow to the dorsal, infra-atrial portion of the left ventricle , caused by the occlusion of PDA (posterior descending artery) – which can arise from either RCA (majority cases) or LCX , depending on coronary dominance. Posterolateral branch occlusion (LCX) with isolated posterior wall MI is very rare. (Posterior OMI is commonly observed due to acute occlusion of the RCA).

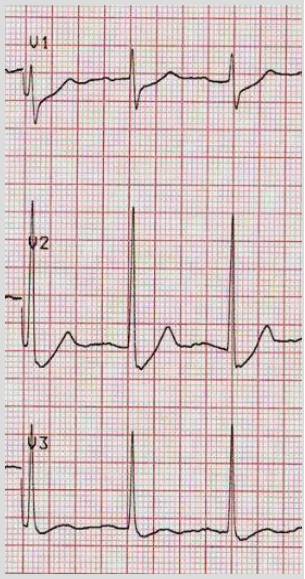


Fig 1.8

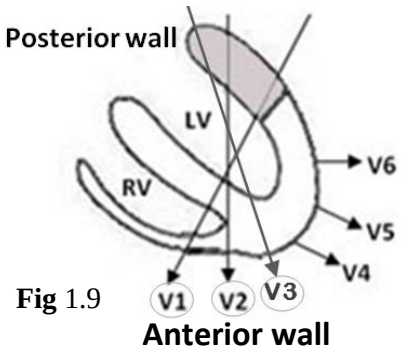


Fig 1.9

The precordial leads V1-3 oriented to the anterior wall , reflecting the inverse - mirror image changes of MI of the posterior wall

👉 Aslanger pattern

–ST elevation in lead III due to acute occlusion of RCA or LCX , associated with concurrent multivessel disease , predominantly due to LAD occlusion in addition.

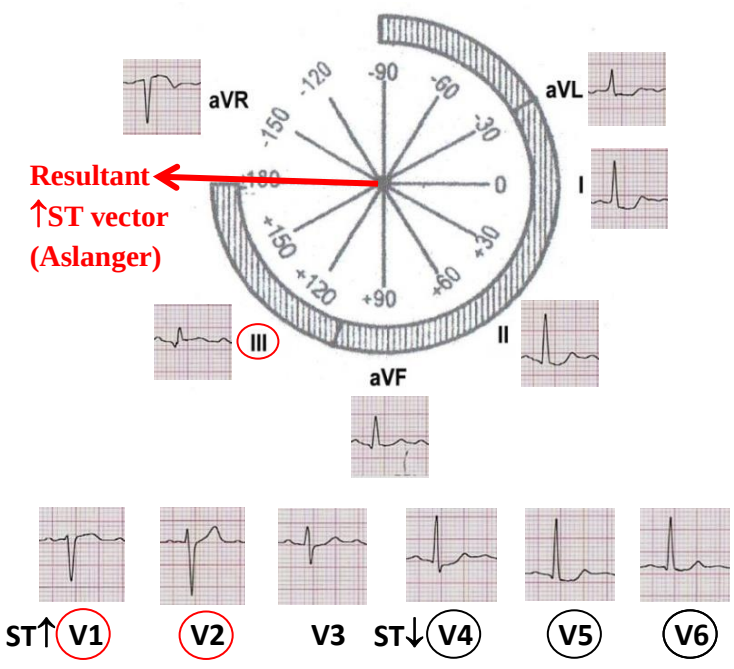


Fig 2.0

Essential :
Aslanger pattern on ECG

- ST↑ only in lead III as evidence of inferior MI
- ST ↑ in V1>V2
- Concomitant ST↓in any of V4-V6, with a positive/terminally positive T-wave

Resultant ST vector (Aslanger)

There are two ST vector forces – one from inferior occlusive MI and the other from subendocardial ischemia. The resultant average ST vector is directed rightwards causing ST elevation in lead III .

👉 **Northern OMI**

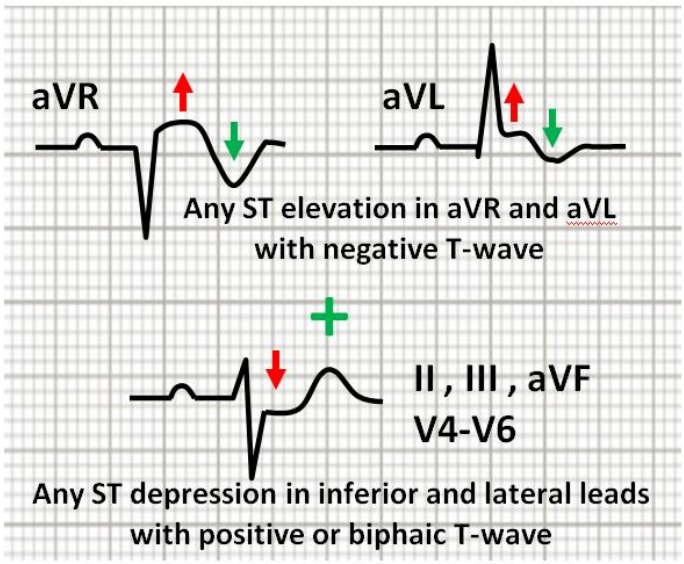


Fig 2.1

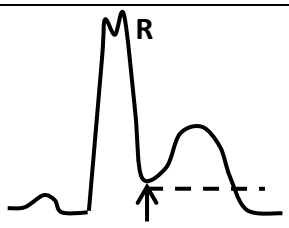
- Any STE in aVR and aVL with negative T-waves
- Any ST-depression in inferior and lateral precordial leads with positive or biphasic T-waves

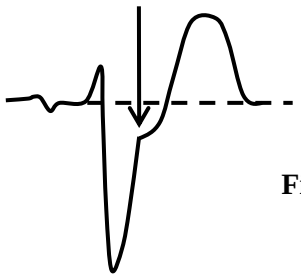
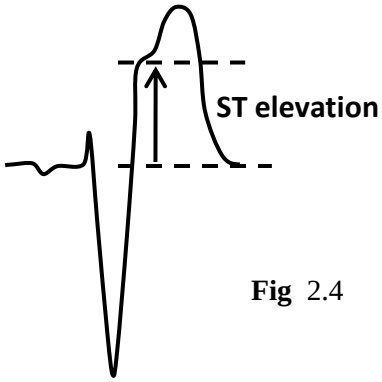
This occlusion MI as the name indicates is having northern vector orientation , more towards aVR with unique diagnostic ECG findings but prospective studies are needed to confirm its specificities and sensitivity.

☐ Miscellaneous group

👉 **Modified Sgarbossa-Smith criteria** (Occlusion MI in the presence of LBBB)

Occlusion MI if superimposed upon the pattern of left bundle branch block (LBBB) brings about some fundamental changes in the morphology of repolarization pattern. Thus, new pattern of repolarization sets in , as depicted with the following table (concordant ST elevation /depression or excessive discordant ST elevation).

Changes during repolarization	**Illustration by concerned sketches**
1. Concordant ST elevation ≥1mm in ≥ 1 lead (with a positive QRS complex)	 **Fig** 2.2 **Concordant ST elevation**

2. Concordant ST depression ≥1 mm in ≥ 1 lead of V1-V3	**Concordant ST depression**  **Fig** 2.3
3. Proportionally excessive discordant STE in ≥ 1 lead anywhere with ≥1 mm STE , as defined by ≥ 25% of the depth of the preceding S-wave. **(Smith Modified Sgarbossa Criteria)**	 **Fig** 2.4

NB : Smith-Modified Sgarbossa criteria improves diagnostic accuracy for occlusion MI in the presence of LBBB. The definition of excessive discordant is put in a specific manner , as discussed above in row 3.

✓This modified criteria is more specific to diagnose MI with LBBB , compared to original sgarbossa criteria (any one above mentioned criteria is suffice to diagnose MI in the presence of LBBB).

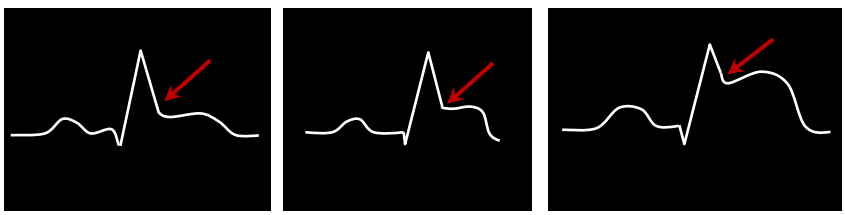
👉**Terminal QRS distortion**

Sometimes during the evolution of acute Anterior STEMI (ST Elevation Myocardial Infarction) on 12-lead ECG, one can witness the terminal S-wave beginning to rise above the baseline , seen usually with leads V2/V3. **This phenomenon of terminal QRS distortion is almost 100% specific for rapidly evolving STEMI in the territory of proximal anterior descending artery. Reasoning** Normally the terminal portion of the QRS reflects late basal-anteroseptal activation and the associated ischemia in proximal LAD distorts or extinguishes this phase → **Terminal QRS distortion**.

In association with the loss of S-wave and J-notch/slur, the remaining R-wave may take on as a qR configuration , as an imprint of ongoing myocardial insult with its uplifting with >50% above its baseline.

A concept of evolving 'terminal QRS distortion' , as illustrated below :

Fig 2.5



This loss of terminal S-wave means the normal late activation of the basal-anteroseptal wall is missing.

👉 **New-onset RBBB and LAFB**

It had been demonstrated that the proximal LAD septal perforators (S1,S2,S3) perfuse the right bundle branch and the anterior fascicle of the left bundle branch is also supplied by proximal LAD. This new bifascicular block may be the only pointer for OMI. Either of block may occur in isolation or as bifascicular block. Anterior wall involvement usually occurs with RBBB in occlusion MI.

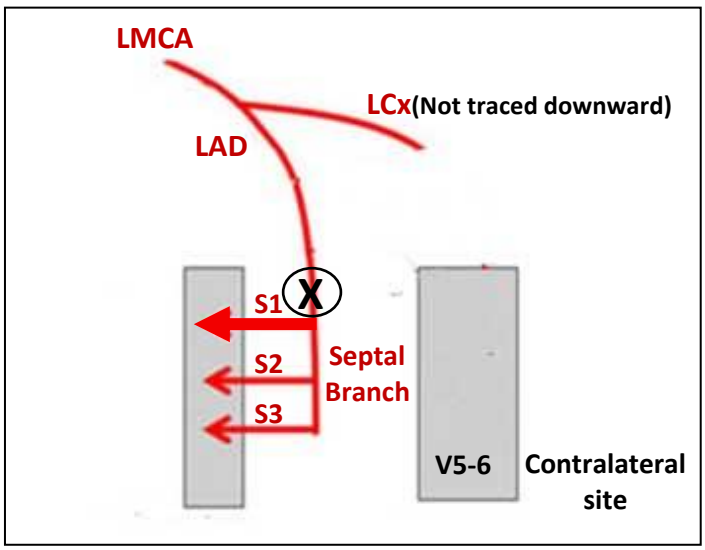


Fig 2.6

5. High-risk ECG patterns indicative of occlusion MI (Tablewise summary)

Table 1 :

Occlusion MI with alteration in T-wave	
OMI patterns and its anatomical site	**ECG findings**
Hyperacute T-waves **(HATW)** mostly with proximal LAD occlusion (RCA or LCX rarely)	• Hyperacute T-waves appear taller and broad-based , often generally more symmetrical than the normal T-waves ; with ≥ in two contiguous leads • As per Dr. Stephen W. Smith T-wave / QRS complex ratio in any V1-4 greater than 0.36 represents acute MI , not subacute or old.
de-Winter pattern **(dWp)** (Proximal LAD occlusion)	• Tall prominent symmetrical T waves in the precordial leads • Upsloping ST segment depression > 1mm at the J point in the precordial leads • Absence of ST elevation in the precordial leads • Reciprocal ST segment elevation (0.5-1mm) in aVR
Wellens' syndrome (Transmural ischemia – reperfusion injury leading to myocardial oedema) Usually proximal LAD occlusion – mainly over leads V2-3 (but may be extended to the other arterial territory as well)	• Type A Wellens' (Biphasic T-wave : the initial positivity with terminal negativity) • Type B Wellens' (Deeply inverted and symmetrical T-wave)

Table 2 :

Occlusion MI with ST changes

OMI patterns and its anatomical site	ECG findings
Precordial Swirl Sign (LAD occlusion prior to S1)	• ST elevation in leads V1 and aVR with reciprocal ST depession in leads V5 and V6
South African Flag Sign (LAD-D1 territory)	• ST elevation primarily localized to leads I , aVL ± V2 • Reciprocal ST depression and / or T-wave inversion in inferior leads , most prominent in lead III
Posterior OMI (Usually RCA through posterior descending artery , LCX with left-dominant coronary circulation)	Mirror images changes of MI of the posterior wall • ST depression , tall R-waves and prominent positive T-waves (V1-3) should be considered posterior OMI until proven otherwise.
Aslanger pattern (RCA or LCX occlusion with multivessel disease)	Resultant ST vector (Aslanger) directed rightwards causing ST elevation in lead III only , sparing the other inferior leads II and aVF • ST↑ only in lead III as evidence of inferior MI • ST ↑ in V1>V2 • Concomitant ST↓ in V4-V6, with a positive/terminally positive T-wave (concomitant subendocardial ischemia)
Northern OMI (LAD and first diagonal bifurcation occlusion in multivessel disease)	• Any STE in aVR and aVL with negative T-waves • Any ST-depression in inferior and lateral precordial leads with positive or biphasic T-waves

Table 3 :

Occlusion MI with miscellaneous group

OMI patterns and its anatomical site	ECG findings
Modified Sgarbossa-Smith Criteria (Occlusion MI in the presence of LBBB)	• Concordant ST elevation ≥1mm in ≥ 1 lead (with a positive QRS complex) • Concordant ST depression ≥1 mm in ≥ 1 lead of V1-V3 • Proportionally excessive discordant STE in ≥ 1 lead anywhere with ≥1 mm STE , as defined by ≥ 25% of the depth of the preceding S-wave.
Terminal QRS distortion (Proximal LAD occlusion)	• Absence of an S wave and J-notch/slur in leads V2/V3 (an imprint of ongoing myocardial insult with uplifting of R-wave with > 50% above the baseline)
New-onset RBBB and LAFB (Proximal LAD occlusion)	• New-onset RBBB • New-onset LAFB

6. Take Home Message

- **In 2018 , Meyers , Weingart and Smith** put a new concept of 'occlusion myocardial infarction' (OMI) in the place of STEMI equivalent – a more reliable paradigm for detecting acute coronary occlusion.
- Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation , leading to imminent infarction of the affected myocardium - to be diagnosed in the absence of traditional ST elevation on ECG remains a 'Herculean task'.
- The concept of occlusion MI with its detection facilitates early reperfusion therapy.
- **Predictive value of ST segment elevation in aVR vs V1** : Since in proximal LAD occlusion , as with precordial swirl pattern , ST elevation is seen in both the leads V1 and aVR with reciprocal ST depression in leads V5 and V6 , it becomes essential here to get it excluded from that of proximal LAD / LMCA occlusion or severe 3VD.
    - ST segment elevation in aVR greater than in V1 indicates LMCA occlusion or severe 3VD or proximal LAD occlusion and it is in association with widespread ST depression.
    - ST elevation in V1 > aVR favours proximal LAD occlusion with anterior transmural injury, as seen in early anterior STEMI or Precordial Swirl Sign (PSS) , distinguished by in LAD occlusion, ST elevation extending across V2–V4, whereas in PSS, it remains isolated to V1 and aVR with reciprocal ST depression in lateral leads V5-V6.
- For having familiarity with the different patterns of occlusion MI one should consult 'Tablewise summary' as illustrated on Page 10-11.
- In context with occlusion MI , always consider dynamic ECG assessment , compared with prior ECGs , correlate with symptoms and serial troponin concentrations , rule-out mimics.
- The natural history of occlusion MI is not so well documented as that of STEMI , where extensive data on the evolutionary pattern are available.
- Occlusion MI should not be missed, as delayed recognition may lead to its progression into a full-blown STEMI

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dE WINTER pattern (dWp) ECG: ANTERIOR STEMI EQUIVALENT (OCCLUSION MI)

de Winter pattern (dWp) ECG : ANTERIOR STEMI EQUIVALENT (Occlusion MI)

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OUTLINE

de Winter pattern (dWp) ECG : anterior STEMI equivalent (Occlusion MI)

A Narrative Review

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A destructive 'Fire for the Heart' is standing in front but in disguise – how to bend the crisis is the main concern. This is the emergency to recognize this wavy tide , otherwise the battle of life would be lost.

- ☐ **Anterior STEMI equivalent – representing proximal LAD occlusion without meeting the traditional criteria of ST elevation with characteristic ECG findings.**
- ☐ **ECG findings : Tall prominent symmetrical T waves in the precordial leads ; Upsloping ST segment depression > 1mm at the J point in precordial leads and Reciprocal ST segment elevation (0.5mm – 1mm) in aVR**

Its recognition is essential in acute atherothrombotic myocardial infarction to have timely reperfusion therapy to conquer the battle of life in the interest of humanity.

1. Introduction

This ECG pattern was first reported in **2008** in a case series elaborated by a group of Dutch cardiologists Robbert Jan de Winter , Verouden NJ, Wellens HJJ et al. They recognized this entity as an **anterior STEMI equivalent** – representing proximal LAD occlusion without meeting the traditional criteria of ST elevation. The 12-lead ECG recorded the following mass of information as observed by this group of researchers.

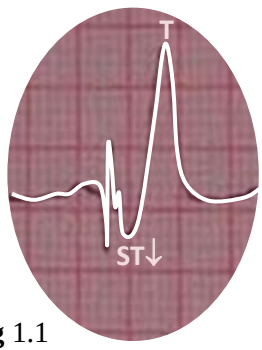


Fig 1.1

ECG diagnostic criteria

- Tall prominent symmetrical T waves in the precordial leads
- Upsloping ST segment depression > 1mm at the J point in precordial leads
- Absence of ST elevation in precordial leads
- Reciprocal ST segment elevation (0.5mm – 1mm) in aVR

These findings as a group are nomenclatured as '**de Winter pattern ECG'**. This pattern is a marker of acute occlusion of proximal left anterior descending (LAD) coronary artery , and this proves to be a very helpful in diagnosing this condition. This group of researchers observed this ECG pattern in 30/1532 patients with acute LAD occlusion pointing to its **prevalence to be 2% of acute LAD occlusion** , but a recent study suggests a higher prevalence.

The history was repeated in 2009 when Verounden et al. also reported the same findings in their new case series study. They observed this de Winter ECG pattern in 35/1890 patients requiring revascularization by PCI to the culprit LAD coronary artery. They also reported its

prevalence to be 2% with such cases. The patients with their group were younger, more often to be male and with a higher incidence of hypercholesterolemia , compared with a classic STEMI pattern.

Unfamiliarity with this atypical high risk ECG pattern with the clinicians may land the patient with negative effects on morbidity and mortality. Immediate reperfusion therapy by PCI or thrombolyis if PCI facility is not available within the range , is the need of this emergency. **Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation resulting in acute myocardial infarction – to be diagnosed in the absence of traditional ST elevation on ECG.**

2. Pathological basis of 'de Winter pattern ECG'

The basic lesion is subtotal occlusion of proximal LAD (mostly tight) resulting in subendocardial ischaemic insult without touching a narrow rim of subepicardial tissue.

There are two important points to be discussed here :

- ☐ The pattern of subendocardial ischaemic insult.
- ☐ The role of ischaemia induced hypoxia with the activation of sarcolemmal K_{ATP} .

The pattern of subendocardial ischaemic insult.

The pathophysiological changes observed in this condition are supported by CE-CMR (Contrast-enhanced cardiovascular magnetic resonance).

Transmural myocardial oedema with full thickness of the involved myocardium but non-transmural late-enhancement (not reaching to the epicardium), pointing towards that the outer subepicardial layers is preserved from dying. This means there is considerable subendocardial necrosis with a preservation of a peripheral rim of subpericardial tissue.

The presence of collateral circulation associated with this condition might have provided subsidiary coronary circulation with saving the subepicardial myocardium from necrosis.

The role of ischaemia induced hypoxia with activation of sarcolemmal K_{ATP}

- Under normal metabolic conditions , cardiac sarcolemmal K_{ATP} channels keep themselves closed. However , these channels can be opened when being exposed to a severe metabolic stress such as ischaemia induced hypoxia.
- K_{ATP} activation during ischaemia causes hyperpolarization of the cardiac membrane with a downregulation of Ca2+ - dependent excitation-contraction coupling and thus , preserving cardiac energy with a temporary protection of a small number of surviving cardiac myocytes. That's why , the K_{ATP} activation during ischaemia is likely to be cardioprotective in nature. The entire concept of cardioprotection is illustrated by the following sketch :

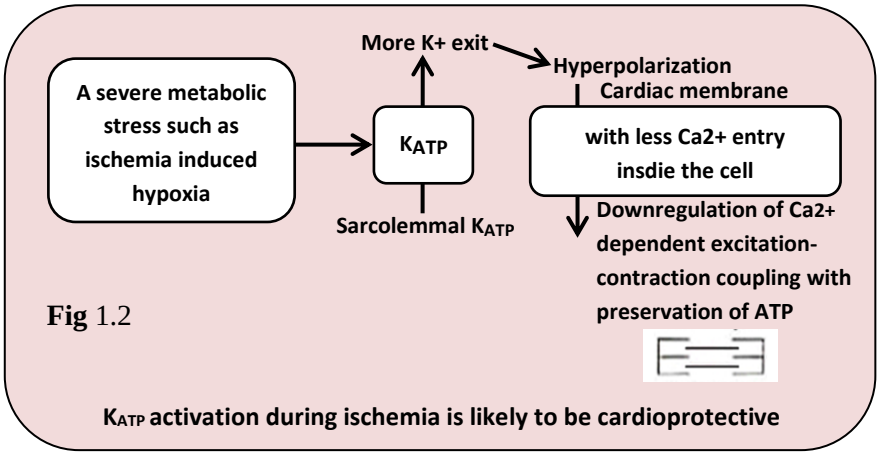


Fig 1.2

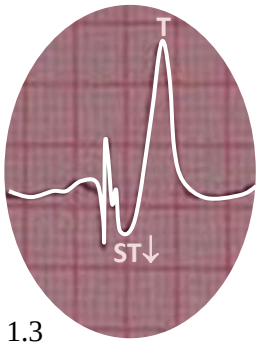
Opens ATP-sensitive k+ channels → ↑K+ exit without accompanying Ca2+ entry →transient cessation of excitation-contraction coupling.

Keynotes

This impact resulting from almost total occlusion of proximal LAD keeps a considerable subendocardial region in the state of ischaemic insult with sparing of the small rim of subepicardial region from its catastrophic effect.

It should be noted here that the activation of sarcolemmal-sensitive potassium (K_{ATP}) channel – an important cardioprotective mechanism is presumed to be operative due to a mutation in Kir 6.2 gene.

3. A possible interpretation of de Winter wave on the basis of Ventricular Action Potential



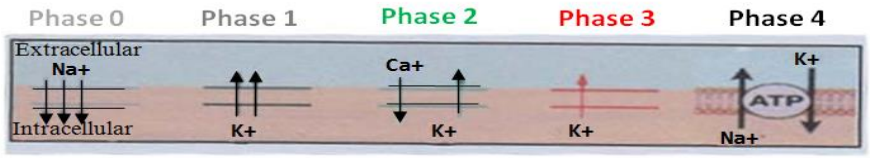
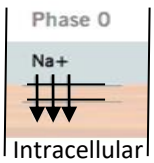


Fig 1.3

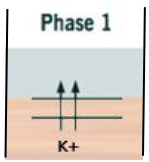
The most probable pathophysiological background in de Winter pattern :

- Hypoxic-driven opening of the ATP-dependent Potassium channels (K_{ATP}) limits subendocardial injury through cardioprotective mechanism , with simultaneous preservation of subepicardium from dying.
- Myocardial protection is also provided through collateral circulation preserving a small rim of subepicardial tissue.

NB : Voltage is the measure of energy available to transport electrons. This requires a gradient (more energy on one side than the other – here is the voltage gradient in between subendocardial and subepicardial myocardial tissue) – the flow continues towards the exploring electrode to be recorded on ECG as a positive wave.

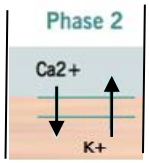


☐ **Phase 0** : The QRS waves are usually normal but occassionally some loss of R wave or poor R wave progression in the precordial leads may be observed.



☐ **Phase 1**: Normally this phase corresponds to the outward movement of K^+ channels for a transient period – following J notch on the descending limb of the QRS complex.

J-point moves rapidly downwards resulting in depressed negative ST segment due to the mechanism of hyperpolarization, as previously discussed.

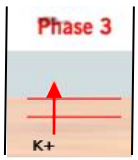


☐ **Phase 2** (plateau phase) : **Normally** during this phase , there is a mutual exchange of Ca^{2+} and K^{+} ions across the cardiac membrane travelling across a homogenous ventricular gradient with no current of injury resulting in isoelectric segment on ECG (Corresponding to ventricular contraction). This is inscribed as isoelectric ST segment on ECG.

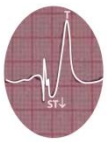
In de Winter wave there is upsloping ST segment depression > 1mm at the J-point in the precordial leads , as depicted with the preceding illustration :

Reasoning :

- Upsloping of ST segment is possibly due to the rapid return of the depressed ST segment-when the hyperpolarization mechanism gets ended.

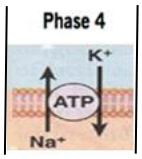


☐ **Phase 3** : This phase corresponds to repolarization being reflected on ECG in the form of T wave (Normally T wave amplitude is <10 mm in precordial leads).



Here in de winter pattern :

- Peaked tall T wave is the expression of the shorter time duration between subendocardial and subpericardial repolarization – a sudden return from hyperpolarized state to repolarization phase 3, inscribing tall prominent symmetrical T waves.



☐ **Phase 4** : This phase is the resting phase on ECG being reflected by TP segment – an isolectric line.
No abnormality detected during this phase.

---

4. Evolution of dWp to STEMI – a real catastrophe

dWp was considered as a static ECG pattern in previous observations but a recent retrospective study seems to demonstrate that it is dynamic ECG phenomenon.
As the ischemic insult continues , the epicardial zone may be completely lost leading to enhancement as denoted by transmural extension (i.e. endo-epicardial) , with the flow of current from endo-to-epicardium giving rise to ST segment elevation , manifested as evolving STEMI. **The de Winter ECG pattern is always dynamic**. Patients with this ECG pattern can evolve to be presented as ST-segment elevation MI in precordial leads.

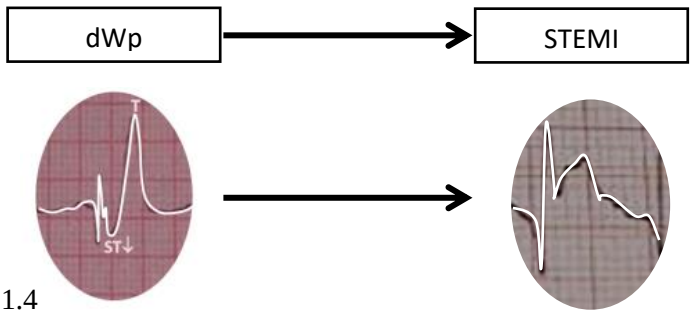


Fig 1.4

There would be no surprise at all if the clinician concerned observes the different facies of such evolution on ECG with the ensuing hours of follow up , highlighting the variable nature of this occlusive dWp.

The clinical presentation of such de Winter pattern may lead to left ventricular dysfunction in course of time and possibly explains the frequent recurrences of chronic heart failure in such patients whose ECG has not reported significant decreased LVEF at the initial presentation.

5. The reasoning behind ECG changes in precordial leads in dWp

✓ Here the proximal occlusion of LAD means occlusion before the origin of its diagonal branch – thus facilitating the ECG recording through V1-V6 (mainly V1-3 , the rest V4-6 being also supplied by left marginal artery of LCx). The entire scheme is illustrated with the following sketch :

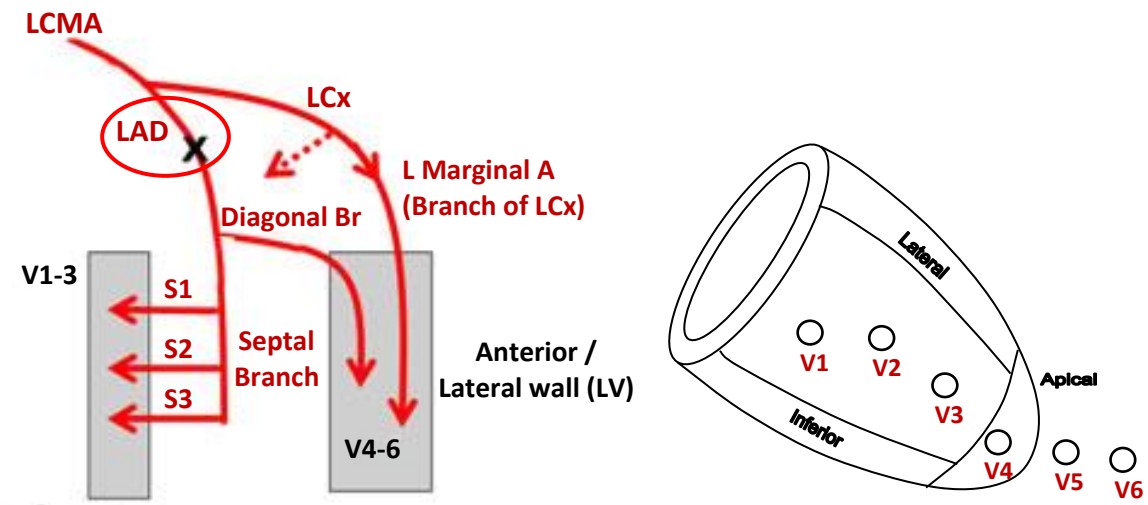
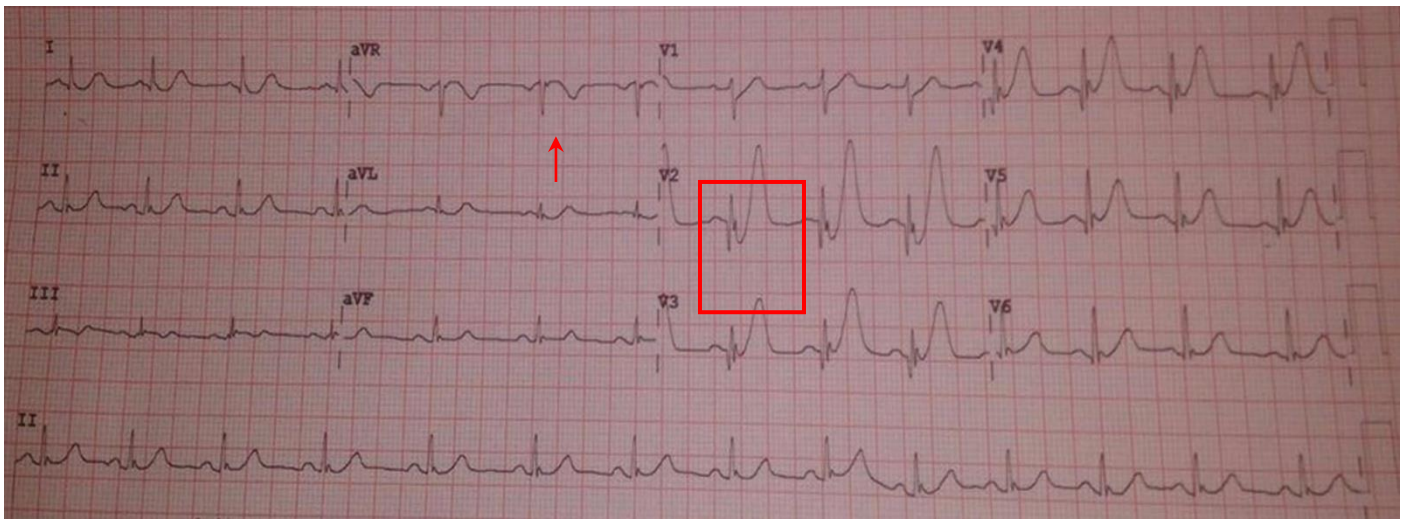


Fig 1.5

Leads V1-3 (V4-6) are the reflecting mirror for de Winter pattern to be recorded. This is also to be mentioned here that there is a reciprocal ST segment elevation in aVR i.e. the reversed mirror reflection of the precordial leads. Therefore , if the clinicians find tall , prominent , symmetrical T wave in the precordial leads, they should extend their vision of observation towards the other subsets of ECG findings in favour of de Winter pattern ECG. **This would be very much helpful in diagnosing this condition in the presence of ongoing chest pain. Some clinicians regard leads V1-6 as the eyes through which dWp can be visualized easily.**

6. An interesting case study

ECG of a middle aged man presenting as severe chest pain since 3 hours.



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

Comments :

- Tall prominent symmetrical T waves in the precordial leads
- Upsloping ST segment depression > 1mm at the J point leads , most marked in V2
- Absence of ST elevation in the precordial leads
- Reciprocal ST segment elevation (0.5mm) in aVR

✓✓ **de winter pattern has a high positive predictive value (95.2%-100%) suggestive of acute occlusion of proximal LAD coronary artery.**

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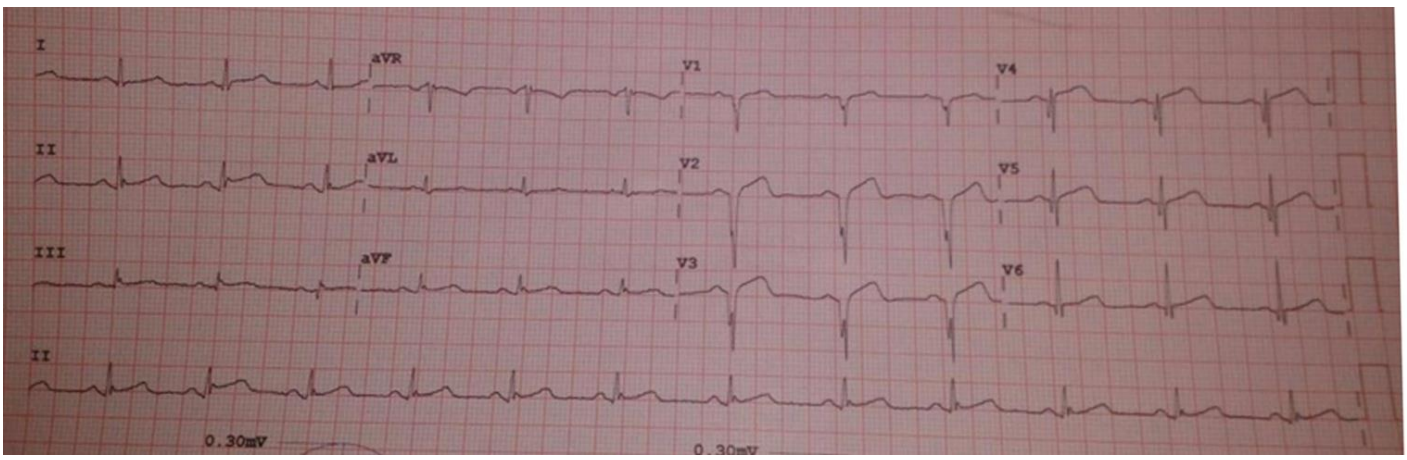
de winter pattern ECG

☐ First reported by de winter in 2008 – **ECG pattern is an anterior STEMI equivalent**

☐ **KEY DIAGNOSTIC POINTS = peaked T waves with preceding upsloping ST depression in precordial leads**

Management : Thrombolysis (PCI facility was not available within the approach)
Post Thrombolysis ECG (persisting Q wave from V1-V5 with the normalization of tall T waves)



7. Take Home Message

- ☐ de Winter pattern (dWp) is an anterior STEMI equivalent – representing proximal LAD occlusion without meeting the additional criteria of ST elevation (Nowadays this is termed as **occlusion MI**)

- ☐ **ECG diagnostic criteria**
  - Tall prominent symmetrical T waves in the precordial leads
  - Upsloping ST segment depression > 1mm at the J point in precordial leads
  - Absence of ST elevation in precordial leads
  - Reciprocal ST segment elevation (0.5mm – 1mm) in aVR

- ☐ The basic lesion is subtotal occlusion of proximal LAD (mostly tight) resulting in subendocardial ischaemic insult without touching a narrow rim of subepicardial tissue.
  K_{ATP} activation during ischaemia causes hyperpolarization of the cardiac membrane with a downregulation of Ca^{2+} - dependent excitation-contraction coupling and thus , preserving cardiac energy with a temporary protection of a small number of surviving cardiac myocytes in some epicardial region. That's why , the K_{ATP} activation during ischaemia is likely to be cardioprotective in nature.

- ☐ Patients with this ECG pattern can evolve to be presented as STEMI (a real catastrophe)

- ☐ There would be no surprise at all if the clinician concerned observes the different facies of such evolution on ECG with the ensuing hours of follow up , highlighting the variable dynamic nature of this occlusive dWp.

- ☐ de winter pattern has a high positive predictive value (95.2%-100%) suggestive of acute occlusion of proximal LAD coronary artery.

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WELLEN'S SYNDROME :

A DYNAMIC ECG SIGNATURE OF OCCLUSION MI

WELLENS' SYNDROME : A DYNAMIC ECG SIGNATURE OF OCCLUSION MI

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OUTLINE

Introduction
Wellens' syndrome represents the reperfusion state of atuo-thrombolysed Proximal LAD occlusion without having the impact of myonecrosis (Type A and B)

Historical background : with Red Tails Story- a pre-infarction state

A diagnostic approach to Wellens' syndrome

Pathophysiology

- Wellens' syndrome follows a temporary obstruction of the proximal LAD which is commonly caused by the rupture of an atherosclerotic plaque , with subsequent autothromobolysis before complete myocardial infarct sets in.
- The exact mechanism of the ECG changes in Wellens' syndrome is still unknown but this is postulated that it is caused by **transmural ischemia-reperfusion mechanism leading to myocardial oedema.**
- There is a red alert because 75% of such cases may turn into acute anterior wall myocardial infarction due to reocclusion of proximal LAD coronary territory.

(A concept of Pseudo-Wellens' syndrome also mentioned)

Understanding Wellens' syndrome on the basis of ventricular action potential

Diagnostic leads for T-wave changes in Wellens' syndrome

Differential diagnosis with inverted T wave in context with Wellens' syndrome

Illustration of Wellens' syndrome by ECGs

Take Home message

References

Wellens' Syndome : A Dynamic ECG Signature of Occlusion MI

A Narrative Review

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Two phenomena – protection and destruction have been chasing each other since time immemorial. Protection keeps us alive and luminous in life– upholding the Bio-tissues to run smoothly with hero-force. On the contrary , the destruction does the reverse and the hour of adversity sets in life. These are two sides of the same coin.

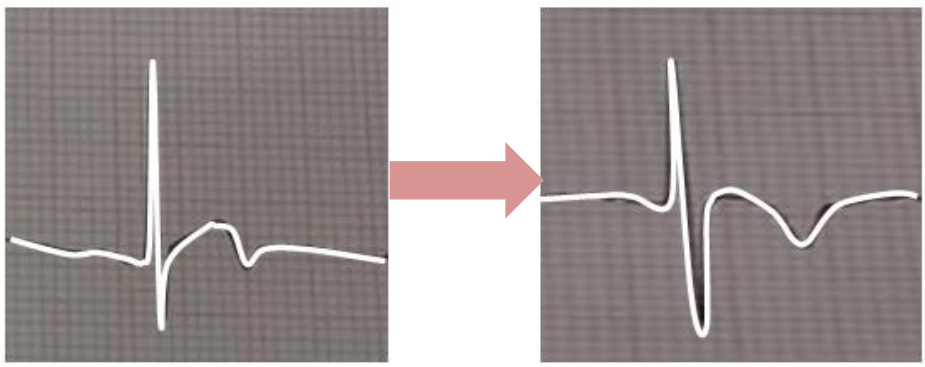
- ☐ **O heart thou is protected from the throatling hands of thrombosis in coronary circulation by the process of auto-thrombolysis with reperfusion – but might be ruined again by the gloomy blow with the resetting of thrombosis.**
- ☐ **Protection by auto-thrombolysis is the comedy and destruction by reocclusion is the tragedy.**

So is the basis of Wellens' syndrome – protection is chased by destruction. Pre-infarction state may be turned into true infarction state.

1. Introduction

Wellens' syndrome denotes an abnormal electrocardiography (ECG) pattern – biphasic or deeply inverted T-waves mainly on chest leads V2 and V3. To start with there is a critical occlusion of proximal LAD artery presenting as unstable angina but followed by auto-thrombolysis of so formed clot resulting in pain-free period with so typical T-waves changes. The artery may be reoccluded resulting in myocardial infarction.

Nowadays this entity is kept under the heading of 'Occlusion MI'.



Biphasic T-wave (the initial positivity and terminal negativity)

Type A Wellens' Syndrome

Deeply inverted and symmetrical T-wave

Type B Wellens' Syndrome

Fig. 1.1

This is worthwhile to mention here that the patients with this syndrome come to the clinicians in a pain-free state and with normal or slightly elevated cardiac markers with the recent history of preceding anginal pain in the background.

This syndrome results from temporary obstruction of LAD coronary artery , usually caused by the rupture of an atherosclerotic plaque leading to LAD occlusion with subsequent auto-thrombolysis of the clot before complete myocardial infarction sets in.
THIS IS CARDIO-PROTECTIVE PHENOMENON.

This is also to be noted here that 75% of the patients with such ECG findings would lapse into acute anterior wall myocardial infarction usually within a week.
THIS IS THE CARDIO-DESTRUCTIVE PHENOMENON.

Protection is the comedy and destruction is the tragedy. To prevent such a tragedy, intervention by urgent PCI (percutaneous corornay intervention) is needed.

2. Historical background with Red Tails story

The **"Red-Tails"** where the tuskegee airmen a group of heroic fighter pilots during the second world war – their planes were painted with distinct red tails , making them easy to identify. This embraced story is powerful in visual analogy often used to describe the urgent warning sign that Wellens' syndrome represents – especially in a patient with pain-free period but harbouring a ticking time-bomb in the proximal LAD.

Dr. Zwaan , Wellens et al (1982) , a group of cardiologists from Netherlands reported some notable findings , while observing their patients admitted for unstable angina.

Their findings are summarized as below :

- ☐ They described a subgroup of patients with an unstable angina (26/145 = 18%) having typical T-wave changes in precordial leads with negative cardiac enzymes but with poor outcome.
- ☐ All patients received relief of chest pain on conservative management but 8 of the first 9 patients developed anterior mycoardial infarction and three died – a poor prognostic outcome (The average time for infarction following such ECG changes was ranging from Day 1 to 23 ; with the average of 8.5 days).
- ☐ Some patients who underwent cardiac catheterization with coronary angiography were found to have greater than 90% stenosis in their proximal LAD.

In a second prospective study initiated by de Zwaan et al , 180 out of 1260 (14%) patients for unstable angina were detected to have typical T changes on ECG. All 180 patients underwent catheterization and all were found to have at least 50% blockade of LAD.

In nutshell as per their observation , there exists a subgroup of unstable anginal patients showing typical T-wave changes on ECG with poor outcome - such patients are usually turned to anterior myocardial infarction within a few days. **That's why , this ECG pattern with typical T changes are in particularly having high risk of developing an extensive myocardial infarction representing as Red Tails story.**

3. A diagnostic approach to Wellens' syndrome

It is obvious by the foregoing discussion that **Wellens' syndrome represents the reperfusion state of thrombolysed LAD circulation without having the impact of myonecrosis**. Therefore , the definition of this syndrome must include **the evidence of reperfusion-injury on ECG as T-wave changes without the evidence of myonecrosis.**

Diagnostic criteria for Wellens' syndrome include :

- **Reperfusion injury-related repolarization**
  Biphasic T-waves (with initial positivity and terminal negativity) or deeply inverted T-waves are commonly seen on leads V2 and V3. However , if the lesion is more proximal in the LAD , the T-wave changes would be more widely spread along the precordial leads.
  These T changes on ECG are present with a pain free state.

PLUS

- **Without the evidence of acute anterior wall myocardial infarction**
  such as
  - ECG without Q wave with isoelectric or minimally elevated ST segment - <1mm and with the absence of precordial poor R wave progression.
  - Normal or slightly elevated cardiac marker (in one prospective study, only 12% of such patients had elevated cardiac enzymes, and these elevations were less than twice the upper limit of normal).
- **Recent history of unstable angina coinciding with the previous temporary LAD occlusion.**

It is to be noted that there is a lack of universal definition for a preserved precordial R-wave progression but common criteria include :

- R-wave > 2-4 mm in V3 or V4
- R-wave in V4>V3 or V3>V2
- R-wave in V3 $\geq$ 3mm

There are two patterns of T-wave in Wellens' syndrome : namely **Type A** biphasic T-wave with initial positivity and terminal negativity (approximately in 25% of cases) and **Type B** with deeply and symmetrically inverted T-wave (approximately in 75% of cases). **Type A T-waves are usually evolved into Type B T-waves**. These T-wave abnormalities may be persisting for hours to weeks.

Wellen's syndrome is not always an acute process – can develop over days to weeks. Already mentioned that this ECG pattern develops when the patient is not experiencing the chest pain. The recurrence of chest pain indicates the onset of impending acute anterior wall myocardial infarction.

4. Pathophysiology

The pathophysiology of Wellens' syndrome can be summarized as follows :

- ❖ Wellens' syndrome follows a temporary obstruction of the proximal LAD artery which is commonly caused by the rupture of an atherosclerotic plaque , with subsequent thromobolysis before complete myocardial infarct has set in.
- ❖ The exact mechanism of the ECG changes in Wellens' syndrome is still unknown but this is postulated that it is caused by **transmural ischemia-reperfusion mechanism leading to myocardial oedema.**
- ❖ There is a red alert because 75% of such cases may turn into acute anterior wall myocardial infarction due to reocclusion of proximal LAD coronary territory.

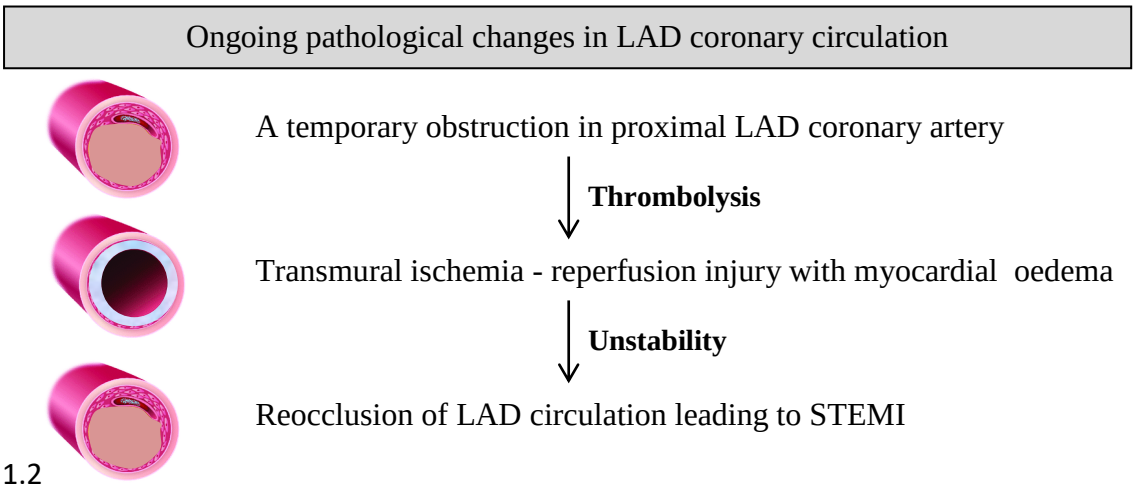


Fig. 1.2

Evolution of ECG changes in the light of pathological scenario

The Wellens' syndrome is dynamic journey in continuum–pre-infarction state might be converted to infarction state

Pathological changes	ECG changes
➢ Temporary obstruction in proximal LAD coronary artery	This phase may cause anginal chest pain. It may not be successfully captured on ECG – showing mild ST changes, or negative deflection of the T-wave in V2 and V3.
➢ Reperfusion oedema ± pre-hospital antiplatelets	There is resolution of the chest pain - ST changes improve with typical biphasic or inverted T-waves (If the artery remains open the biphasic T-wave would evolve to deeply inverted T-wave due to transmural oedema in vicinity).
➢ LAD can reocclude at any time	With the recurrence of anginal chest pain , the ST segment and T-wave pattern appear to normalize into hyperacute T-wave. So called **"Pseudo normalization"**. This is a sign of hyperacute STEMI. **If the artery remains occluded** , the patient can develop an evolving anterior STEMI with its characteristic features on ECG.
➢ "Stuttering pattern" with intermittent reperfusion and reocclusion	As alternating ECGs patterns – Wellens' and Pseudo normalization.

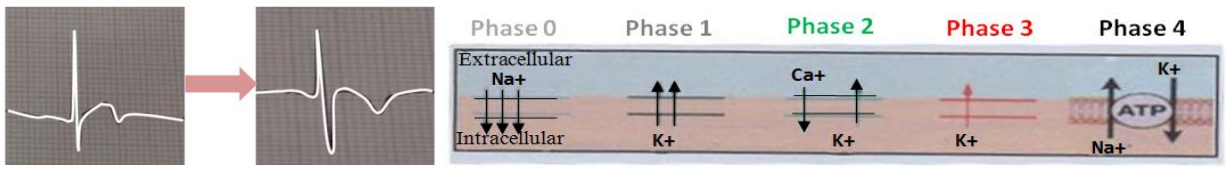
NB : These pathological events as discussed may be considered as gentle ripples on the ECG , hiding a storm in the coronaries. The paradox of wellens' is the recurring cycle of pain-free period and pain-rich period unless the clinician acts by timely reperfusion therapy.

Pseudo-Wellens' syndrome

Coronary artery spasm in LAD may also be responsible for the genesis of Wellens' syndrome , nomenclatured as Pseudo-Wellens' syndrome – rarely reported in the literature , after the resolution of the spasm. Coronary flow is restored leading to reperfusion injury-related repolarization abnormalities depicted on ECG in precordial leads with typical biphasic or inverted T-waves, as discussed before.

The causative factor for 'Pseudo-Wellens' syndrome' may include the use of Cocaine / Marijuana and another rare cause is myocardial bridging in which the coronary artery tunnels under a bridge of myocardium resulting in LAD coronary artery stenosis. Additionally, the Wellens' pattern can be seen in Takotsubo cardiomyopathy, which is thought to be the result of myocardial oedema.

5. Understanding Wellens' syndrome on the basis of ventricular action potential



Biphasic T-wave (initial positivity and terminal negativity)

Deeply inverted and symmetrical T-wave

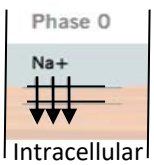
Fig. 1.3

Type A Wellen's syndrome Type B Wellen's syndrome

The pathophysiologic background in Wellens' syndrome :

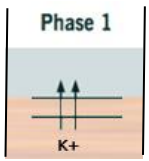
- A temporary obstruction of the LAD coronary artery , commonly caused by the rupture of an atherosclerosis plaque , the subsequent auto-thrombolysis before complete myocardial infarct sets in.
- The next is transmural ischemia – reperfusion injury leading to myocardial oedema.

NB : The description as noted below is in context with Wellens' syndrome (pain-free period).

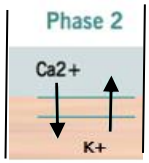


☐ **Phase 0** : This phase corresponds to depolarization reflected on ECG as QRS wave.

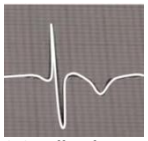
Since there is reperfusion in the initial stage without myocardial infarction , the changes recorded are absence of pathological Q wave without poor R wave progression.



☐ **Phase 1**: Normally the phase corresponds to the outward movement of K^+ channels for a transient period – inscribed by a J notch on the descending limb of the QRS complex. **No abnormality detected during this phase.**



☐ **Phase 2** (plateau phase) : Normally during this phase , there is a mutual exchange of Ca^{2+} and K^{+} ions across the cardiac membrane travelling across a homogenous ventricular gradient with no current of injury resulting in isoelectric segment on ECG (Corresponding to ventricular contraction). This is inscribed as isoelectric ST segment on ECG.

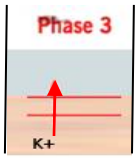


Minimally elevated ST segment

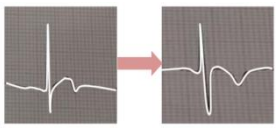
Isoelectric or minimally elevated ST segment < 1mm

Reasoning :

- Isoelectric ST segment is inscribed due to the preservation of myocardium without myonecrosis.
- Minimally elevated ST segment (<1mm) might represent the very minimal residual impact by the temporary occlusion of LAD artery.



☐ **Phase 3** : Normally this phase corresponds to repolarization being reflected on ECG in the form of upright T-wave.



Biphasic T-wave (initial positivity and terminal negativity) Type A Wellen's syndrome

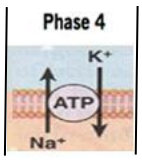
Deeply inverted and symmetrical T-wave

Type B Wellen's syndrome

Biphasic T-wave (with initial postivity and terminal negativity) may be followed by deeply inverted T-wave.

Reasoning :

- **Myocardial ischemia – reperfusion injury results in local myocardial oedema** which can change the direction of ongoing repolarization process. Due to the delay in passing the current through this oedematous myocardium – the current flows uniformly but through the opposite direction i.e. away from the subepicardial zone. This produces symmetrical deep T-wave inversion.
- In the initial stage of partial oedematous myocardium the first half of the wave with initial positivity and the terminal part with negativity exist, as seen in Type A Wellens' syndrome.



☐ **Phase 4** : This phase is the resting phase brought about by '$Na^{+}K^{+}$ -ATPase' mechanism.

No abnormality detected during this phase.

---

6. Diagnostic leads for T-waves changes in Wellens' syndrome

- Despite proximal LAD occlusion before the septal branches , the first septal perforator (S1) – often larger and anatomically dominant may remain functionally perfused via residual flow or collaterals. This relative preservation of the basal septum explains why V1 aligned with S1 lesion , may escape the classical T-wave inversion in Wellens' syndrome. Mainly V2-V3 leads aligned to S2-S3 perforators may be involved and reflect changes in the Wellens' syndrome.
- In cases of proximal LAD occlusion before the diagonal branches , the T-wave changes would be more widely spread along the precordial leads ranging from V1 to

V6 including even leads I and aVL , provided these leads represent myocardium supplied by the LAD system. If the high lateral wall is instead supplied by an unoccluded via the marginals , leads I and aVL changes may escape.

- This would be better to mention here that the right coronary artery tends to be relatively escaped from early atheromateous changes due to its low intraluminal pressure , steadier flow dynamics , and reduced wall shear stress. Its anatomical and physiological environment leads to a lower incidence of endothelial injury and subsequent atherosclerosis – explaining why RCA lesions are of less frequent of severe compared to those in the left coronary system , particularily the LAD and therefore , RCA does not show T-changes of Wellens' syndrome.

The entire concept is depicted with the following sketch :

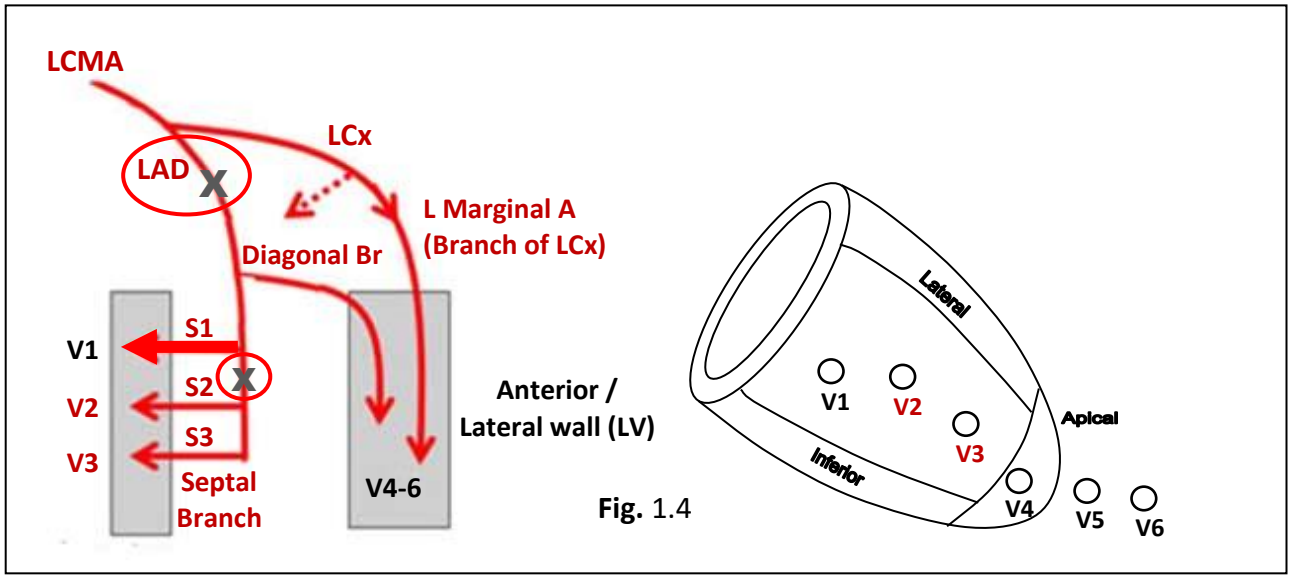


Fig. 1.4

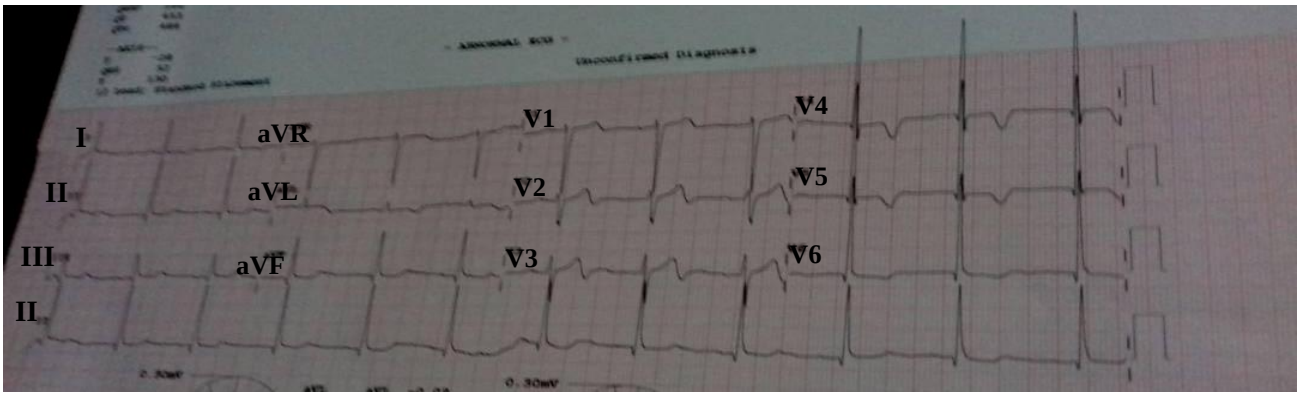
7. Differential diagnosis with inverted T-wave in context with Wellens' syndrome

☐ It becomes essential to differentiate between wellens' sydnrome and ongoing acute coronary insufficiency as de novo.

- Acute coronary insufficiency may slow the propagation of cardiac impulse takeover by endocardium and therefore , it may present with deep TWI over the anterior chest leads if there is an involvement of proximal LAD.
- In clinical cardiology, symmetrical deep T-wave inversions in anterior leads (V2–V3) as discussed are often associated with Wellens' Syndrome, a known marker of critical proximal LAD stenosis. However, similar T-wave changes may also be observed as a de novo presentation of acute coronary insufficiency, particularly if involving the same artery.
- Both patterns demand urgent attention but the differentiation depends upon recognizing whether one is seeing the lesion with prior unstable angina or a heart still under the impact of Acute coronary insufficiency going on.

8. Illustration of Wellens' syndrome by ECGs

ECG 1 : Type A Wellens' syndrome



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

This ECG was recorded in a middle aged smoker male with a preceding history of chest pain with Trop I negative.

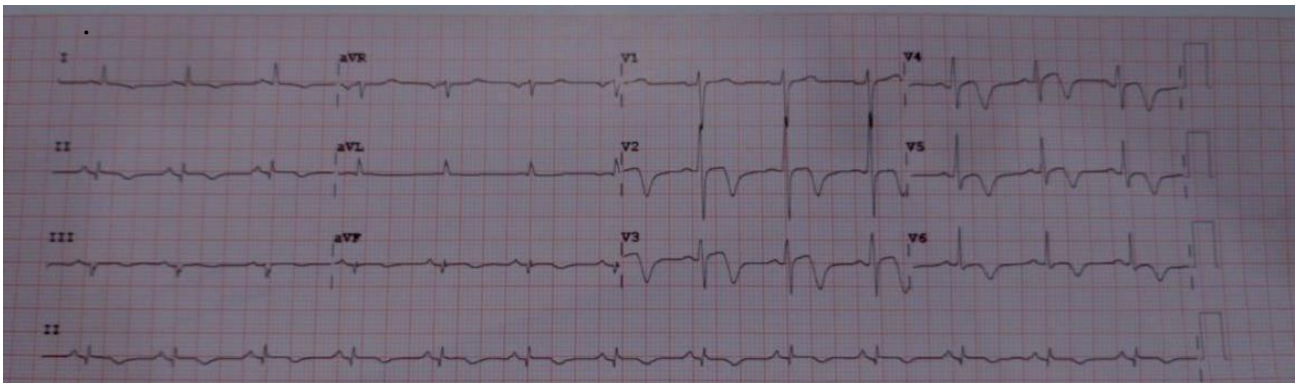
Findings :

- Biphasic T-wave with initial positivity and terminal negativity in leads V1-V3.
- Absence of precordial poor R wave progression.
- T-waves inversion in leads V4-V5 , also in lead aVL.

Discussion :

- These T-wave changes are more widely spread along the precordial leads. Therefore , the lesion is more proximal in the LAD.
- Since the lesion is admixture of both biphasic and negative T-wave in precordial leads, this indicates the lesion is progressing towards the next phase Type B.

ECG 2 : Type B Wellens' syndrome (with another patient)



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

This ECG shows :

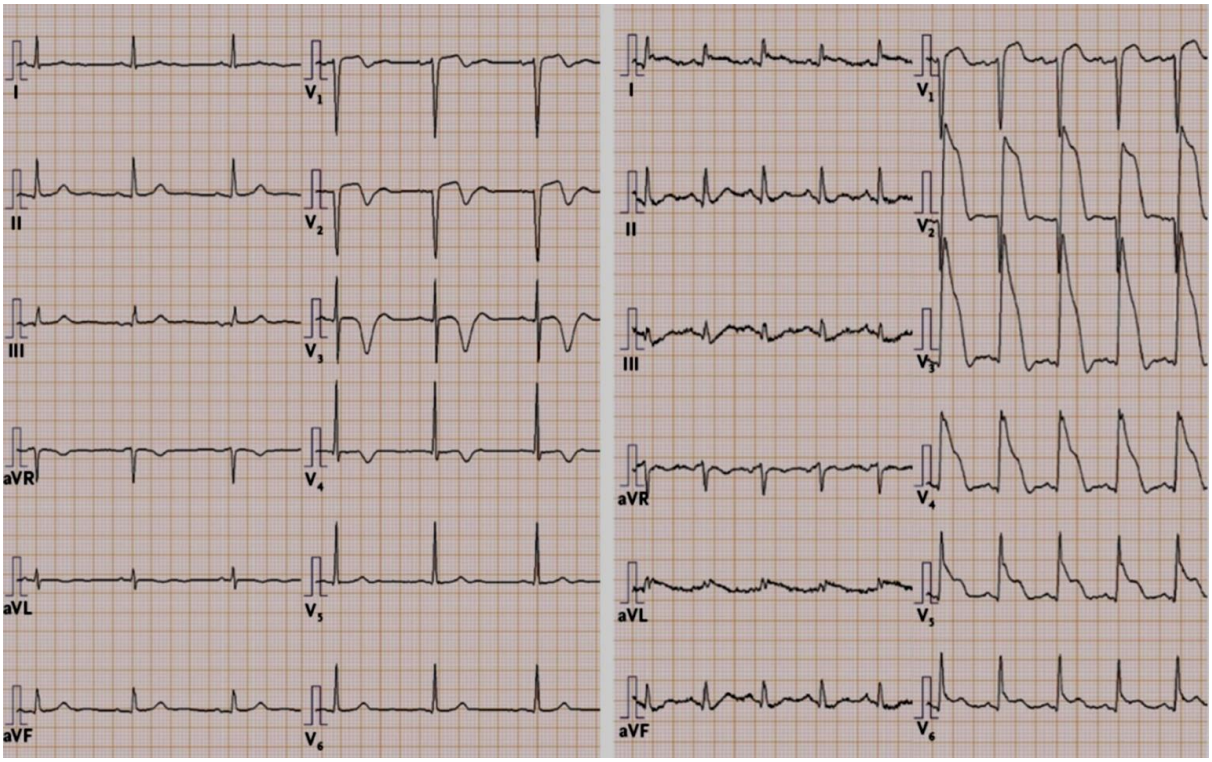
- Deeply inverted T-waves in leads V1-V6 with minimally elevated ST segment less than 1 mm.
- The absence of precordial poor R wave progression
- Troponin I : normal

Discussion :

The findings are consistent with Type B Wellens' syndrome.

ECG 3: Turning of Type B Wellens' syndrome into acute anterior ST elevation myocardial infarction (a separate patient)

There is a tendency to reocclude LAD branch of coronary circulation , the accurate reasoning of this reocclusion is still remaining in darkness. This fact should be emphasized here that undiagnosed cases might be missing the opportunity of intervention by PCI. This may result in MI catastrophe - illustrated by ECGs on the next page.



Type B Wellens' syndrome → Acute ST elevation MI

Source : Dr. N.K Singh , Director , Diabetes and Heart research centre , Dhanbad , Editor , www.cmeindia.in posted on CME INDIA.

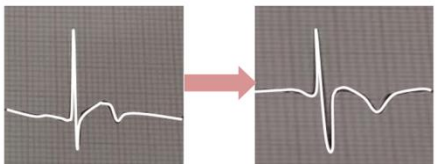
MI with "Shark Fin" pattern – formed by the confluence of QRS and T wave as a result of extreme ST elevation.

9. Take Home message

- Wellens' syndrome represents the reperfusion state of atuo-thrombolysed proximal LAD circulation without having the impact of myonecrosis.
- This syndrome results from temporary obstruction of proximal LAD coronary artery , usually caused by the rupture of an atherosclerotic plaque leading to LAD occlusion with subsequent auto-thrombolysis of the clot before complete myocardial infarction sets in.

  75% of such patients would lapse into acute anterior wall myocardial infarction usually within a week.

- On ECG Wellens' syndrome denotes an abnormal electrocardiography (ECG) pattern – biphasic or deeply inverted T-waves mainly on chest leads V2 and V3



Biphasic T-wave (initial positivity and terminal negativity)
Type A Wellen's syndrome

Deeply inverted and symmetrical T-wave
Type B Wellen's syndrome

Fig. 1.5

If the lesion is more proximal in the LAD , the T-wave changes are widely spread along the precordial leads.
These T-changes on ECG are present with a pain free state.

- Diagnostic criteria for Wellens' syndrome include :
  - Reperfusion injury-related repolarization , as T-changes on ECG (discussed on the preceding page)

    PLUS

  - Without the evidence of acute anterior wall myocardial infarction such as
    - ECG without Q wave with isoelectric or minimally elevated ST segment - <1mm and with the absence of precordial poor R wave progression.
    - Normal or slightly elevated cardiac marker (in one prospective study, only 12% of such patients had elevated cardiac enzymes, and these elevations were less than twice the upper limit of normal).
  - Recent history of unstable angina coinciding with the previous temporary LAD occlusion.

Pseudo-Wellens' syndrome , as with Coronary artery spasm in LAD , the use of Cocaine / Marijuana and another rare cause is myocardial bridging over the concerned proximal LAD , sometimes seen with Takotsubo cardiomyopathy, due to accompanying myocardial oedema.

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18. Referred to ChatGPT whenever needed

PRECORDIAL SWIRL SIGN- A NEW POINTER TOWARDS PROXIMAL LAD OCCLUSION MI

PRECORDIAL SWIRL SIGN – A NEW POINTER TOWARDS PROXIMAL LAD OCCLUSION MI

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OUTLINE

Introduction
Epicardial current of injury so produced over the subepicardial zone in occlusion MI might inscribe ST elevation , somewhat different in distribution from that of classical STEMI.

What is Precordial Swirl Sign ?
ST-segment elevation and /or hyperacute T-waves in V1-V2 and reciprocal ST-depression and /or T-wave inversion in V5-V6

Electrophysiological mechanism – a consideration

- Blood supply of the concerned interventricular septum
- The concept of current of injury
- A concept of ST elevation with reciprocal ST depression in context with 'Precordial Swirl Sign'

An interesting case study (Precordial Swirl Sign)

A mindfullness attention to the associated LVH, LBBB, etc to exclude the false impression of Precordial Swirl Sign

Take Home message

References

Precordial Swirl Sign – A new pointer towards proximal LAD Occlusion MI

A Narrative Review

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A culprit – if does the offence in disguise , it would be be a 'Herculean task' to get him identified.

This is also true with occlusion MI – a new term to denote a near or total occlusion with insufficient coronary circulation.

- ☐ **Epicardial current of injury so produced over the subepicardial zone in occlusion MI might inscribe ST elevation , somewhat different in distribution from that of classical STEMI.**
- ☐ **This 'Precordial Swirl Sign' exhibits basically a rightward ST-segment elevation vector with elevation in V1 and aVR and reciprocal ST-depression in V5-V6.**

This is a dire emergency to realise this concept , otherwise this occlusion MI might be missed , it may end the life of the patient.

1. Introduction

There are certain occlusion myocardial infarction patterns which don't show the classical pattern of STEMI and therefore , by keeping only STEMI concept in mind one may be misdirected in arriving at the diagnosis of acute coronary artery occlusion.
In 2018 , Meyers , Weingart and Smith put a new concept of 'occlusion myocardial infarction' (OMI) in the place of STEMI equivalent – a more reliable paradigm for detecting acute coronary occlusion.
Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation resulting in acute myocardial infarction - to be diagnosed in the absence of traditional ST elevation on ECG remains a 'Herculean task'.

Classical STEMI is diagnosed as a group of symptoms consistent with acute coronary syndrome in the presence of new ST-segment elevation at J-point in at least two anatomical continuous leads of $\geq$ 2 mm in a male or $\geq$ 1.5 mm in a female over leads V2-V3 and/or at least 1 mm in other continuous leads or limb leads , in the absence of bundle branch block , LVH , or other non - MI ST-segment elevation situations.

In the month of october 15 , 2022 , there emerged a new pattern of occlusion MI , introduced by Smith and Meyers et al. – indicating LAD Occlusion proximal to the first septal perfomating branch S1 , what is known as 'Precordial Swirl Sign'.

2. What is Precordial Swirl Sign ?

As per Smith and Meyers et al. the precordial swirl sign illustrates the following facts :

- ☐ This occlusion MI is the resultant of **septal ischemia** , including the corresponding anterior wall.
- ☐ This entity is recognized on ECG by ST elevation in leads V1 and aVR. There is associated reciprocal ST depression in leads V5 and V6.
- ☐ Since precordial swirl sign is dynamic in nature , evolving hyperacute T-wave changes are also included therein :
  'ST-segment elevation and /or hyperacute T-waves in V1-V2 and reciprocal ST depression and /or T-wave inversion in V5-V6'.

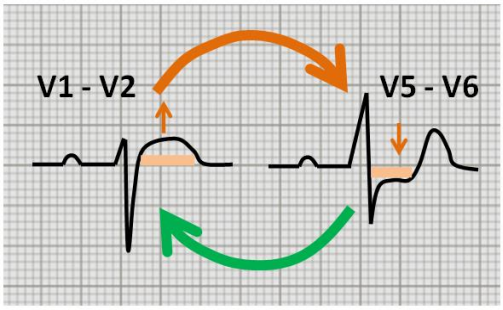


Fig 1.1

ST segment changes swirling across precordial leads :
ST elevation V1-V2 → depression V5-V6 , hence the name

3. Electrophysiological mechanism – a consideration

This 'Precordial Swirl Sign' denotes septal ischemia and therefore, the following facts should be considered to have its proper insight :

- **Blood supply of the concerned interventricular septum**

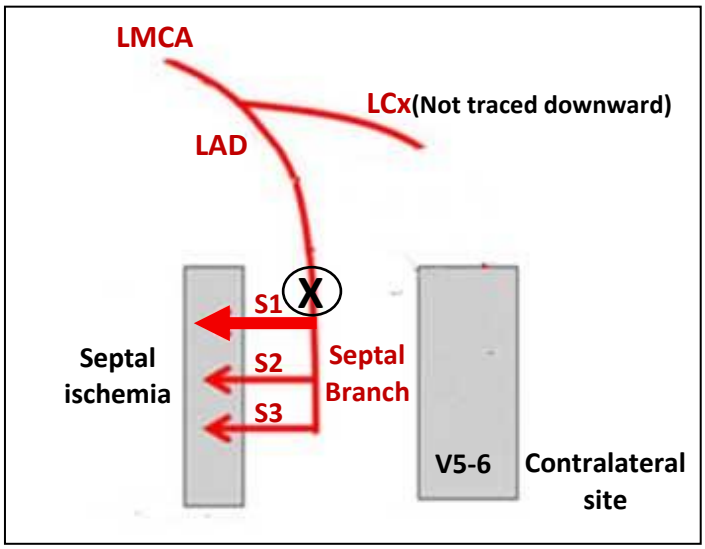


Fig 1.2

The site of lesion in precordial swirl sign is proximal to 1st septal perforating branch (**S1**) of LAD, as illustrated above.

The anterior 2/3rd interventricular septum is supplied by the proximal left anterior descending (LAD) through septal branches (S1, S2, S3)

Why S1 is critical --

S1 is the first and largest septal branch of its septal group , supplying mostly V1-V2 territory. So , the septal ischemia of S1 territory (near total occlusion) is the earliest –

with most obvious ECG changes as ST elevation in V1-V2 territory , with reciprocal ST depression in contralateral leads V5-V6.

The concept of current of injury

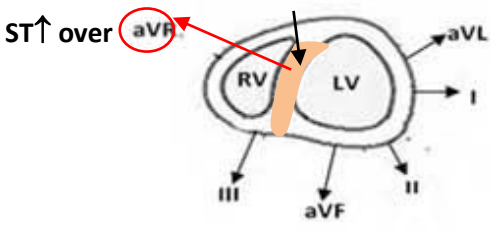
Since the current of injury is at the subepicardial region , ECG changes are reflected as ST segment elevation over the concerned leads within the range of 90^0, as the flow of current is towards the exploring electrode And the reciprocal ST depression are recorded on the contralateral side over leads V5-V6.

Concept of Rule of 90^0

> Any exploring lead placed within the range of 90^0 in respect to ST vector records ST elevation , at 90^0 no deflection and beyond 90^0 ST depression.

A concept of ST elevation with reciprocal ST depression in context with Precordial Swirl Sign

Subepicardial aspect over the interventricular septal ischemia – S1 is the first and largest septal branch of this group , mainly supplying V1±V2 territory



ST vector is directed posteriorly and rightward towards lead aVR due to obliquity of interventricular septum towards the right

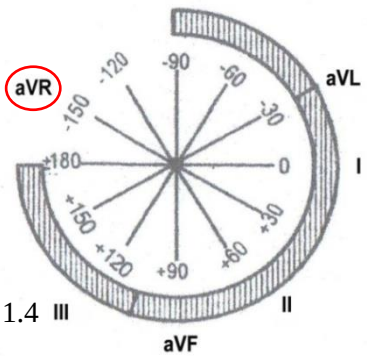


Fig 1.3 **HORIZONTAL PLANE**

Fig 1.4 **VERTICAL PLANE**

V1 is in alliance with interventricular septal injury (within the range of 90^0) → ST elevation therein

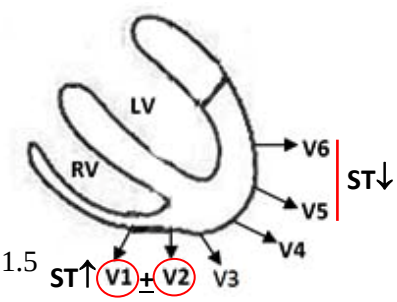
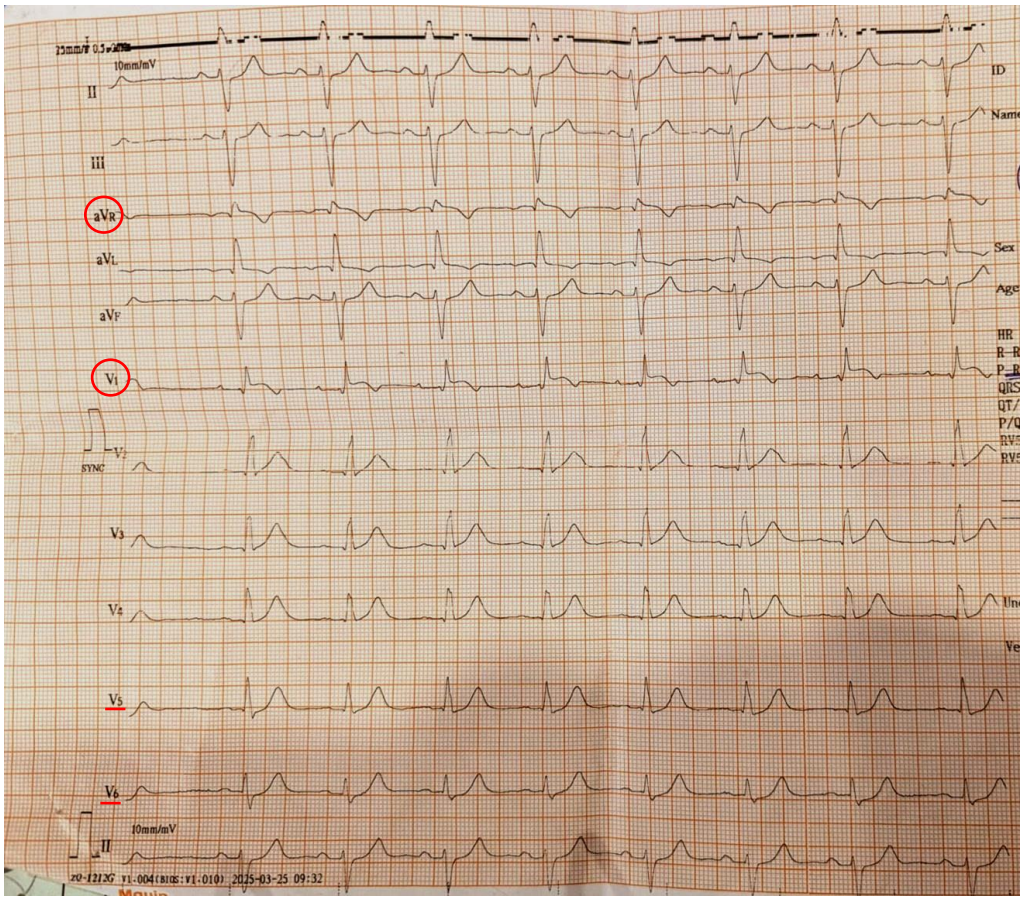


Fig 1.5 **HORIZONTAL PLANE**

- Since here the subepicardial injury is proximal to septal branch S1 of LAD, the basal part of interventricular septum is jeopardized with the resultant vector pointing towards lead aVR with ST elevation therein.
- Since V1 lies in alliance with interventricular septal injury , it also records the ST elevation.
- Since precordial leads V5-V6 are facing the contralateral region over the opposite side, these leads record reciprocal ST depression.

4. An interesting Case

87 years old male was admitted with the complaint of chest pain assocaited with ghabrahat (unease feeling) and sweating on 25.03.2025 with the following ECG :



Source : Dr. Satish kumar , Senior Consultant Physician and Cardiologist , Wellmark Hospital , Bokaro

Findings on ECG :

- ☐ ST elevation in aVR and V1.
- ☐ Reciprocal ST depression in leads V5 and V6.

Coronary angiography

Left ant. desc. Proximal long segment 80% occlusion (near total occlusion) , other coronary arteries are normal

PTCA : stent to LAD done

Discussion :

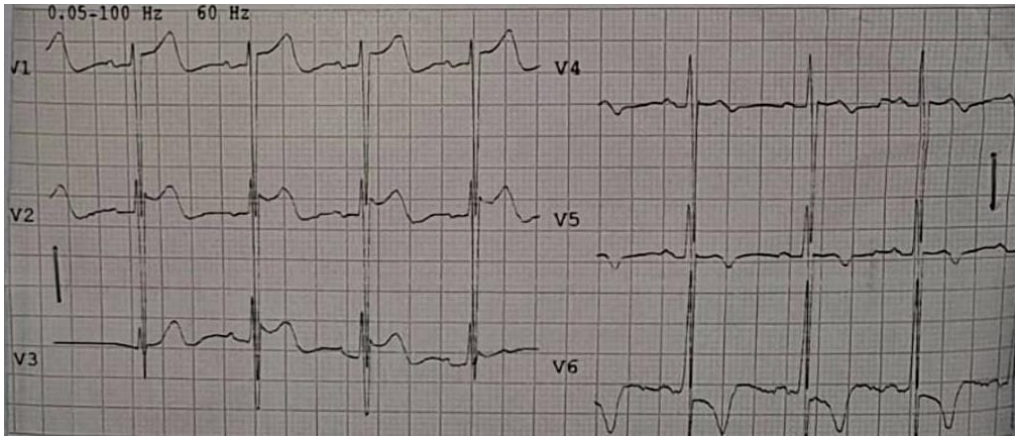
- On this ECG, there is ST elevation in V1 and aVR , with reciprocal ST depression in V5 and V6.
- This is attributed to the septal ischemia due to near total occlusion of LAD proximal by 80% and it was limited to V1.

Impression

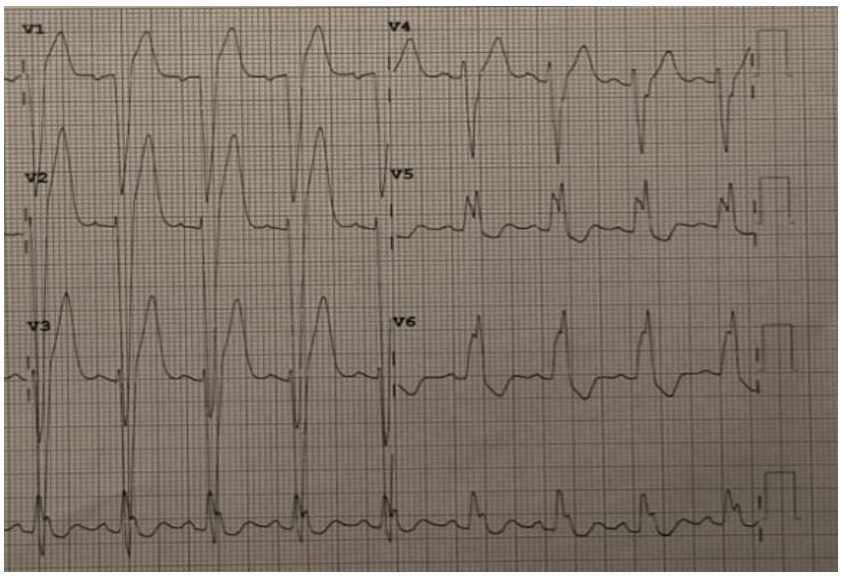
Based on the ECG findings and coronary angiography, this appears to be a case with a positive 'Precordial Swirl Sign'.

5. A mindfullness attention to the assoicated LVH , LBBB , etc to exclude the false impression of precordial swirl sign

- There may be the presence of ST depression in leads V5-V6 with contralateral ST elevation in V1 in cases of LVH and LBBB. Therefore , before commiting precordial swirl sign to be positive , it become essential to exclude these conditions.



Hypertensive patient
This is ECG dictum that discordant ST elevation over right precordial leads V1-3 , specially with up concavity in the presence of ECG evidences in favour of LVH should be treated as a part of LVH itself , not as Precordial Swirl Sign.



Left bundle branch block pattern is characterized by the wide notched QRS complexes in lead V5-V6 with secondary repolarization abnormality in the form of ST segment depression and T wave inversion. **The opposite pattern of a wide S wave with upsloping ST and upright tall T is seen in right precordial leads.**
This fact must be kept in mind while interpreting Precordial Swirl Sign.

(In both of these two conditions ECGs tracings are recorded only over the precordial leads)

NB : Other D/D : coronary lesion : LMCA / proximal LAD occlusion (presence of ST elevation in aVR and V1).

6. Take Home Message

- ☐ Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation resulting in acute myocardial infarction - to be diagnosed in the absence of traditional ST elevation on ECG remains a ‘Herculean task’

- In the month of october 15 , 2022 , Smith and Meyers et al. introduced a very useful sign to illustrate the proximal LAD myocardial occlusion (proximal to the first septal perforating branch – **S1**) – 'Precordial Swirl Sign'.
  This occlusion MI results in **septal ischemia**

- **'Precordial Swirl Sign'**
  - ST-segment elevation and /or hyperacute T-waves in V1-V2 and reciprocal ST-depression and /or T-wave inversion in V5-V6
    (The inclusion of hyperacute T-wave analysis with V1-V2 evolution offers a broader , more sensitive window into early anterior OMI – particulary when first septum (S1) involvement is present)

- **Reasoning** : ST vector is directed somewhat posteriorly and rightward towards lead aVR due to obliquity of interventricular septum towards the right – this fact is responsible for ST elevation in lead aVR. Since V1 lies in alliance with interventricular septal injury, it also records the ST elevation therein.
  Since precordial leads V5-V6 are facing the contralateral region over the opposite side, these leads record reciprocal ST depression.

- The Precordial Swirl Sign (PSS) is a sentinel warning – an early marker that occlusion MI has begun , but transmural infarction is not yet complete.

- Precordial Swirl Sign is never diagnosed in the presence of LVH and LBBB. There may be the presence of ST depression in leads V5-V6 with contralateral ST elevation in V1 in cases of LVH and LBBB. Therefore , before commiting precordial swirl sign to be positive , it become essential to exclude these conditions

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HIGH LATERAL OCCLUSION MI :

ECG PATTERN PARALLEL TO THE SUPERIOR INJURY VECTOR

HIGH LATERAL OCCLUSION MI :

ECG PATTERN PARALLEL TO THE SUPERIOR INJURY VECTOR

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OUTLINE

Introduction

LAD-D1 occlusion is associated with the following features on ECG

- ST elevation primarily localised to leads I , aVL (+/- V2)
- Reciprocal ST depression and/or T wave inversion in inferior leads, most prominent in lead III.

Etiology

Obstructive coronary artery (mainly)

- Acute atherosclerotic plaque rupture with subsequent thrombosis

Non-obstructive coronary artery (nearly around 6% of total cases)

Electrophysiology (concerned with high lateral MI)

In high lateral MI, the ST vector points towards anywhere in between 0^0 to -90^0 on the frontal plane. Running through the left and higher than the centre of the heart , the frontal plane projection of lead V2 is also pointing towards the same vector direction as that of leads I and aVL.

An interesting case discussion

Take Home Message

References

High lateral occlusion MI : ECG pattern parallel to the superior injury vector

A Narrative Review

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Knowledge is always rewarding — but only when the light is properly focused on the area of concern. A critical clue may remain unnoticed, slipping silently into the shadows of ignorance, if not observed with precision. This is how one can become entangled in the web of phantom ignorance, missing subtle signs for silence.

- ☐ **The high lateral wall — though small in area — can generate a superiorly directed injury current, producing subtle yet important changes on the ECG.**
- ☐ **If unrecognized, these changes may escape our attention, and a significant event may be dismissed as minor or missed entity.**

Clinicians recognize this unique ECG pattern with ST elevation in area , corresponding to LAD-D1 territory as a surrogate marker for high lateral MI.

1. Introduction : Keypoints

- High lateral MI is noticed as a specific pattern of ST elevation caused by acute occlusion of the first diagonal branch (**D1**) of left anterior descending artery , associated with the following features on ECG.

  - ST elevation primarily localised to leads I , aVL (+/- V2)
  - Reciprocal ST depression and/or T wave inversion in inferior leads, most prominent in lead III.

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

- This is considered to be the smallest area occupying MI , accompanied by chest pain and complications as occur with other MIs.
- In resource-poor countries lacking the facilities of PCI at the peripheral centres , the recognition of high lateral MI pattern on ECG is a simple and cost effective to have the selection of such patients for the earliest reperfusion therapy by thrombolysis and time honoured referral for PCI.

These keypoints highlight the importance of recognizing this unusual pattern on ECG for the prompt diagnosis of high lateral MI.

2. Etiology

More commonly , LAD-D1 occlusion causes high lateral MI due to the following factors :
Obstructive coronary artery (mainly).

- **Acute atherosclerotic plaque rupture with subsequent thrombosis**
- Failure of drug compliance by a patient with stent in situ resulting in restenosis

Non-obstructive coronary artery (nearly around 6% of total cases)

- Coronary artery spasm
- Myocardial bridging
- Spontaneous coronary artery dissection
- Coronary artery embolism (CAE) associated with : rheumatic valve disease (mitral stenosis) , atrial fibrillation, infective endocarditis, Atrial myxoma, etc.
- Autoimmune conditions such as Takayasu arteritis

3. Electrophysiology (concerned with high lateral MI)

The following points are to be considered to have a clear concept of high lateral MI on ECG.

- High lateral occlusion MI occurs usually due to occlusion of the first diagonal branch of LAD (D1) , at times high OM branch
- This acute infarction is localized to the superior portion of the lateral wall of the left ventricle (hence , the nomenclature high lateral MI) and thereby , resulting in superior injury current axis with ST elevation in leads I and aVL with reciprocal ST depression and / or T wave inversion in inferior leads , most prominent in lead III.
- Associated ST elevation in chest lead V2 is due to the fact that this lead corresponds to the left and higher up with respect to the centre of the heart. This causes lead V2 projection onto the frontal plane directed towards the superior injury current axis , registering ST elevation also in this lead.

The entire concept is illustrated by the sketches , as below :

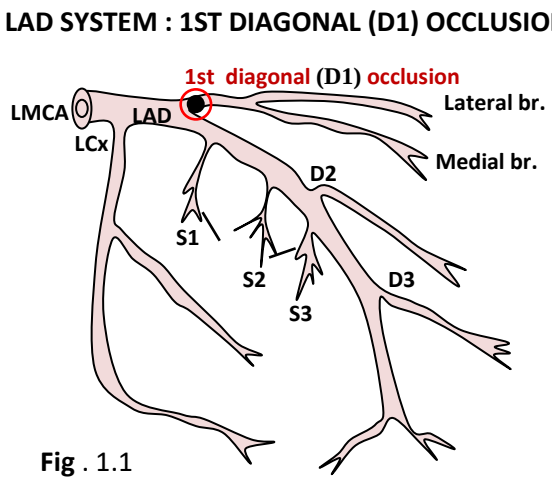


Fig . 1.1

Diagonal branches are usually denoted as D1, D2, D3 , etc. and variable in number (often 2 to 9). The first diagonal branch (D1) tends to be most prominent ; its occlusion is an obstruction just distal to the point of its origin from LAD , as illustrated.

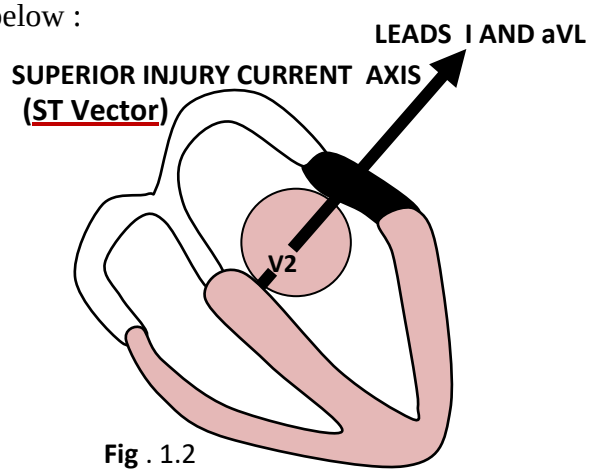


Fig . 1.2

HIGH LATERAL MI WITH SUPERIOR INJURY CURRENT AXIS (ST Vector)

BASIC PRINCIPLE OF ELECTROPHYSIOLOGY : essential to understand ST vector in high lateral MI

- Initially the current of injury is directed towards the site of myocardial infarction revealed as ST segment elevation.
- The hallmark of acute injury is ST segment elevation which is usually accompanied by reciprocal ST segment depression.
- In a patient with acute ST segment elevation with no otherwise cause for this , the simultaneous presence of reciprocal ST segment depression has a specificity and positive predictive value of 93% for the diagnosis of acute myocardial infarction.

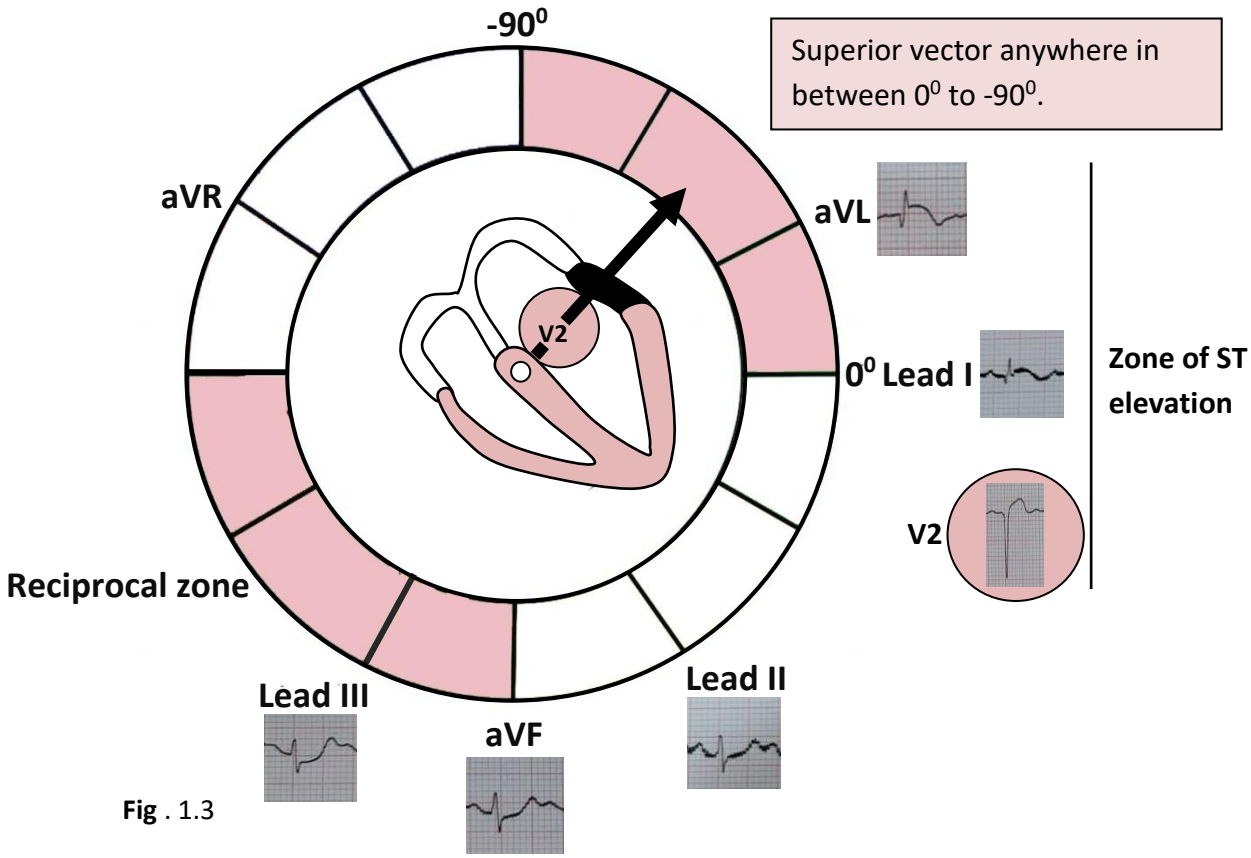


Fig . 1.3

A model to understand ST vector in high lateral MI with its changes on ECG

In high lateral MI , the ST vector points towards anywhere in between 0^0 to -90^0 onto the frontal plane. Running through the left and higher than the centre of the heart , the frontal plane projection of lead V2 is also pointing towards the same vector direction – having the ST vector elevation in this lead also.

✓ Now it becomes easier to understand the concept of high lateral MI on ECG – characterised by ST elevation in leads I , aVL and V2, accompanied by reciprocal ST depression in inferior leads , most prominent in lead III.

With this common upward direction of ST vector the leads I , aVL and V2 are considered as lying in a contiguous alignment in respect to the vector. Therefore , these leads are in reality the ‘contiguous leads’ in respect to high lateral STEMI – a very specific pattern on ECG.

4. An interesting case discussion

History : 56 years male admitted with acute chest pain and sweating.
Mild lateral hypokinesia with LVEF 50% on cardiac echo. The concerned ECG is as below :

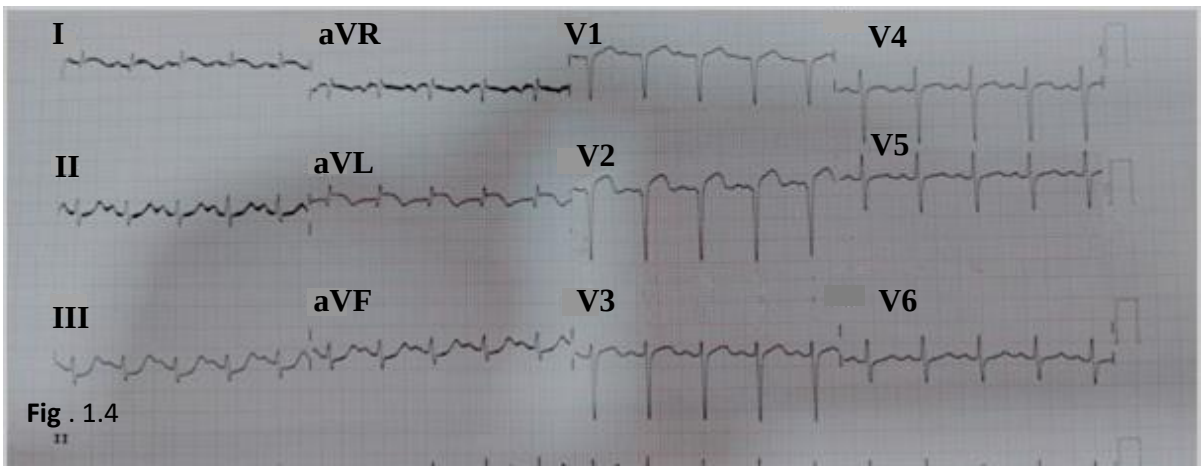


Fig . 1.4

Source : **CME INDIA** , Feb 13 2023 by **Dr. Satish Kumar** , Senior Consultant Physician and Cardiologist , Bokaro

Interpretation : The following ECG changes are suggestive of high lateral myocardial infarction

- ST elevation primarily localised to leads I, aVL and V2
- Reciprocal ST depression in inferior leads , most prominent in lead III

The findings on this ECG are plotted over South African flag as a very specific sign.
This should be easier to memorize the ECG changes of high lateral MI by putting all these over the South African flag. It also imparts a concept that ST elevation seen in V2 is running with the frontal plane in the same direction with that of common ST vector (leads I and aVL).

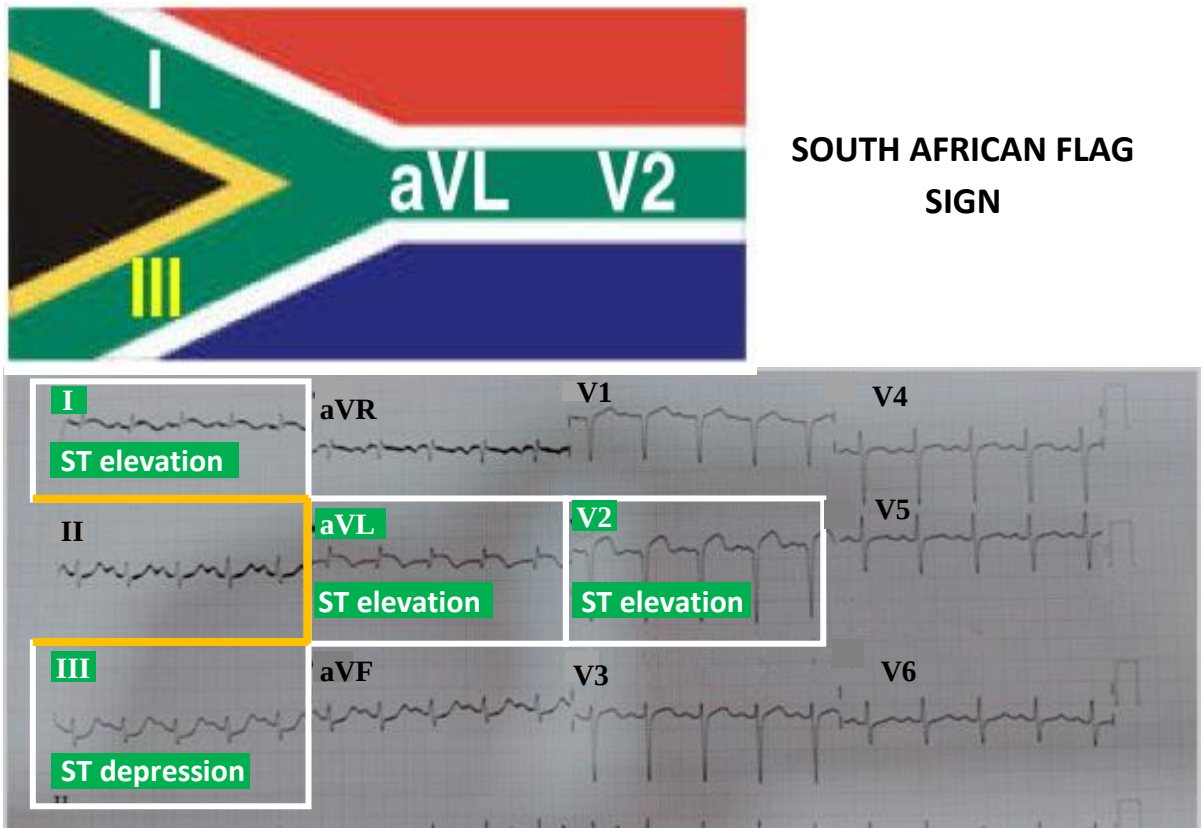
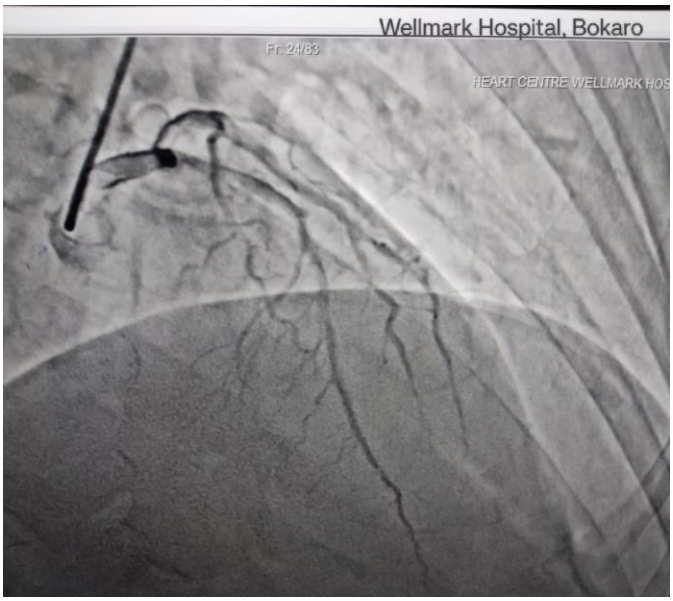


Fig . 1.5

Further to say

Coronary angiography (CAG) of the same patient shows the site of occlusion at D1 (thrombotic)



D1 occlusion is an obstruction distal to the point of its origin from LAD (Please see also figure 1.1 in this context)

Fig . 1.6

The same patient post PCI at D1

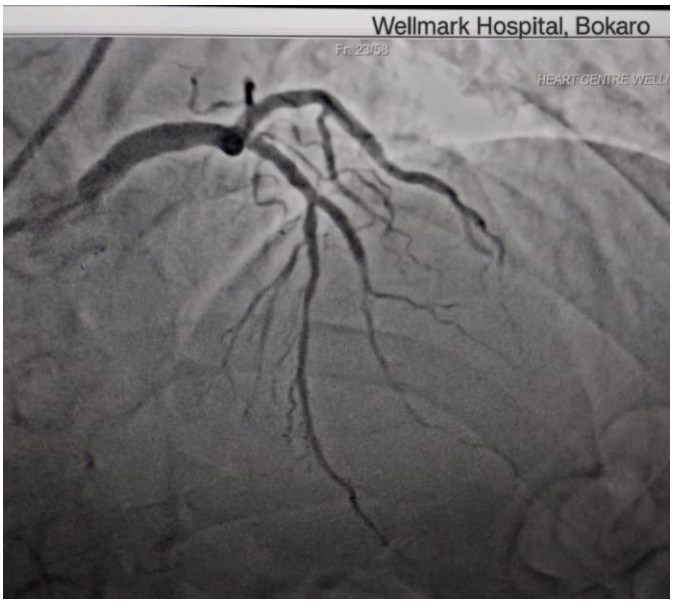


Fig . 1.7

Source : **CME INDIA** , Feb 14 2023 by **Dr. Satish kumar** , Senior Consultant physician and Cardiologist , Bokaro

5. Take Home Message

- In a patient with symptoms suggestive of acute anginal pain with ST elevation in leads I , aVL and V2 with reciprocal ST depression in inferior leads , most prominent in lead III on ECG – thereby indicating high lateral occlusion MI.

- High lateral occlusion MI occurs usually due to occlusion of the first diagonal branch of LAD (D1) , at times high OM branch
- This acute infarction is localized to the superior portion of the lateral wall of the left ventricle (hence , the nomenclature high lateral MI) and thereby , resulting in superior injury current axis with ST elevation in leads I and aVL with reciprocal ST depression and / or T wave inversion in inferior leads , most prominent in lead III.
- Associated ST elevation in chest lead V2 is due to the fact that this lead corresponds to the left and higher up with respect to the centre of the heart. This causes lead V2 projection onto the frontal plane directed towards the superior injury current axis , registering ST elevation also in this lead.
- These very specific findings associated with this occlusion MI help a lot in the selection of such patients for the earliest reperfusion therapy by thrombolysis and time honoured referral for PCI , specially in poor-resource settings. The cluster of these ECG findings can be plotted over South African flag as a very specific sign.

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POSTERIOR OCCLUSION MI :

RECIPROCAL MIRROR REFLECTION ON ECG

POSTERIOR OCCLUSION MI : RECIPROCAL MIRROR REFLECTION ON ECG

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OUTLINE

Introduction
A posterior wall MI occurs when posterior myocardial tissue is caught in its fire – this area is represented by a dorsal , infra-atrial portion of the ventricle

Electrocardiographic changes in PMI

- **Background basis**
  The anteroseptal leads (V1-V3) are visualized to witness the reciprocal mirror changes occurring over the posterior surface of the ventricle
- **Electrocardiographic criteria of PMI on standard 12-lead ECG**
  ST depression , prominent R-wave and upright T-wave limited to anteroseptal leads.
- **ECG changes in context with posterior leads V7-9**
  The direct changes may appear to be much smaller than the reciprocal changes in leads V1-V3. Here special emphasis should be given to the amplitude of ST elevation.

An interesting case study

Take Home Message

References

Posterior occlusion MI : reciprocal mirror reflection on ECG

A Narrative Review

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We often remain unnoticed of what approaches us from behind — until it steps onto our direct view. Place a mirror in front, and suddenly, the unseen becomes seen. This is the impact of reflection: it reveals what's hidden, anticipates the unexpected to our vision. Such is the power of mirror image reflection.
The same holds true with posterior wall myocardial infarction.

- ☐ **Posterior wall MI occurs when the posterior myocardial wall is caught in its fire**
- ☐ **This culprit is reflected onto the anteroseptal leads V1-V3 as reciprocal mirror reflection of the behind – thus the clinician catches the hidden scenario.**
  - **ST elevation becomes ST depression**
  - **Q wave becomes R wave**
  - **Terminal T-wave inversion becomes an upright T wave**

The diagnosis of posterior wall myocardial infarction is usually missed , it does not conform to the ST segment elevation pattern of traditional STEMI – hence , nomenclatured as 'Posterior occlusion MI.'

1. Introduction (keypoints)

- **Posterior View (Heart)**

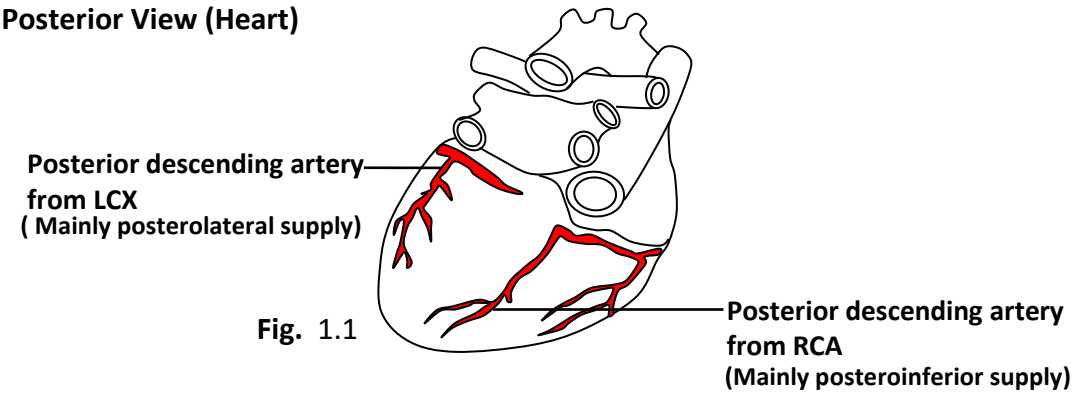


Fig. 1.1

A posterior wall MI occurs when posterior myocardial tissue is caught by its fire – this area is represented by a dorsal , infra-atrial portion. This occurs due to acute occlusion in blood flow through the posterior descending artery (PDA), a branch of RCA in the majority of affected population, the remaining through posterior descending artery as a branch of LCX.

If the posterior descending artery is supplied by the right coronary artery (RCA), then this coronary circulation is classified as **"right-dominant"** (In 85 % of cases).
If the posterior descending artery is supplied by the left circumflex artery (LCX), then the coronary circulation is classified as **"left-dominant"** (in 15% of cases).

- Posterior wall MI has been observed in 15-20% when compared to traditional STEMIs. There is a frequent involvement of the inferior wall or lateral wall due to the shared blood supply through the corresponding posterior descending artery.
  Isolated posterior MI is less common , with its prevalence more in men than women – 72% and 28% respectively (Isolated PMI often corresponds to the LCX territory).

- The diagnosis of posterior wall MI is usually missed or delayed , it does not conform to the ST segment elevation pattern of STEMI. This would be worthwhile to mention here that 12-lead ECG does not catch the involved posterior area directly –this myocardial infarction pattern is visualized through the anteroseptal leads V1-V3 as reciprocal mirror reflection.

- Rapid recognition of acute PMI is of much significance due to the following reasons :
  - PMI is a large area infarct with the increased risk of left ventricular dysfunction and even death.
    (Inferior or lateral extension indicates a larger area of ischemic insult , and these patients are at increased risk of complications related to MI).
  - In keeping view with its larger size the posterior MI is benefited more from the earlier reperfusion therapy.
    Isolated posterior infarction by itself is an indication for emergent coronary reperfusion. However , the lack of obvious ST elevation in this context may cause the diagnosis to be missed.
  - 22% of the patients had been observed to have moderate to severe mitral regurgitation as a result of the involvement of concerned papillary muscles.

NB : **If a patient comes with an evidence of inferior ± lateral STEMI , one should be very vigilant to search for the electrocardiographic evidence of posterior MI.**

- Here , risk factors are not different than those seen with other myocardial infarctions.

2. Electrocardiographic changes in PMI

☐ **Background basis :**

The anteroseptal leads (V1-V3) on 12-lead ECG are chosen for the detection of posterior wall myocardial infarction to be recorded as its reciprocal mirror reflection. In other words , it can be said that posterior electrical activity is recorded from the anterior side of the heart, as figured below :

- ST elevation becomes ST depression
- Q wave becomes R wave
- Terminal T-wave inversion becomes an upright T wave

There is usually associated evidence of inferior MI ± lateral MI due to the extension of PMI towards these areas.

The further confirmation of these changes can be obtained by placing the leads through V7-V9 (posterior leads), wherein the true image of posterior STEMI would be observed.

☐ **Illustration of PMI recording on ECG through V1-V3 as reciprocal mirror changes :**

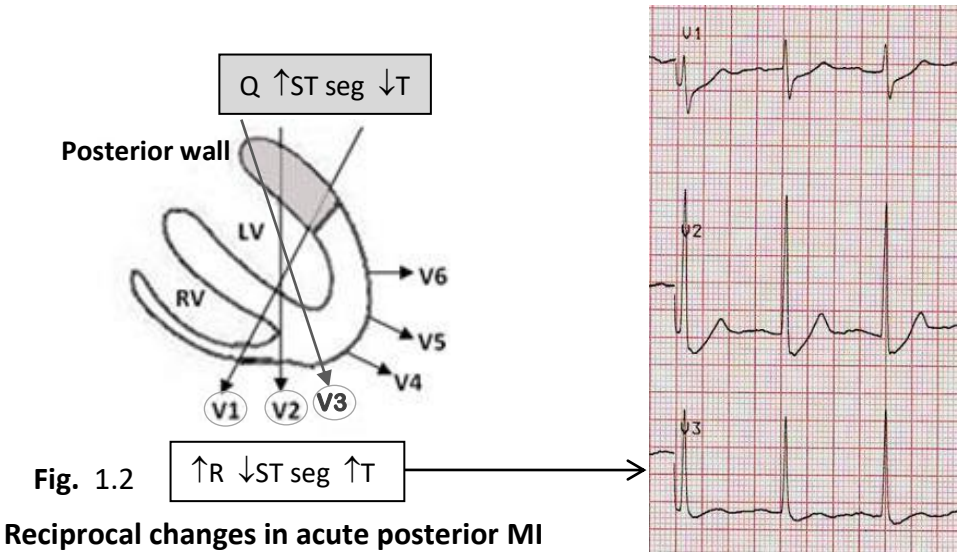


Fig. 1.2

Reciprocal changes in acute posterior MI on V1 V2 V3

Electrocardiographic criteria of PMI on standard 12 lead ECG :
(The anteroseptal leads V1-V3 record reciprocal mirror changes of the posterior wall).

- The ST depression is often deep (>2mm) and flat (horizontal >> downsloping / upsloping).
- R-wave with increased amplitude and duration (i.e. , "a pathological R-wave" is a mirror image of a pathological Q-wave).
  R/S ratio in V1 or V2 > 1 (i.e., prominent anterior forces)
  These prominent R-wave in V2-3 greater than those observed in V4-6.
- Prominent-upright T-wave (a noticeable difference between the voltage of the T-waves in leads V2 and V6 → T in V2 >T in V6. If the value of the T2 to T6 index equals or extends the value of 0.38 mV, then the possibility of PMI is more than likely.
- There may be subtle (or overt) signs of an inferior ± lateral MI in addition.

NB : It is essential to keep the following facts in mind :

- If **Posterior MI** occurs with **inferior MI → think RCA -PDA**
- If **Posterior MI** occurs with **lateral MI → think LCX –PDA**
- **Think of dominant proximal LCX occlusion** if changes occur in the favour of posterior + lateral + inferior MI together on ECG. This is a high risk situation since it involves the larger area of myocardium and thus , it may present with hypotension , bradycardia (if AV node involved) , heart failure or arrhythmias.
- If the anteroseptal leads denote a mirror image of the posterior wall , then the concerned ECG may be turned upside-down showing the tall anterior R-waves becoming deep posterior Q waves , the ST-depression becoming ST elevation and upright T-waves becoming terminal T-wave inversion but this is to be kept in mind that one cannot use this trick to rule out posterior STEMI with a safe predictability.

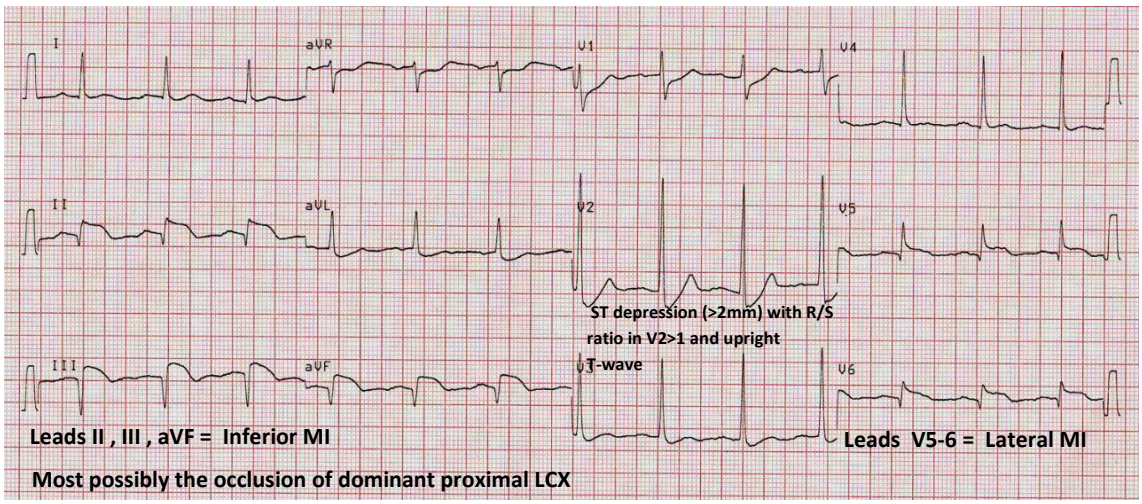
- The combination of horizontal ST depression with upright T-wave limited to leads V1-V3 in the absence of concurrent R-wave might be observed in the early phase of evolution of PMI. Here the diagnosis remains in question , which needs further confirmation by placing the leads over V7-9.
- This fact should also be kept in mind that late normalization of ST-T in V1-V3 may also be seen.

☐ **Explanation of ECG changes in posterior leads V7-9**

Sites of leads V7-9 placement	
• V7 – Left posterior axillary line, in the same horizontal plane as V6. • V8 – Tip of the left scapula, in the same horizontal plane as V6. • V9 – Left paraspinal region, in the same horizontal plane as V6.	Lead cable V4 should be placed to V7 Lead cable V5 placed to V8 Lead cable V6 placed to V9
Additional findings (V7-9) ☐ ST elevation consistent with posterior MI (V7-9) > 0.05 mV (>0.1 mV in men older than 40 years of age). ☐ The direct changes may appear to be much smaller than the reciprocal changes over leads V1-3.	

3. An interesting case study

History : Middle aged man presenting as sudden severe chest pain since 4 hours with ↓ECG



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

- Dominant R-wave with ST depression in V1-V3 , more than 2 mm ST↓ in V2 plus accompanying upright T-waves in these leads.
- ECG shows Q waves with ST elevation in II , III , aVF (ST in II > III) and also over V5-6. These findings are suggestive of proximal dominant LCX occlusion.

Comment : Most possibly the occlusion of dominant proximal LCX

4. Take Home Message

- A posterior wall MI occurs when the posterior myocardial tissue is caught in its fire - this myocardial infarction pattern is visualized through anteroseptal leads V1-V3 as reciprocal mirror changes.
- The clinicians should concentrate over ECG findings such as ST-segment depression with upright T-waves and prominent tall R-waves limited to V1 to V3 while interpreting the posterior wall MI.
- Posterior territory is rich in collaterals from surrounding vascular beds (RCA-LCX). This reduces the chance of a complete isolated transmural infarction until the occlusion is very proximal and collaterals are poor.
- Using posterior leads (V7-9) in patients presenting with suspicious symptoms for MI would reveal more patients with PMI – with more benefit coverage by early reperfusion therapy.
- If a patient comes with an evidence of inferior ± lateral STEMI , one should be very vigilant to search for the electrocardiographic evidence of posterior MI.

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ASLANGER PATTERN ON ECG : A TWIN SUFFERING CRY

ASLANGER PATTERN ON ECG : A TWIN SUFFERING CRY

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OUTLINE

Aslanger pattern on ECG : A twin suffering cry

A Narrative Review

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Two persons walking together in the morning were suddenly injured , each with a different impact of injury on their bodies – both cried together with a single merged voice of pain.
The same is true with 'Aslanger pattern' on ECG.

- ☐ **On one side there is evidence of acute inferior occlusive MI and on the other side the evidence of subendocardial ischemia , both are present on the same ECG.**
- ☐ **There is a single resultant ST vector from these two sites of injury – A twin suffering cry**

Dr. Aslanger et al. group was the first to recognize this entity on ECG , aiming at emergent reperfusion therapy to rescue the life of the patient.

1. Introduction

The April year 2020 had witnessed a new era when Aslanger et al. identified a specific ECG entity dealing with acute inferior occlusion MI in patients with concomitant subendocardial ischemia resulting from critical lesion (s) in coronary arteries other than the infarct-related artery – surprisingly to say this does not display contiguous ST-segment elevation to fulfil the classical STEMI criteria with this acute inferior occlusion MI (International guidelines include STE in two contiguous leads for localizing individual site of infarction). This is a new ECG pattern seen in 6.3% sufferers of so misdiagnosed as Non-STEMI and the sufferer may not have the emergent reperfusion therapy to rescue his life.

2. Illustration by ECG , as below

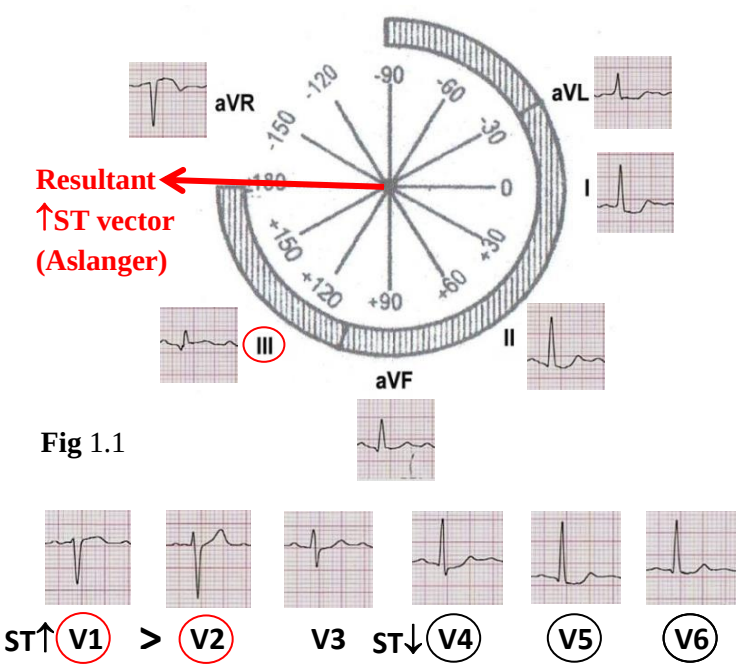

Fig 1.1

Source : CME INDIA , dated 13.01.2021 by Dr. Satish kumar Sr.Consultant Cardiologist , Bokaro (here in modified form)

Essential :
Aslanger pattern on ECG

- ST↑ only in lead III as evidence of inferior MI
- ST ↑ in V1>V2
- Concomitant ST↓ in V4-V6, with a positive/terminally positive T-wave

Resultant ST vector (Aslanger)

There are two ST vector forces – one from inferior occlusive MI and the other from subendocardial ischemia. The resultant ST vector is directed rightwards causing ST elevation in lead III only , sparing the other inferior leads II and aVF.

3. Electrophysiology with Aslanger pattern

There is a twin injury in the myocardium of the heart due to coronary insufficiency on two different sites with different nature :

- **On one side** there is evidence of acute inferior occlusive MI , inscribing a separate ST vector of its own injury on ECG , which is directed inferiorly rightward , as indicated by the dotted arrow Ⓐ .
- **On the other side** there is concomitant subendocardial ischemia resulting from critical lesion(s) in coronary arteries other than the infarct related artery , inscribing its own separate ST vector of injury , which is directed towards the lead aVR irrespective of involved coronary artery , as also indicated by dotted arrow Ⓑ
- **The resultant average ST vector (Aslanger) :**
  The summation of these two vectors results in a net direction towards $\pm 180^0$. In other words to say , this net vector translates the heart's electrical response into a single vector.

☐ **Step 1 (plotting of resultant ST vector – Aslanger on hexagonal lead system)**

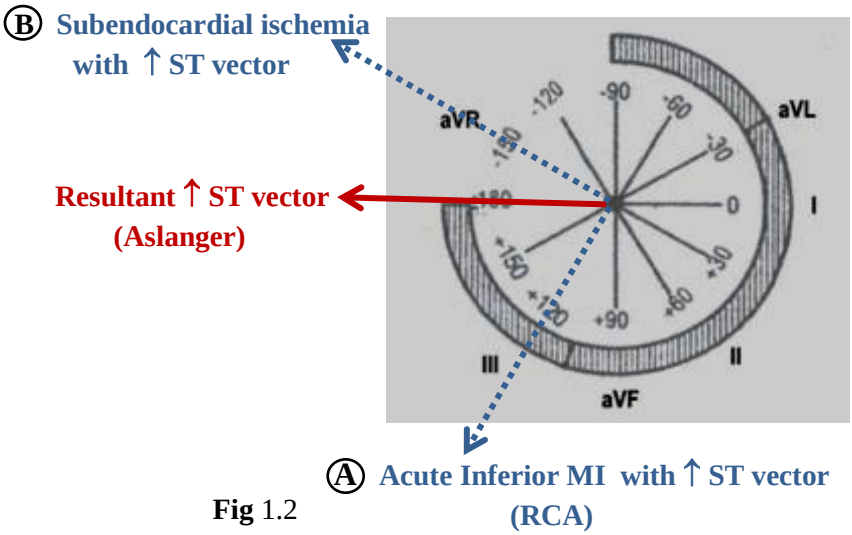

Fig 1.2

☐ **Step 2 (ST elevation vs ST depression as per direction of the resultant vector)**

> **A simplified approach**
> Any exploring lead placed within the range of 90^0 in respect to resultant average ST vector records ST elevation , at 90^0 no deflection and beyond 90^0 ST depression.

Since resultant ST vector is passing through $\pm 180^0$ of hexagonal lead system , this records ST elevation within 90^0 of its range as ST↑ in leads III and aVR (and away over the opposite side – ST↓ in leads aVL , I and II).

Right sided lead V1 is having greater proximity to lead aVR , compared to lead V2 → ST↑ V1 > V2. On the contralateral side ST depressions might be recorded in precordial leads V4-V6 without disturbing the positive T-wave polarity.

4. Historical background

This becomes essential to understand at least in brief the findings of the study done by Aslanger et al. , which may be summarized as below :

This retrospective study included

Group 1 : 1000 patients with a diagnosis of Non-STEMI

Group 2 : Two subsets
one with inferior STEMI and the other with 1000 as a healthy control group with no myocardial infarction

- 6.3% in Non-STEMI group was identified as having Aslanger pattern.
- 13.3% of inferior MIs also showed the Aslanger pattern.
- 0.5% from healthy subsets (No MI) was also identified to have this pattern as well.

As a whole the patients having Aslanger pattern with acute inferior MI were found to have a larger infarct size as documented by 24 hours troponin estimation and higher frequency of angiographic culprit lesion (s) with complex coronary occlusion compared to their Non-STEMI counterparts.

5. Limitations

- ☐ Aslanger et al. group also identified this sort of pattern on ECG in 0.5% from control group (No MI) possibly due to chronic changes from a previous ischemic insult .
- ☐ The presence of previous infarctions in association with acute inferior MI might have changed the direction of ST vector resulting in a similar pattern.

This is to be mentioned here that study done by Aslanger et al. was a retrospective study which warrants a further perspective study with its proper analysis to explain the whole scenario.

6. Clinical significance

- ☐ The recognition of this Aslanger pattern ECG by the clinicians means early institution of emergent reperfusion therapy and thus , this improves the outcome.
- ☐ Due to the presence of concomitant multivessel coronary occlusions it becomes difficult to identify the culprit lesion (s) at the time of angiography. This pattern on ECG guides the interventional cardiologist to open inferior wall culprit vessel first and accordingly the others as per need.

7. Take Home Message

☐ Aslanger et al . defined a new ECG pattern having acute inferior myocardial infarction pattern with concomitant critical lesion (s) in coronary arteries other than the infarct-related artery. Recognizing this entity would be saving the life of the sufferer by the institution of emergent reperfusion therapy. This would also improve short-and-long term complications associated with this entity.

- Essential: Aslanger Pattern on ECG
  - ST↑ only in lead III as evidence of inferior MI
  - ST ↑ in V1>V2
  - Concomitant ST↓ in V4-V6, with a positive/terminally positive T-wave
- **The resultant average ST vector (Aslanger) :** the summation of these two vectors results in a net direction towards $\pm 180^0$. In other words to say , this net vector translates the heart's electrical response into a single vector.
- There is every possibility that such entity might be misdiagnosed as Non-STEMI and the sufferer may not have the emergent reperfusion therapy to rescue his life.
- To keep in mind : when the ECG reveals this twin pattern of ST elevation with ST depression , as discussed , the heart is warning through such scenario , one must not miss this.

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DECODING OCCLUSION MI IN THE PRESENCE OF LBBB :

A DIAGNOSTIC APPROACH

DECODING OCCLUSION MI IN THE PRESENCE OF LBBB : A DIAGNOSTIC APPROACH

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OUTLINE

Introduction
To make the diagnosis of occlusion myocardial infarction (OMI) in patients exhibiting left bundle branch block (LBBB) places a challenging diagnostic task to the attending clinician.

Consideration of ST-elevation in context with LBBB and occlusion MI

The basic electrophysiology in combined scenario of acute MI with LBBB
Practice has now emerged to assess for concordant ST elevation/depression ± excessive discordant ST segment elevation indicative of occlusion MI in the presence of LBBB

Outline of different diagnosis criteria for acute MI in the presence of LBBB

- ☐ The origianal Sgarbossa criteria
- ☐ The Smith-modified Sgarbossa criteria
- ☐ Barcelona criteria
- ☐ Champan's sign
- ☐ Cabrera's sign

Stepwise interpretation of ECG having acute MI in the presence of LBBB

Interesting cases

Take Home Message

References

Decoding Occlusion MI in the Presence of LBBB : A Diagnostic approach

A Narrative Review

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When the heart stumbles with occlusion MI under the shadowy imprint of LBBB, it becomes difficult to catch the real culprit red-handed. Really to say that there are many lacunae on the ECG that may hide the diagnostic clues, making it difficult to unearth the true image

- ☐ **The disruption of normal ventricular conduction in LBBB alters repolarization pattern , making a challenging task to diagnose occlusion myocardial infarction (OMI) in its presence.**
- ☐ **Timely diagnosis of occlusion MI in the presence of LBBB with precision opens the gateway to a better patient outcome**

There had been some researchers who had tried their best to curtain off the mist of this ignorance – to save the suffering humanity from this dilemma.

1. Introduction (Keynotes)

- To make the diagnosis of occlusion myocardial infarction (OMI) in patients exhibiting left bundle branch block (LBBB) places a challenging diagnostic task to the attending clinician.
- This diagnostic dilemma happens so due to the repolarization abnormality – encountered in both LBBB and acute occlusion MI
  - ECG with LBBB normally have a distinct ST elevation mainly over the right sided precordial leads
  - The occlusion MI , traditionally called STEMI is also having a particular pattern of ST elevation on ECG , as per involvement of coronary territory
- The presence of LBBB in a patient with symptoms suggestive of CAD had been conventionally considered as an ECG equivalent to ST-segment elevation MI and accordingly the 2017 European Society of Cardiology guidelines still recommend emergent reperfusion in such cases. However , increasing clinical evidence suggests that LBBB is a major cause of false activation of the cath lab. In view of this evidence there runs an opinion that LBBB should not be considered as a diagnostic norm of AMI in isolation.
- These facts put a diagnostic mist to the attending clinician what to do under the prevailing circumstances – occlusion MI in the presence of LBBB may not receive timely repurfusion therapy with potential consequences to the suffering patient.

2. Consideration of ST-elevation in context with LBBB and occlusion MI

What about the ST elevation ?

- **LBBB** causes ST elevation over the right sided precordial leads - due to the fact that right ventricular repoarization occurs earlier than that of left ventricular repolarization , keeping the right one somewhat non-synchronized to that of left one. That's why , while left sided ventricular leads exhibit ST depression , right sided precordial leads show somewhat ST elevation.
- **Occlusion MI** (=STEMI for the purpose) in isolation causes epicardial injury , the axis of repolarization is directed towards the injured epicardial surface resulting in ST elevation over the leads as dictated by the involvement of concerned coronary territory.

Sequence of conduction in LBBB

- ☐ When LBBB is present the septum depolarizes from right to left and not from the left to right (as indicated by **red arrow 1**) → loss of the normal septal r wave in lead V1, and in normal septal q wave in lead V6 .
- ☐ Then via the right branch (as indicated by **red arrow 2**) to reach the right ventricle.
- ☐ The impulse then passes around blocked left bundle to activate LV (as indicated by **red arrow 3**)

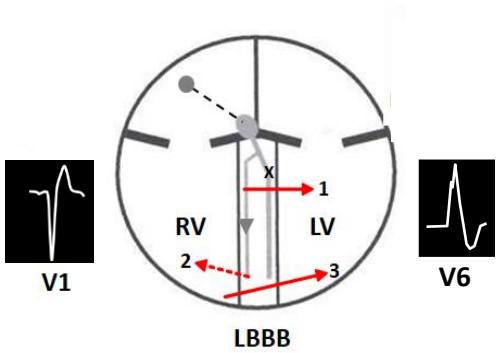


Fig. 1.1

To be noted that impulses from right ventricle (RV) pass somewhat slowly through the interconnecting myocardial mass to the left ventricle (LV) with consequent widening of depolarization wave (QRS) – this is known as **'abnormal depolarization'** in context with LBBB (since this occurs through the abnormal path of depolarization).

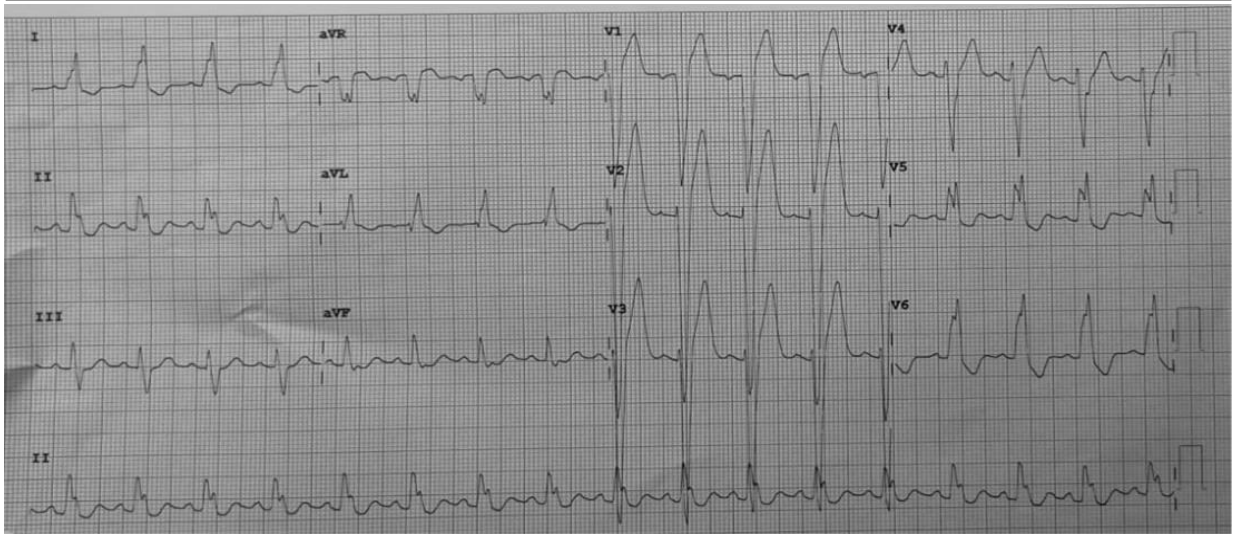
Here it would be worthwhile to mention about **'appropriate discordance'**

- ➢ Appropriate discordance means that abnormal depolarisation is followed by abnormal repolarisation, which appears discordant to the preceding QRS complex , as illustrated above with V1 and V6 leads.
- ➢ Lateral leads with slurred, broad R waves will often have associated ST-segment depression and T-wave inversion, and those with deep S waves over the rt sided precordial leads can have a somewhat amount of ST elevation that does not indicate ischemia (generally accepted as normal < 25% of the size of the preceding S wave)

However , this appropriate discordance can mask the signs of associated acute MI either by much ST depression or excessive ST elevation. This scenario may keep the clinicians in diagnostic dilemma.

ECG diagnostic criteria in LBBB (essential to know)

- QRS duration ≥ 120 ms
- Dominant S wave in V1
- Broad slurred R wave in lateral leads (I , aVL , V5-V6)
- Absence of q waves in lateral leads
- Prolonged R wave peak time > 60 ms in leads V5-6



3. The basic electrophysiology in combined scenario of acute MI with LBBB

The purpose of writing this article is to sort out the way how ST segment behaves in acute MI with LBBB.

The electrophysiological rule governing appropriate discordance in LBBB has been well discussed in the previous page. It is also well known fact that the occlusion MI , traditionally called STEMI is also having a particular pattern of ST elevation on ECG , as dictated by the site of coronary involvement. The combined impact of both the situations creates on ECG an altered ST pattern to be accounted for.

The basic electrophysiological concept is outlined below :

Practice has now emerged to access for concordant ST elevation/depression ± excessive discordant ST segment elevation indicative of myocardial infarction in the presence of LBBB

This practical outline had been adopted by some researchers – as to diagnose acute MI in the presence of LBBB , initially by Dr. Elena Sgarbossa in 1996 and further laid down with some modification by Dr. Stephen Smith in 2012.

✓And the history rolls over – Barcelona et al added their additional observation by adopting a new algorithm – discordant ST deviation ≥ 1 mm in any lead where the R or S is ≤ 6 mm – seems to be very useful index with additional MI where the QRS is having somewhat low amplitude.

4. Outline of different diagnosis criteria for acute MI in the presence of LBBB

☐ **The original Sgarbossa criteria**

This original criteria included the three clues to diagnose infarction in patient with LBBB :

- **Concordant ST elevation > 1mm** in any lead with a positive QRS complex (score 5)
- **Concordant ST depression > 1 mm** in V1-V3 (score 3)
- **Excessively discordant ST elevation > 5 mm** in leads with a negative QRS complex (score 2)

A cumulative score of 3 or more provides a diagnostic criteria for the purpose , having sensitivity (36%) and specificity (90%).

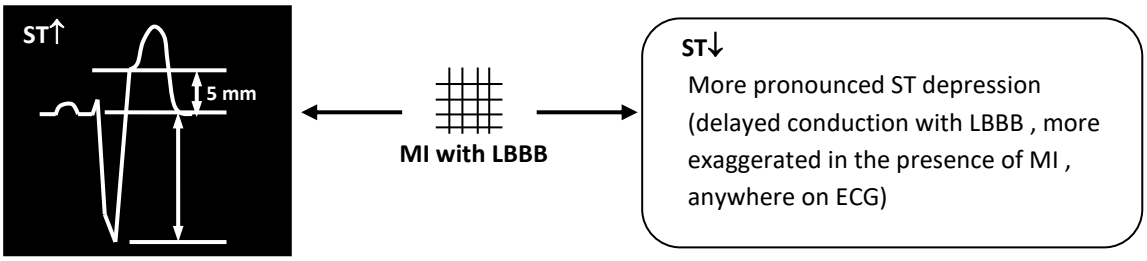
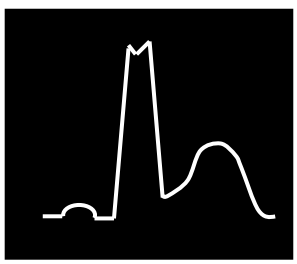


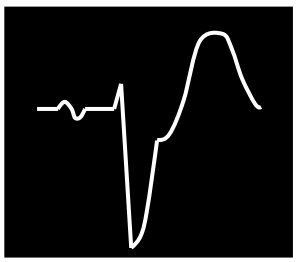
Fig. 1.2

ST-segment depression and elevation are having reverse relationship with each other. More ST depression on one side would be recorded as more ST elevation over the opposite side. Here this has been sketched to clarify the mechanism of **excessive discordant ST elevation** .

NB : Concordant ST elevation or depression is not the characteristic of alone LBBB – this occurs usually in the presence of additional MI. The smallest unit of 1 mm is used as a denominator as > 1 mm



Concordant ST elevation > 1mm in any lead with a positive QRS complex



Concordant ST depression > 1 mm in V1-V3 with negative QRS complex

Fig. 1.3

☐ **The Smith-modified Sgarbossa criteria**

- **Concordant ST elevation** ≥ 1 mm in ≥ 1 lead
- **Concordant ST depression** ≥ 1 mm in ≥ 1 lead of V1-V3
- ***Proportionally*** **excessive discordant STE** in ≥ 1 lead anywhere with ≥ 1 mm STE, as defined by ≥ 25% of the depth of the preceding S-wave

Stephen W.Smith modified the third diagnostic element of original Sgarbossa criteria , mentioned above as the proportionally excessive discordant STE. This yields more sensitive (80%) and specific (90%) criteria for OMI diagnosis – all ECG findings are evaluated better with proportional excessive discordant ST elevation , discarding many false positives and false negatives of the original absolute 5 mm criterion.

☐ **Barcelona criteria :** It is based on symptoms plus troponin level (troponins levels barely higher than the upper reference limits) but lacking the fundamental evidence of angiographic confirmation of coronary occlusion as primary outcome.

- Concordant ST elevation in any lead ≥ 1mm
- Concordant ST depression in ***any*** *lead*
- Discordant ST deviation ≥ 1mm in any lead where the R or S is ≤ 6mm

➢ As with the prior 2 criteria, criteria 1 is the same. Criteria 2 is expanded to all leads (not just V1-3). Criteria 3 is a bit unique.

➢ For criteria 3 , one is looking at low voltage.

➢ Barcelona criteria is claimed to have the sensitivity and specificity 93% and 94% respectively by internal validation. This criteria development was fatally flawed and the data is not good and also does not meet face validity. There is one good aspect to it : concordant ST depression, otherwise its use in clinical practice meets lots of issues.

☐ **Chapman's sign**

- The presence of a notch in the ascending limb of the R-wave in leads I , aVL and/or V6 is called Chapman's sign
- This sign is attributed to abnormal / delayed conduction through the LBBB , which is further aggravated in the presence of damaged cardiac muscle.
- It is one of the criteria that is also used to diagnose acute MI in the presence of LBBB
- This sign may also be seen in some patient of dilated cardiomyopathy or any cause of left ventricular dilatation with dysfunction.
- This has a low sensitivity , but specificity of about 90%.

Cabrera's sign

- This sign indicates notching in the ascending limb of the S-wave in leads V3 and V4 (and sometimes V5). The notching must be at least 40 ms in duration which has a low sensitivity for MI around 27%.
- The same reasoning as that of Chapman's sign – abnormal conduction of electrical signal through the damaged myocardium in the presence of LBBB has been attributed to this sign.

5. Stepwise interpretation of ECG having acute MI in the presence of LBBB

Most importantly , one must understand that no ECG criteria would be 100% true ever to identify AMI with LBBB and therefore, it becomes essential to seek the maximum potential of the ECG to identify occlusion MI in this context

The following steps are adopted while interpreting the ECG with acute MI in the presence of LBBB :

Step 1 : Evaluation of symptoms of acute MI

Step 2 : To identify LBBB , as diagnostic criteria has been laid down on page 3

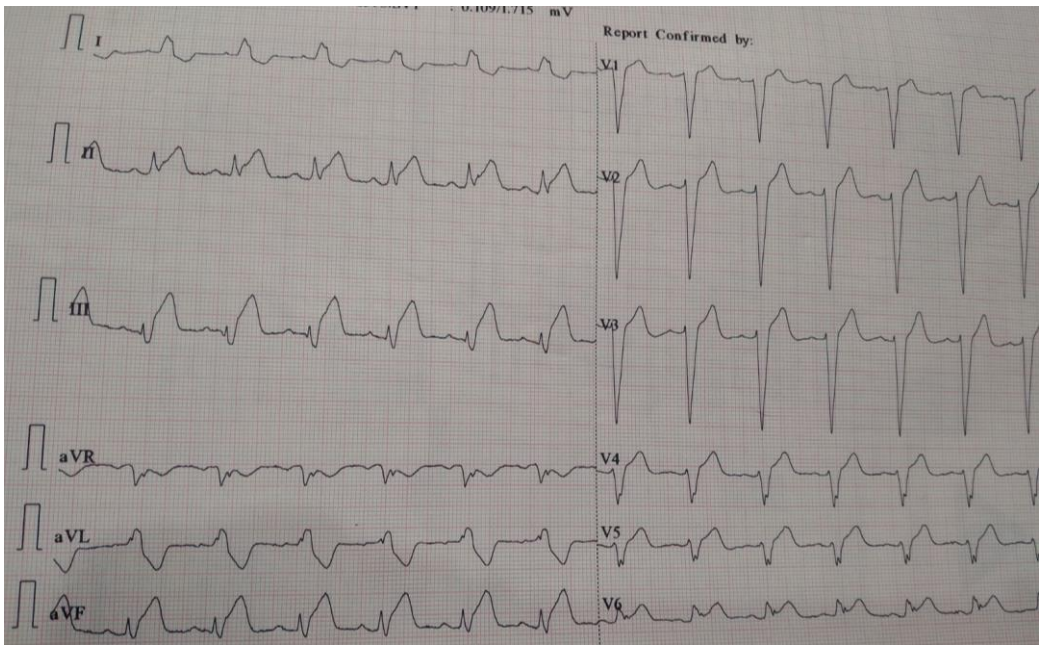
Step 3 : To consider the implementation of different criteria , as enumerated below (In nutshell) :

- ***The original Sgarbossa criteria***
  Sensitivity (36%) and Specificity (90%)
- ***The Smith modified Sgarbossa criteria***
  Sensitivity (80%) and specificity (90%) by the addition of proportional excessive discordant STE
- **Barcelona Criteria**
  There is one good aspect to it : concordant ST depression, otherwise its use in clinical practice meets lots of issues
  Ref :
  Barcelona Rule on Left Bundle Branch Block: Lots of Issues.
  November 25, 2020
  https://hqmeded-ecg.blogspot.com/2020/11/barcelona-rule-on-left-bundle-branch.html
- To see the presence of Chapman's sign and Cabrera's sign in the concerned leads if present.

6. Interesting cases

☐ **Case 1**

61 years male , non-diabetic and non-hypertensive presents with severe chest pain and sweating having BP 150/90 mmHg , pulse 80 bpm



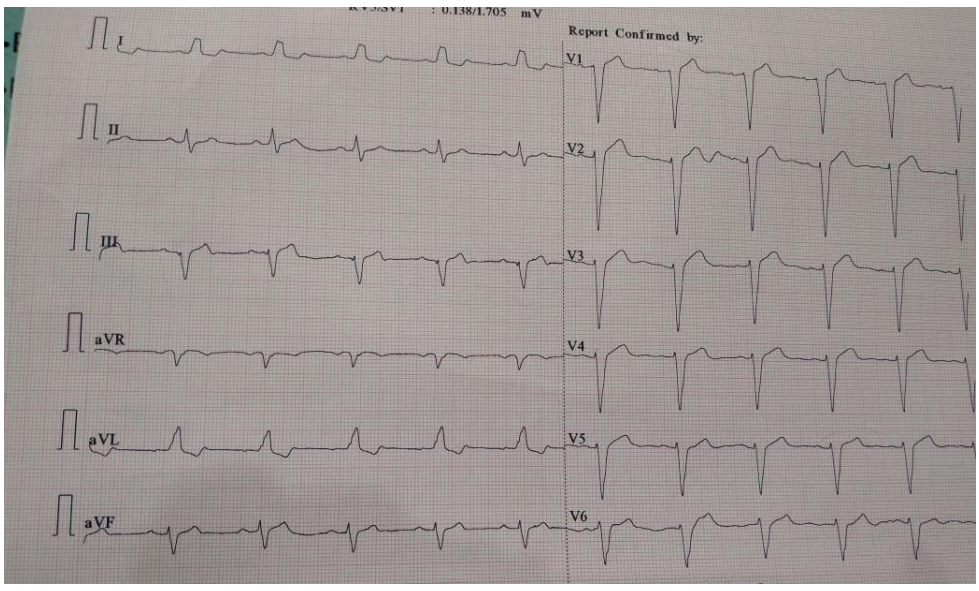
Source : GLOBAL HEART RHYTHM FORUM put by Dr. R.K Gupta , Senior Consultant Physician , Yamunanagar , Haryana on dated 04.10.2024

ECG findings : In the background of complete LBBB

- Concordant ST elevation ≥ 1 mm in lead II , aVF and V6
- Barcelona sign positive in lead V5 (discordant ST elevation of 1 mm and where S wave <6 mm)
- Cabrera's sign positive in lead V4 with a notch on the ascending limb of the S-wave with duration of 40 ms (with the maximum zooming over lead V4)

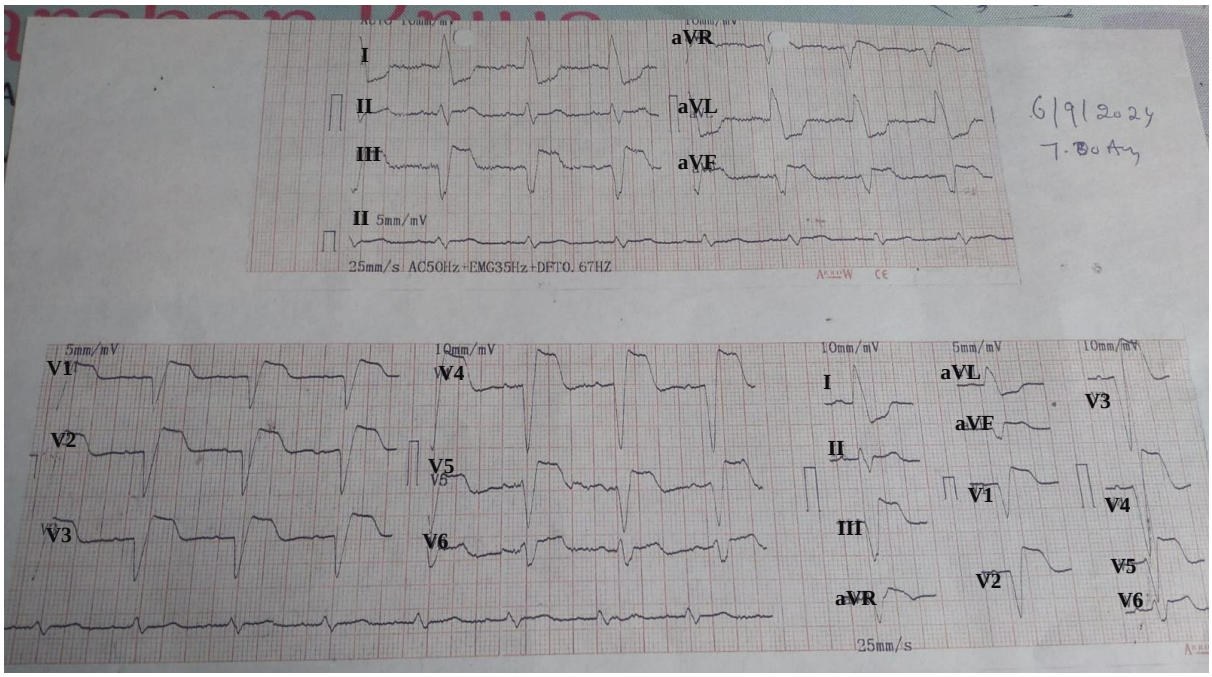
His CAG shows occlusion in RCA and LAD.

On thrombolysis the following is the ECG of the same patient (with pre-existing LBBB)



☐ **Case 2**

60 years female presenting with chest pain

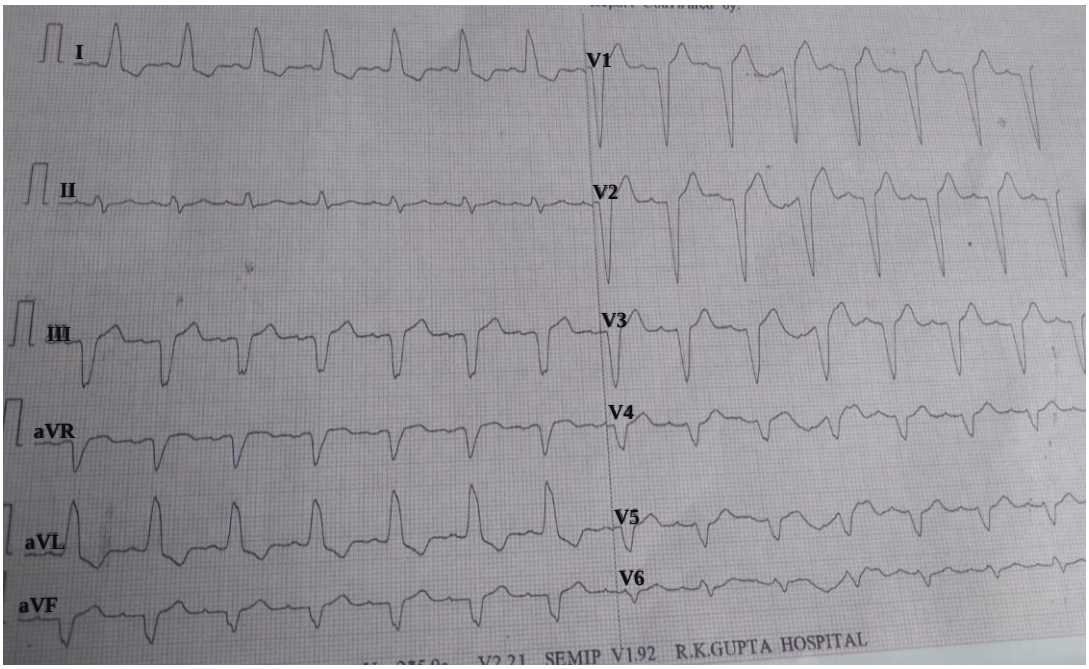


Source : GLOBAL HEART RHYTHM FORUM put by Dr. R.K Gupta , Senior Consultant Physician , Yamunanagar , Haryana on dated 09.10.2024

ECG findings :

- The smith modified Sgarbossa criteria is positive from V1 to V6 (propotionally discordant STE) in these leads with $\geq$ 1 mm , as defined > 25% of the depth of the preceding S-wave
- Barcelona sign positive in lead V6 (also positive over aVR and aVF). (Discordant ST deviation of 1mm where S wave is $\leq$ 6 mm)

Post-thrombolysis ECG of the same patient



7. Take Home Message

- ☐ Both the Sgarbossa and Smith criteria are approved for use to diagnose AMI in LBBB
- ☐ The Barcelona criteria are new (published in 2020). This criteria is claimed to have the sensitivity and specificity 93% and 94% respectively.
- ☐ The Barcelona criteria have been derived and internally validated with promising results. However, it awaits external validation (see page 73 for comments on this criteria).
- ☐ As always, when applying any clinical criteria, always consider the patient's pretest probability.
- ☐ No single criteria is suffice enough to diagnose acute MI in the presence of LBBB. It is not essential for all components of a diagnostic criteria to be positive when diagnosing occlusion MI in the presence of left bundle branch block (LBBB). However, the sensitivity of these criteria can remain limited, meaning that a negative result doesn't definitively rule out MI.
- ☐ Wackers et al. correlated ECG changes in LBBB with localization of the infarct by thallium scintigraphy. The most useful ECG criteria were:

    - Serial ECG changes — 67 percent sensitivity
    - ST segment elevation — 54 percent sensitivity
    - Abnormal Q waves — 31 percent sensitivity
    - Cabrera's sign — 27 percent sensitivity, 47 percent for anteroseptal MI
    - Initial positivity in V1 with a Q wave in V6 — 20 percent sensitivity but 100 percent specificity for anteroseptal MI

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21. Referred to ChatGPT whenever needed

TERMINAL QRS DISTORTION SIGN (TQRSD) : A RED ALTERT ON ECG

ECG

TERMINAL QRS DISTORTION SIGN (TQRSD) : A RED ALERT ON ECG

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OUTLINE

Introduction

The terminal QRS distortion is assessed on ECG by the loss of both S-wave and J-notch/slur with uplifting of the remaining R wave above its baseline usually with > 50% , at least with the precordial leads V2 / V3.

Always to keep in mind that terminal QRS distortion (TQRSD) is 100% specific to proximal LAD occlusion

Electrophysiology of 'Terminal QRS distortion'

- The terminal portion of the QRS complex represents late activation of the uppermost portion of interventricular septum and posterobasal free wall of LV→depolarizing at the end because the wavefront is flowing away from the apex upward → Normal S-wave genesis.
- Proximal LAD occlusion → ischemia involving basal-anteroseptal wall→ loss of S-wave and J-notch/slur
- **The result :** the QRS appears truncated as if someone 'cut off the S-wave' and glued the ST segment directly to the R-wave.

How to proceed to detect this sign on ECG

A special attention to leads V2 and V3 to see the evidence of terminal QRS distortion sign

An interesting case

Take Home Message

References

Terminal QRS Distortion Sign (TQRSD) : A red alert on ECG

A Narrative Review

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MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

This terminal QRS distortion sign , at times noticed during the evolution of acute anterior STEMI at least over leads V2/V3 in the presence of ongoing anginal chest pain , places a storm warning –this altered waveform bears silent witness to hidden, imminent danger—a testament to the heart's vulnerability towards a rapidly evolving STEMI.

- **Terminal QRS distortion is defined as loss of both S wave and J-point notch/slur with simultaneous uplifting of the remaining R-wave above its baseline.**
- **Its presence in the precordial leads has been strongly correlated with a rapidly progressing STEMI in the territory of proximal LAD occlusion , marking an extensive area of myocardium at risk with an urgent need for revascularization.**

This sign is very much specific (100%) for proximal LAD occlusion and whispers to the ears of attending physician to have urgent reperfusion therapy with improved outcome.

1. Introduction (Keynotes)

- At times during the evolution of acute Anterior STEMI (ST Elevation Myocardial Infarction) on 12-lead ECG, one can witness the terminal S-wave beginning to rise above the baseline with the loss of J-point notch , seen at least with leads V2/V3. **When the terminal S-wave rises above the baseline , then it is technically considered no longer S-wave**. This phenomenon of terminal QRS distortion is almost 100% specific for rapidly evolving STEMI in the territory of proximal anterior descending artery.

In association with the loss of S-wave with J-point notch as discussed above , the remaining R-wave may take on as a qR configuration , as an imprint of ongoing myocardial insult with its uplifting usually with >50% above its baseline : (qR is not universal , rather it is a subtype)

A concept of evolving 'terminal QRS distortion' , as illustrated below :

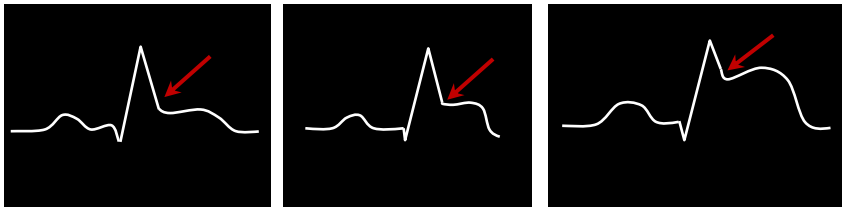


Fig. 1.1

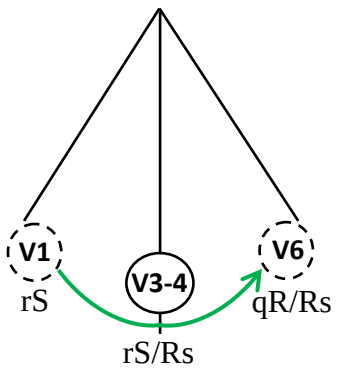
This loss of the terminal S-wave means the normal late activatation of the basal-anteroseptal wall is missing.

- Terminal QRS distortion on ECG can be summarized as below :
  At least in precordial leads V2/V3
  - No distinct S-wave (the QRS complex does not dip below the baseline after the R wave)
  - No J-point notch or slur at the end of the R wave
  - Direct merging of the remaining QRS complex with the ST segment
- 'Terminal QRS distortion increases all-cause mortality by 81%. The concerned study suggests that terminal QRS distortion is an important bedside tool to assess the risk in patients with STEMI' .
- Rapid and reliable diagnosis of terminal QRS distortion sign is critical for the urgent initiation of life-saving reperfusion therapy. Patients with symptoms suggestive of ongoing myocardial ischemia in the presence of TQRSD sign on ECG usually need urgent reperfusion therapy, as timely honoured reperfusion therapy reduces morbidity and mortality of patient with STEMI. A higher mortality rate has been observed with increasing time to primary percutaneous intervention.

- Since terminal QRS distortion is associated with up concavity , it might be confused with early repolarization syndrome (ERS) but this should be always remembered that this sign is never observed in ERS. Its rapid diagnosis with simultaneous exclusion of ERS is beneficial to the patient , as it may pave a way to earlier reperfusion therapy with improved outcome.
  Always to keep in mind that terminal QRS distortion (TQRSD) is 100% specific to proximal LAD occlusion

2. Electrophysiology of 'terminal QRS distortion'

- This would be better to witness how normally QRS complex progresses over the precordial leads from V1-V6 so that the mechanism of QRS distortion over V2-V3 is properly visualized and analysed.



Normal R wave progression

Normally the height of R wave becomes progressively taller from lead V1 through V6

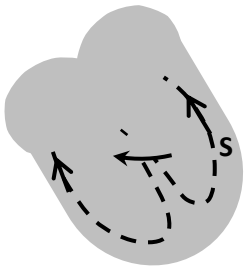
- V1-V2 : R wave of low amplitude and S wave of more depth (R/S <1)
- V3-V4 (Transition zone) rS/Rs
- Onwards R wave amplitude is gradually more compared to S wave → qRs/Rs in leads V5-V6.

Fig. 1.2

- As illustrated in Fig. 1.2 in the preceding page there is one pertinent point that needs attention – the dominance of the voltage across the precordial leads fluctuates from right to the left as increasing amplitude of R-wave with decreasing amplitude of S-wave , considering the site on the precordium where R and S waves approximate each other as the transition zone. It is truly to state that at times the electrical signal cannot flow smoothly through the QRS complex , the terminal S wave inscription may not have the isoelectric baseline – how ? see below :

- Leads V2-V3 play a crucial role in the ECG assessment due to its location and sensitivity to ischemic changes in context with proximal LAD occlusion. This is to be mentioned here that expected changes over V2-V3 appear irrespective of the site of proximal LAD occlusion :
  - Proximal to the diagonal branch (D1)
  - In between the first diagonal (D1) and first septal (S1) branches

- **The following pertinent steps are enumerated and discussed in brief to visualize the concept of 'terminal QRS distortion' sign :**



Genesis of normal S-wave

Fig. 1.3

- The terminal portion of the QRS complex represents late activation of the uppermost portion of interventricular septum and adjoining posterobasal free wall of LV→depolarizing at the end with negative deflection because the wavefront is flowing away from the apex upward → Normal S-wave genesis.
- Proximal LAD occlusion → ischemia involving basal-anteroseptal wall → loss of S-wave.and J-notch/slur
- **The result :** the QRS appears truncated as if someone 'cut off the S-wave' and glued the ST segment directly to the R-wave.

NB : The basal anteroseptal wall is uniquely dependent upon the proximal LAD for its supply.

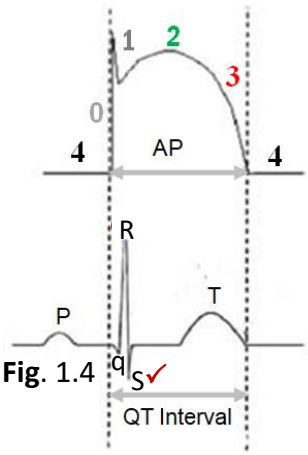
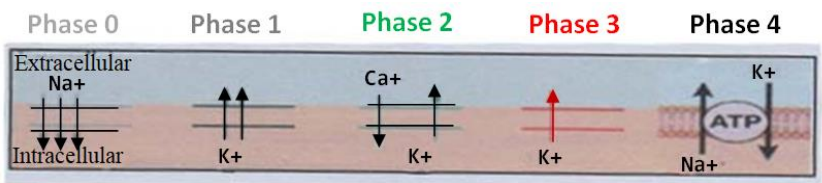


Fig. 1.4



Phase 1 (Nomally phase 1 dictates the genesis of J-point).

Terminal QRS Distortion also clogs the J-point . That's why , there is no J-notch or slur at the end of the R wave (the very distinct point in differentiating this from early repolarization syndrome where J notch or slur is present at the end of the QRS complex)

Now it becomes easier to grasp the definition of terminal QRS distortion – loss of both S wave and J-notch/slur with uplifting of the remaining R wave above its baseline usually with > 50% , at least being present over the precordial leads V2/V3.

3. How to proceed to detect this sign on ECG

QRS distortion is an electrocardiographic (ECG) sign of ongoing critical myocardial ischemia in context with proximal LAD occlusion. One should proceed to evaluate this association between the degree of QRS distortion and myocardium at risk.

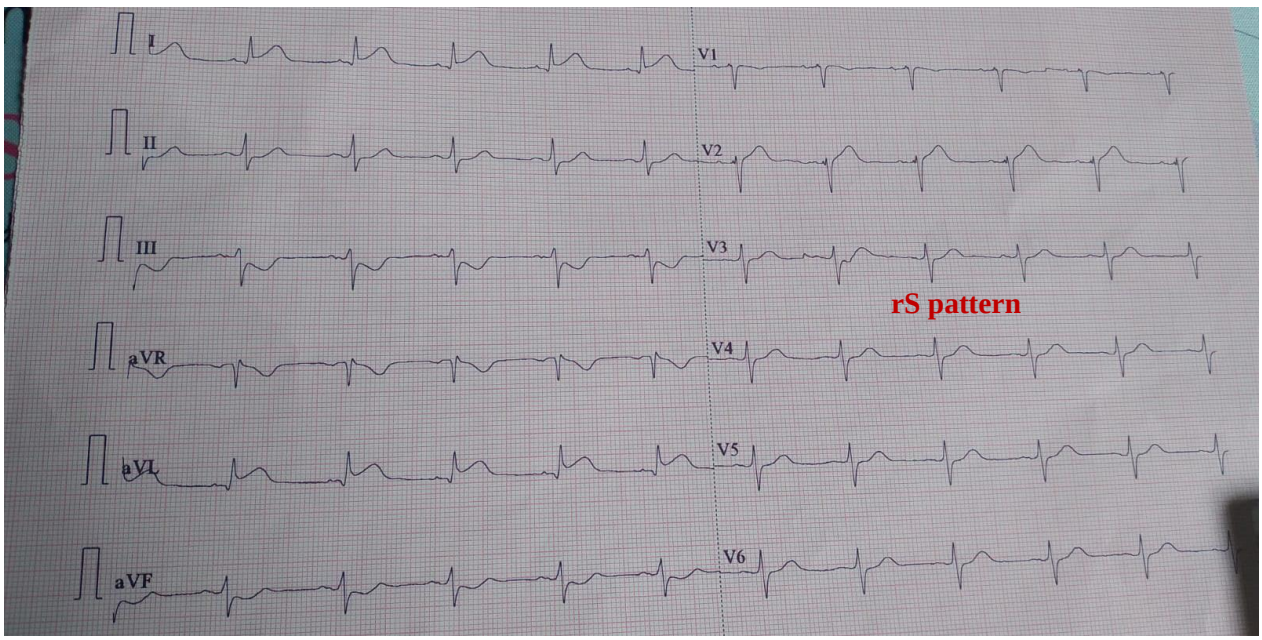
- At this juncture , it is advisable to review all 12-leads on ECG with an aim to detect evidence of ongoing anginal chest pain.
- A special attention to leads V2 and V3 to see the evidence of QRS distortion sign as already discussed.
- And the clinician should always adhere to a strict regiment for each patient under such prevailing circumstances :
    - Clinical correlation
    - Comparision with old 12-lead ECG tracings and old medical records if available.

4. An interesting case

56 years old female presented with chest pain (Non-diabetic, Non-hypertensive) with BP 130/80 mmHg , Pulse rate 80 bpm with hemodynamic stability.

There are **three ECGs recorded in succession one after the other to show the serial changes :**

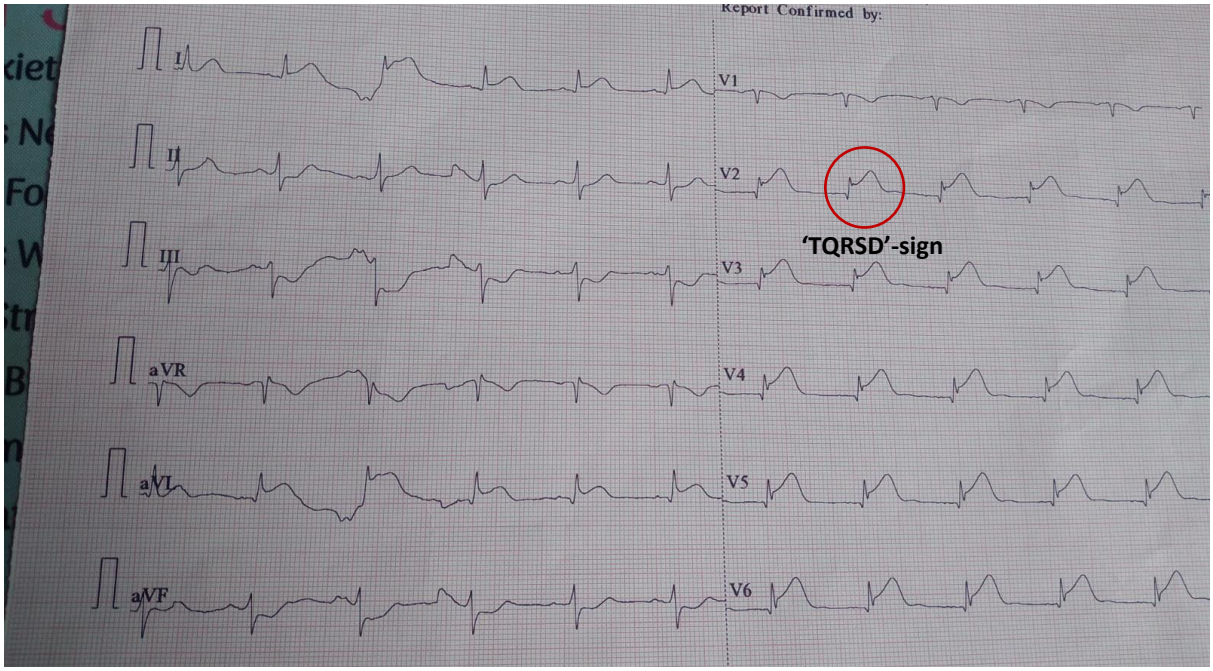
ECG 1



Source : Dr. R.K. Gupta , Senior Consultant Physician , Yamunanagar , Haryana
(On Global Heart Rhythm Forum dated 01.11.2024)

Findings : ST elevation in leads I and aVL with reciprocal ST depression in inferior leads (maximum over lead III) , associated rS pattern from V2-V6.

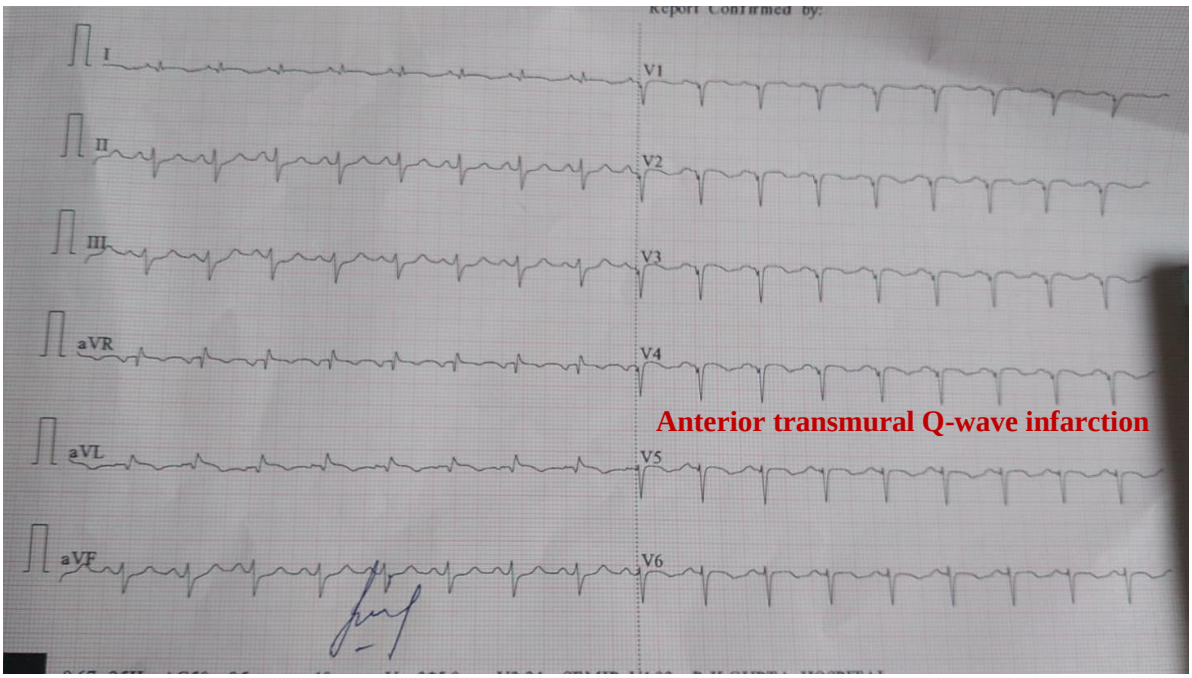
ECG 2 : The next immediate ECG



Source : The same

Findings : In addition , the presence of terminal QRS distortion sign – loss of S-wave with uplifting of the R-wave , extending from V2-V6.

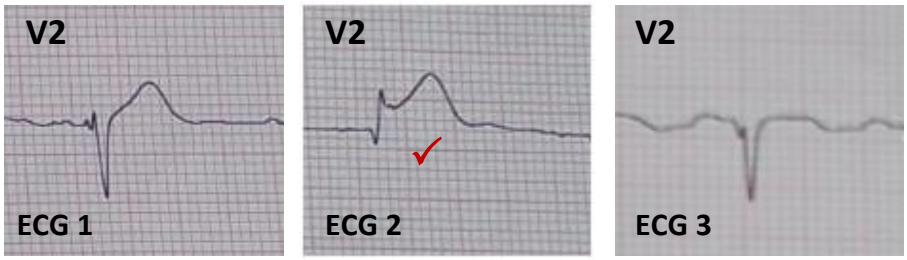
ECG 3 :Post PTCA ECG (of the same patient)



Source : The same

Findings : Evidence of STEMI in lead I and aVL plus full blown anterior transmural Q-wave infarction (more obvious over V1 to V4) indicating further worsening of ECG changes within the territory of LAD occlusion.
(Echocardiography LVEF 35-40% , CART Proximal LAD 90% occlusion)

Discussion on these trio-ECGs recorded in succession one after the other :
(Lead V2 here is the denominator to denote concerned ECG changes , as occurring)



History of ongoing chest pain (ST elevation in leads I and aVL with rS pattern from V2-V6) → **'TQRSD'-sign (loss of S-wave and J-point notch /slur)** → **Full bloom Q-wave (Full blown transmural Q wave infarction)**

- ECG 2 shows the presence of terminal QRS distortion sign (TQRSD) on the precordial leads , a forerunner of transmural myocardium infarction.
- ECG 3 illustrates the phenomenon of full blown anterior transmural Q-wave infarction.

NB :
In leads V2–V3 (inclusive other precordial leads) on ECG 2 , the terminal S wave is absent in the technical sense : although a downward limb follows the R wave, it remains elevated above the baseline due to injury current and therefore is *not* a true S wave **(when the terminal S-wave rises above the baseline, then it is technically considered no longer S-wave).** There is also loss of the normal J-point notch/slur. The R wave merges directly into marked ST elevation (>50% of R-wave height). This morphology, in the appropriate clinical context, predicts a large myocardial territory at risk. Recognition prior to reperfusion should prompt emergent PCI. This is worthwhile to mention here that possibly the lack of timely reperfusion has resulted in full blown transmural Q-wave infarction , as noticed with ECG 3.

5. Take Home Message

- ☐ The terminal QRS distortion is assessed on ECG by the loss of both the S-wave and J-notch /slur with uplifting of the remaining R wave above its baseline usually with > 50% , at least present over the precordial leads V2/V3.
- ☐ Proximal LAD occlusion → ischemia involving basal-anteroseptal wall → loss of S-wave and J-notch/slur. **The result :** the QRS appears truncated as if someone 'cut off the S-wave' and glued the ST segment directly to the R-wave.
- ☐ Its presence in the precordial leads has been strongly correlated with a risk of rapidly progressing STEMI in the territory of proximal LAD occlusion , marking an extensive area of myocardium at risk with an urgent need for revascularization.
- ☐ Terminal QRS distortion increases all-cause mortality by 81%. The concerned study suggests that terminal QRS distortion is an important bedside tool to assess the risk in patients with STEMI.

- A timely honoured urgent reperfusion therapy may reduce morbidity and mortality both in such a situation.
- Since terminal QRS distortion is associated with up concavity , it might be confused with early repolarization syndrome (ERS) . Always to keep in mind that terminal QRS distortion (TQRSD) is 100% specific to proximal LAD occlusion
- It should be rather a rule of thumb that every ECG with ongoing chest pain should be visualized and analysed for the presence of terminal QRS distortion sign so that urgent reperfusion therapy might be instituted to prevent the further catastrophe.

Terminal QRS distortion suggests that the critically ischemic myocardium is more likely to further evolve into the full-thickness transmural Q-wave infarction within a short time if not rapidly managed by urgent reperfusion therapy , it may lead to poorer outcome.

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