ECG

OCCLUSION MI (OMI) : A PARADIGM SHIFT

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OUTLINE

Occlusion MI (OMI) : A paradigm shift

A Narrative Review

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A paradigm shift is a fundamental change in how we review or understand the new changes occurring around us. It is a signal of profound transformation through powerful insights on breaking old habits, rewriting them in a revised way. This shift begins when existing system no longer function.

The medical world now recognized a new term addressed as occlusion MI (OMI) – a challenging situation to be diagnosed , otherwise a significant part of the left ventricle may be jeopardized with a poor outcome.

- ☐ **'Occlusion MI' should be adopted as a new approach to define transmural myocardial ischemia, which dictates urgent reperfusion , even in the absence of classical ST-elevation.**
- ☐ **Unfortunately , approximately half of high-risk patients with occlusion MI fail to meet STEMI criteria.**

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

1. Introduction (keypoints)

- Traditionally STEMI is diagnosed in the presence of new ST-segment elevation at J-point in two contiguous leads : $\geq$ 1 mm in all leads other than leads V2-V3 where the following apply : $\geq$ 2.5 mm in males < 40 years, $\geq$ 2mm in males $\geq$ 40 years, or $\geq$1.5 mm in females regardless of age, in the absence of bundle branch block and /or left ventricular hypertrophy.
- 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 ST-elevation MI – 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’.

- Unfamiliarity with this new occlusion MI concept can lead to diagnostic errors. Developing a strong conceptual understanding is crucial to avoid missing the diagnosis.

2. A basic concept while interpreting ‘occlusion myocardial infarction’

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

- **The Deepest Principle :** In OMI, the ECG should be read not as a search for ST elevation , but as a search for an abnormal electrical vector (altered injury / repolarization vectors) that is clinically and anatomically coherent with acute coronary occlusion.

  Alterations in ventricular repolarization are manifested as ST-segment and T-wave abnormalities. Therefore, attention should be directed toward ST-segment elevation or depression and T-wave changes, interpreting their distribution in the context of anatomical coherence and contiguous lead involvement. It should be kept in mind that ST-segment elevation in one territory may be accompanied by reciprocal ST-segment depression in electrically opposite leads; however, reciprocal changes are not mandatory.

- **Repolarizing abnormalities** (ST segment $\pm$ T-wave abnormalities) occur with this occlusion MI.

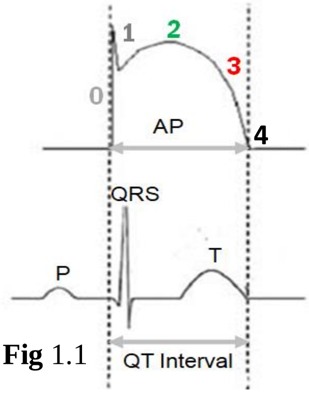
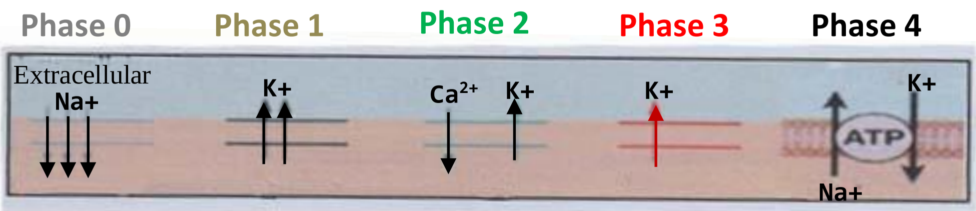


Fig 1.1



Ventricular action potential

Normally

Phase 2 accounts for isoelectric ST segment

Phase 3 accounts for T-wave genesis

- **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 usually observed in occlusion MI.
- If ST depression is diffuse , horizontal/downslowing , think of associated subendocardial ischemia in the presence of concomitant ST elevation. In subendocardial injury, the injury current is directed toward the ischemic subendocardial region and away from the overlying epicardial exploring electrode. This results in ST-segment depression on the ECG.

❖ **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.
- Tall T-wave in 'de Winter pattern' (dWp) with upsloping ST segment depression.
- Transmural ischemia-reperfusion injury with change in T-morphology as 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)

❖ **Essentials to keep in mind the leads oriented coronary supply**
(For better understanding by permutations and combinations) :

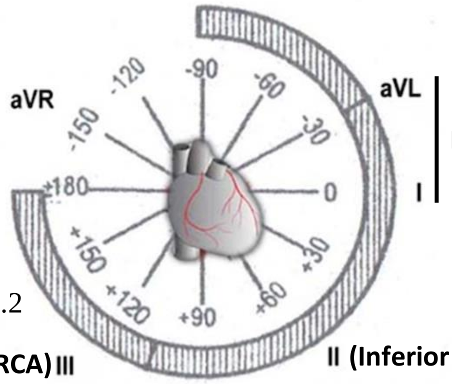


Fig 1.2

- **If there is diffuse ST depression with concomitant ST elevation in aVR , it may indicate LMCA or proximal LAD or severe 3VD depending upon the situation.**
- **LAD : leads V1-V6 (depending upon the main vascular domain involvement)**

- **Suspect RCA involvement (Right ventricular infarct) if there is ST elevation in V1 with inferior MI**
- If **Posterior MI** occurs with **inferior MI → think RCA -PDA**
- If **Posterior MI** occurs with **lateral MI → think LCX –PDA**

NB : Posterior MI is noted by the presence of reciprocal changes through leads V1-V3 as tall R , ST depression with upright T.

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

- ☐ **Alteration in T-wave**
  - Hyperacute T-wave (HATW)
  - 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)** : Hyperacute T waves are not recognized by height alone; they reveal themselves by disproportionate—bulky, symmetric T-wave , compared to the corresponding QRS (≤110 ms), interpreted in the appropriate anatomical and clinical context. It is a quantitative definition of HATW put by H. Pendells Meyers , et.al in 2025 , it is considered as a equivalent to ST-elevation MI (STEMI) in its expression , requiring emergent reperfusion.

Fig 1.3

As originally stated by Dr Stephen W. Smith if T wave /QRS complex ratio in any V1-4 is greater than 0.36, it represents acute MI, not subacute or old. (let's say 0.33 for both convenience and for better sensitivity), then assume there is an acute component to the MI (Dr. Smith's ECG Blog, on October 21, 2017).

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)

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 T**.(**upsloping ST segment depression** is due to K^{ATP} activation , sequences ; Opens ATP-sensitive k+ channels → ↑K+ exit without accompanying Ca2+ entry → transient cessation of excitation-contraction coupling → transient stay of ST segment downward).

- 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

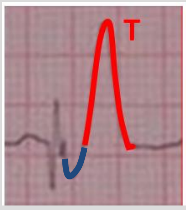


Fig 1.4

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.

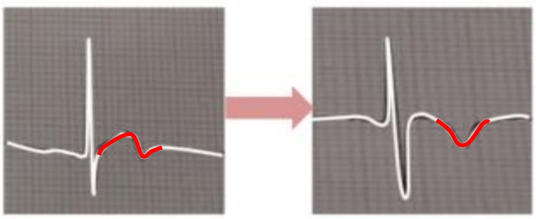


Fig 1.5

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 features :

- ☐ This occlusion MI is the resultant of **septal ischemia** , corresponding to the obliquely placed anterior interventricular septum (LAD occlusion proximal to S1)
- ☐ The obliquity of the interventricular septum catches ST elevation in leads V1-V2 (aVR).There is associated reciprocal ST depression in leads V5-6.

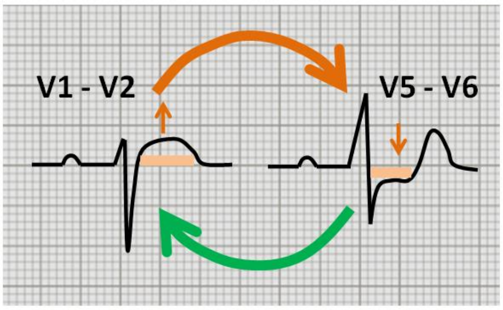


Fig 1.6

- ☐ Since precordial swirl sign is dynamic in nature , evolving hyperacute T-wave changes may also be coexistent therein :
  'ST-segment elevation and /or hyperacute T-waves in V1-V2 with 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.

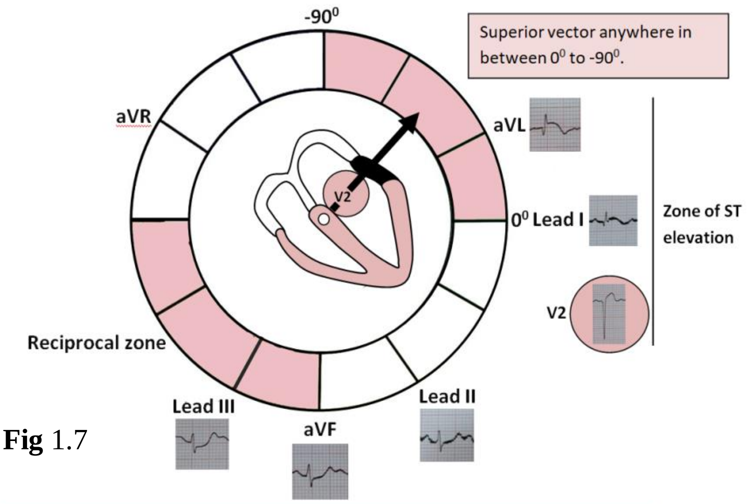


Fig 1.7

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

ST vector passing through leads I , aVL and V2

→ ST↑ in these leads

Reciprocal ST depression in inferior leads , most prominent in lead III, being the farthest away.

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

Fig 1.8

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

Posterior OMI

<u>Reciprocal ECG changes</u> (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.

- If Posterior MI occurs with inferior MI → think RCA-PDA (the commoner)
- If Posterior MI occurs with inferior + lateral MI → think LCX-PDA

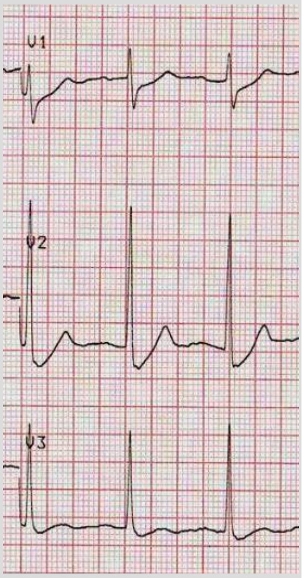


Fig 1.9

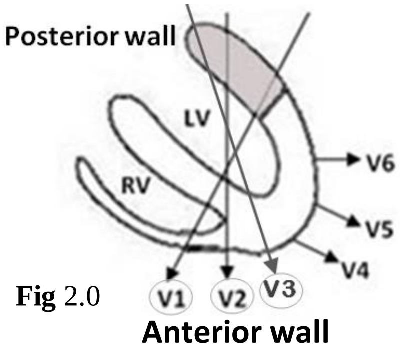
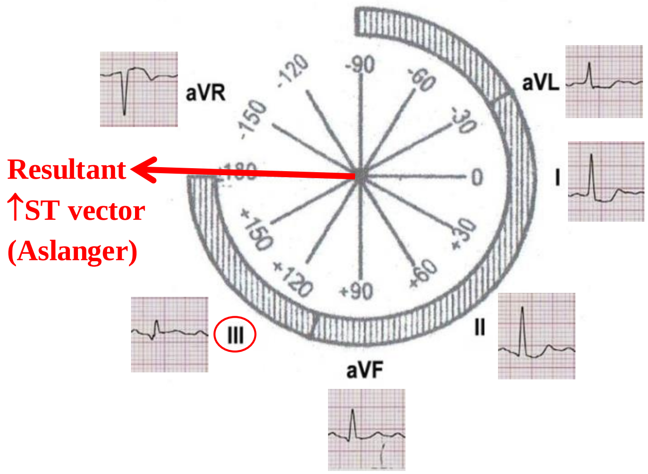


Fig 2.0

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

Aslanger pattern –ST elevation in lead III due to acute occlusion of RCA + concurrent multivessel disease , predominantly due to LAD occlusion in addition.



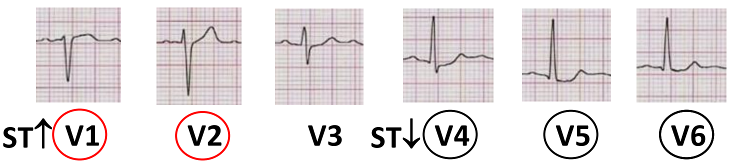


Fig 2.1

Essentials :

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

NB : Aslanger Pattern Lift - An acute RCA occlusion creates a rightward, inferior injury vector that should elevate lead III and V1. However, concurrent multi-vessel subendocardial ischemia pulls a massive competing vector upward and leftward, neutralizing leads II and aVF so that only lead III and V1-V2 survive as isolated ST elevations.

👉 Northern OMI

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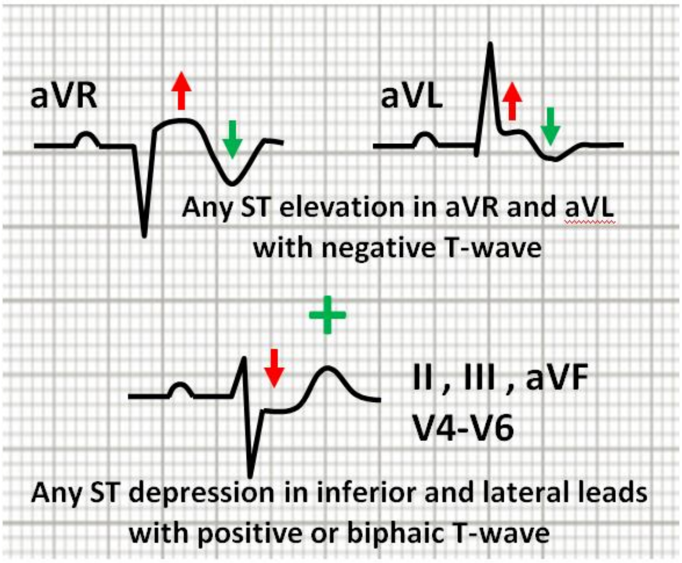


Fig 2.2

ECG pattern :

- 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

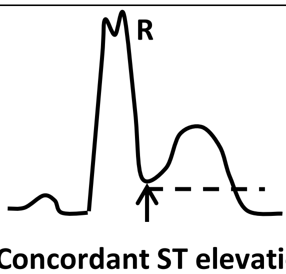
NB : The lesion is typically a subtotal occlusion , or an acute occlusion occurring on top of LMCA or proximal LAD. The Resultant Vector moving towards -90^0 → Subepicardial injury directed towards leads aVR and aVL. And the reciprocal subendocardial injury towards the inferior leads II , III, aVF and V4-V6.

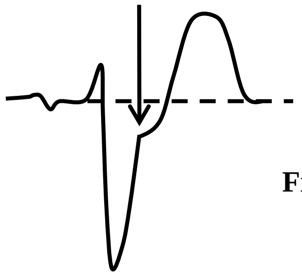
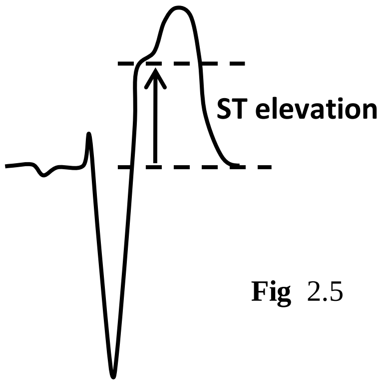
This occlusion MI as the name indicates is having northern vector orientation , more towards aVR with unique diagnostic ECG findings but prospective studies are needed to confirm its specificities and sensitivity.

☐ 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, this new pattern of repolarization sets in due to the cumulative effect of MI ST-elevation upon ST depression of LBBB –
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** 2.3

2. Concordant ST depression $\geq$1 mm in $\geq$ 1 lead of V1-V3	**Concordant ST depression**  **Fig** 2.4
3. Proportionally excessive discordant STE in $\geq$ 1 lead anywhere with $\geq$1 mm STE , as defined by $\geq$ 25% of the depth of the preceding S-wave. **(Smith Modified Sgarbossa Criteria)**	 **Fig** 2.5

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).

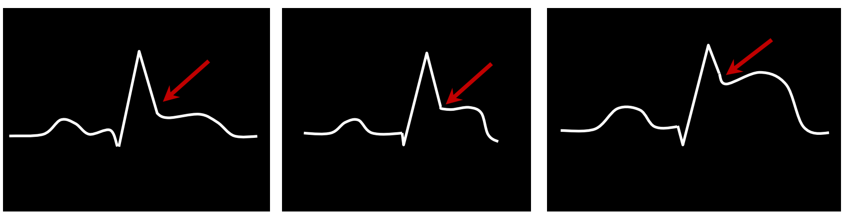
👉 **Terminal QRS distortion**

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 extinguishes ST segment → **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 2.6



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 established that the proximal LAD-septal perforators (S1,S2,S3) perfuse both the right bundle branch and the anterior fascicle of the left bundle branch. This newly onset bifascicular block may be the only pointer for OMI. Either of block may also occur in isolation. Anterior wall MI usually shows RBBB pattern.

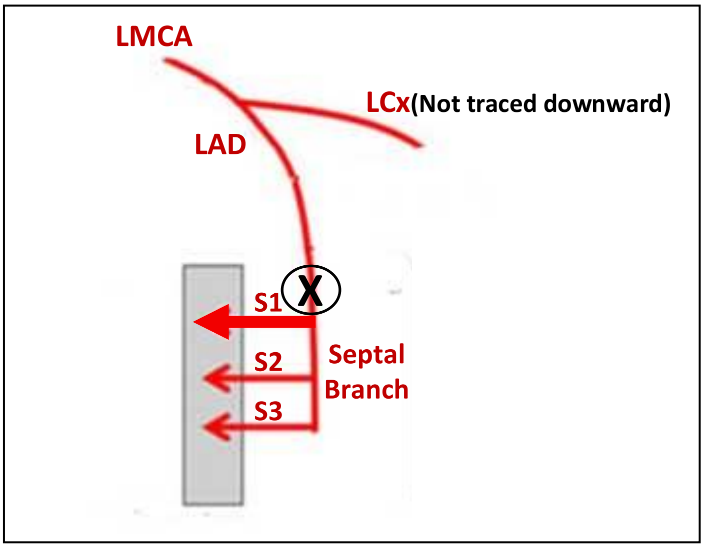


Fig 2.7

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	• Hyperacute T-waves appear taller and broad-based , often generally more symmetrical than the normal T-waves. • 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-V2 (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 $\pm$ 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 posterior MI • ST depression , tall R-waves and prominent positive T-waves (V1-3) should be considered posterior OMI until proven otherwise.
Aslanger pattern (RCA plus multivessel disease , predominantly due to LAD occlusion in addition)	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 (RCA) • ST ↑ in V1>V2 • Concomitant ST↓ in V4-V6, with a positive/terminally positive T-wave (concomitant subendocardial ischemia)
Northern OMI (LMCA or proximal LAD)	• 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 $\geq$1mm in $\geq$ 1 lead (with a positive QRS complex) • Concordant ST depression $\geq$1 mm in $\geq$ 1 lead of V1-V3 • Proportionally excessive discordant STE in $\geq$ 1 lead anywhere with $\geq$1 mm STE , as defined by $\geq$ 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 • Bifascicular block (RBBB + 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 – 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'.
- To have familiarity with the different patterns of occlusion MI one should consult 'Tablewise summary' as illustrated in this article.
- In context with occlusion MI , always consider dynamic ECG assessment , compared with prior ECGs , correlate with symptoms and serial troponin concentrations.
- 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

7. References

All the articles with the references mentioned against the internet links are retrieved from therein.

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