

ECG CHANGES IN CAD

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Einthoven
(1860-1927)



Einstein
(1879-1955)

As per Einstein's quote ' The release of atomic energy has not created a new problem. It has merely made more urgent the necessity of solving an existing one'.

CAD in itself may be considered as an impending atomic explosion or as happening event AND this compels the clinician's mind to ponder upon its solution how to recognize this.

A small leap to understand an ECG (Einthoven's gift) might pave the way how to solve this problem – of course in the light of existing clinical scenario.

This companionship knowledge based on ECG answers to the soul's cry to CAD – a clinician's search to cross the pain of the grieved heart.

➡ ST depression is the primary event in myocardial ischemia / Non-STEMI

**➡ Retrograde changes in a disciplined way –
T (hyperacute) – ST (Coving) – Q (pathological) – ending with T changes – all happenings in a disciplined way – STEMI.**

A concept of CAD based on ST deviation

ST depression

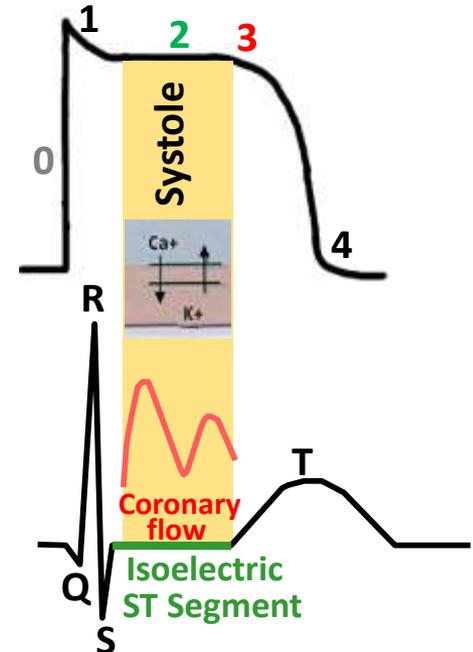
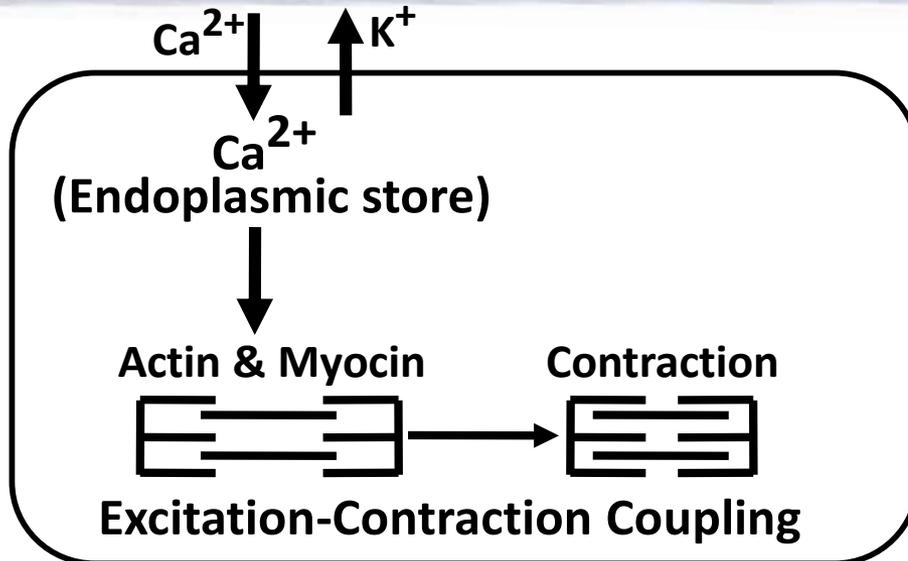
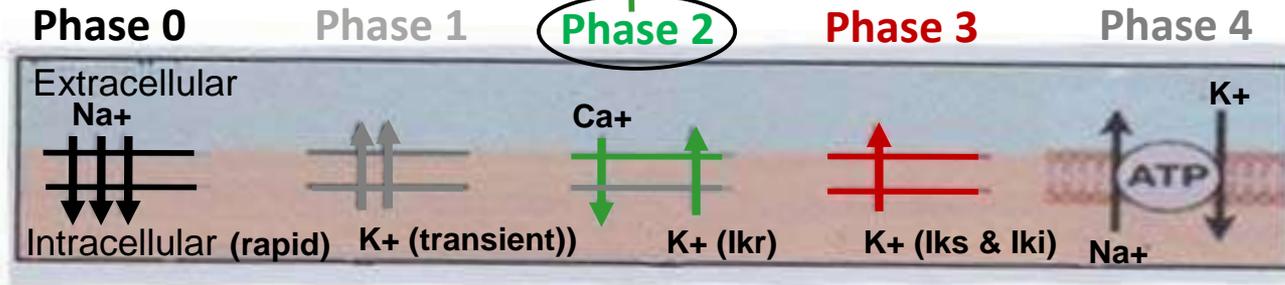
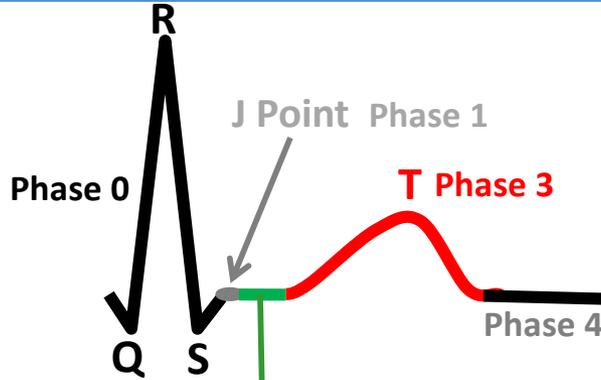
- Angina pectoris on effort
/ Chronic stable angina
- Unstable angina
- Non -STEMI

ST elevation

- STEMI

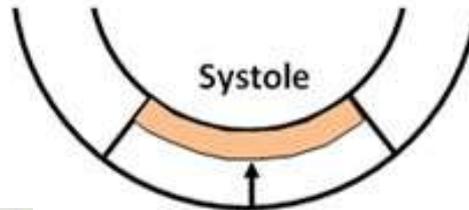
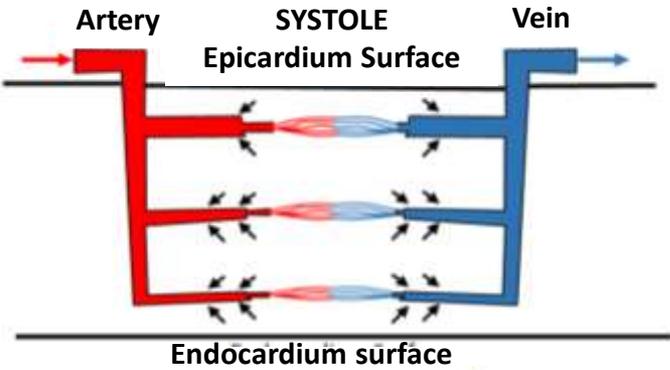
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- Occlusion myocardial infarction (OMI) : A new emerging concept of ACS in the absence of traditional ST elevation.

ST segment (Phase 2) in CAD



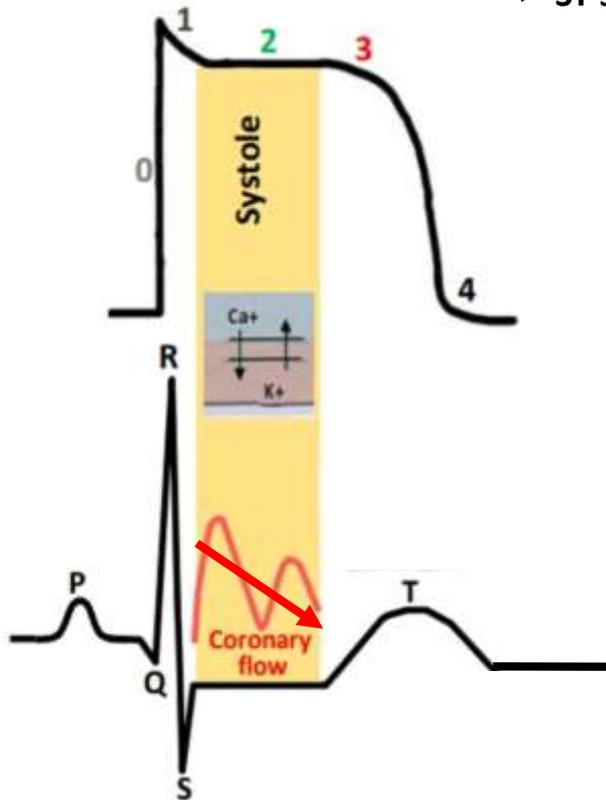
➡ Impaired coronary flow (CAD) → subendocardial / subepicardial injury → **ST segment deviation**

Electropathogenesis of Myocardial ischemia

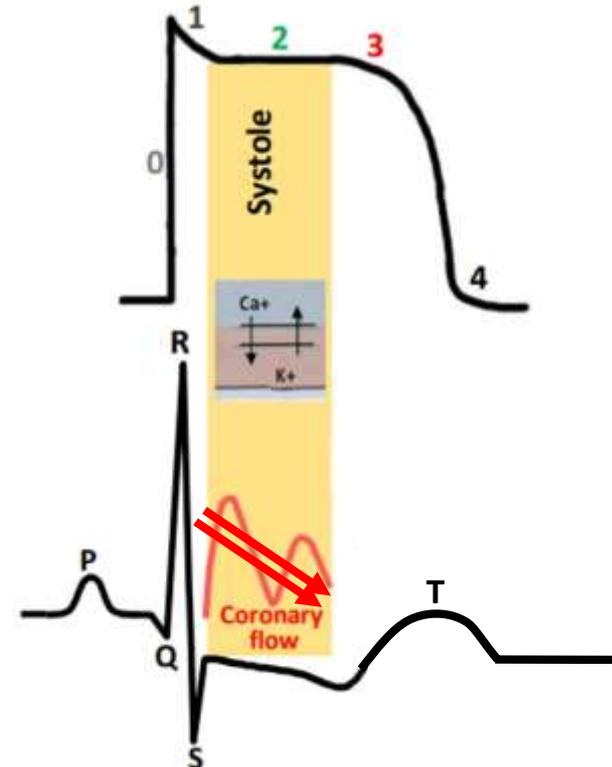


Subendocardial Injury

- The flow of current is away from the exploring electrode
- ST segment depression



Horizontal ST segment depression



Downsloping ST segment depression

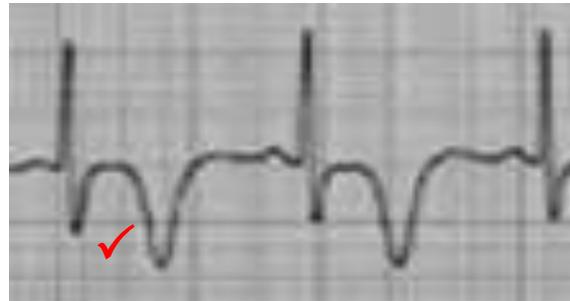
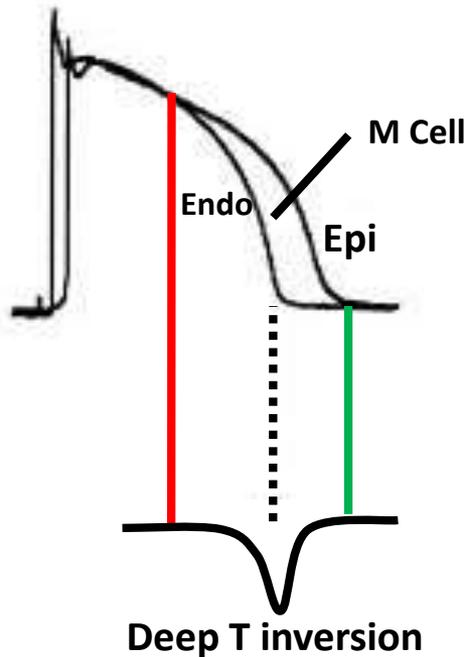
ST depression in myocardial ischemia

➡ Horizontal or downsloping ST segment depression

- ❑ ≥ 0.5 mm ≥ 2 in two contiguous leads \rightarrow myocardial ischemia
- ❑ ST depression ≥ 1 mm \rightarrow a worse prognosis.
- ❑ ST depression ≥ 2 mm in ≥ 3 leads \rightarrow possibility of Non-ST elevation myocardial infarction (NSTEMI) \pm T inversions or flat T
- ❑ Upsloping ST segment (non-specific) is rarely caused by ischemia. It is usually physiological in nature, seen in normal persons usually during physical exercise and resolves rapidly once the exercise is stopped.

Sometimes reverse repolarization in CAD

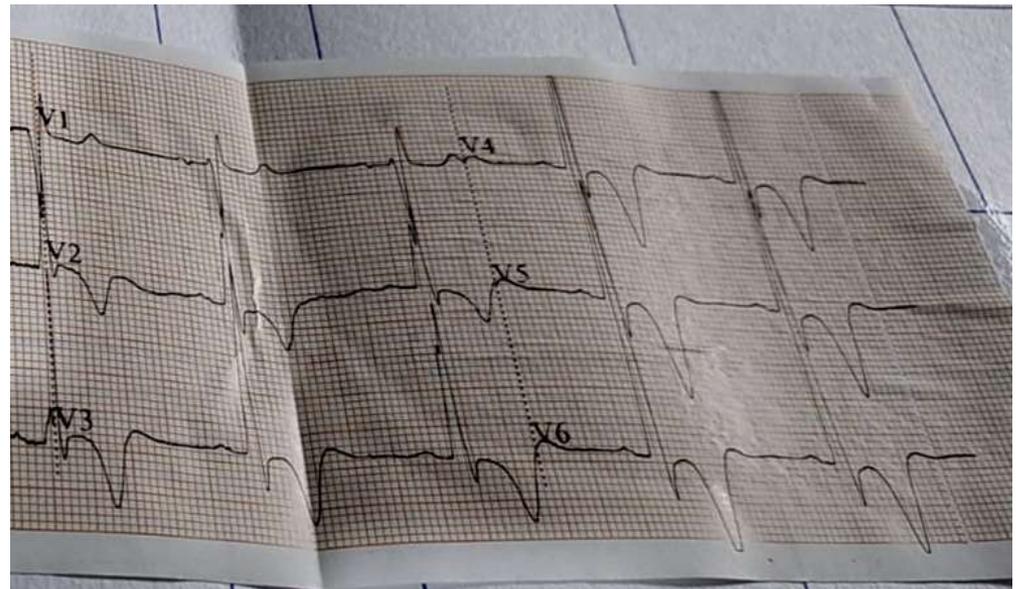
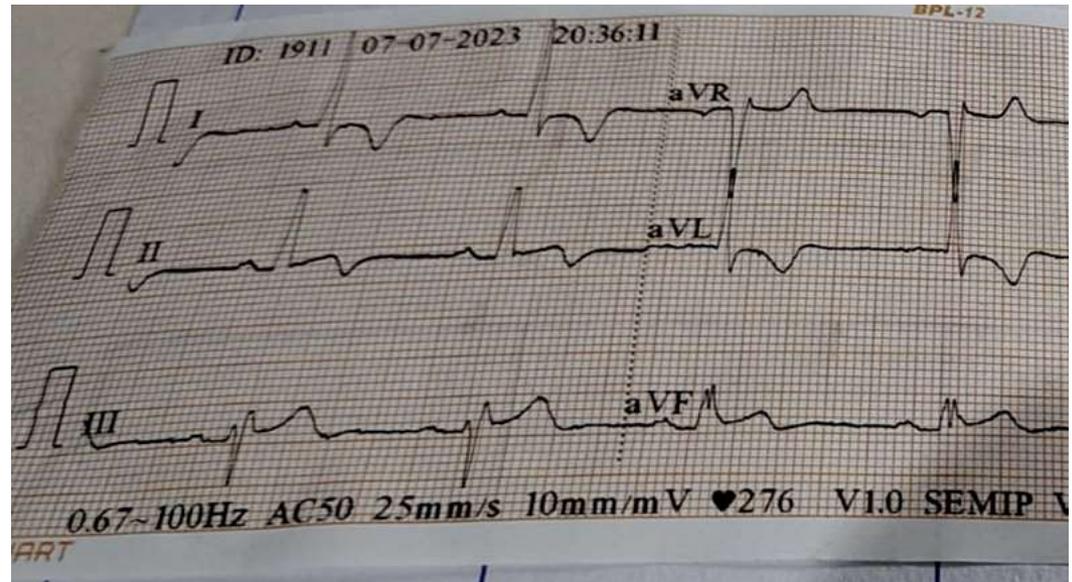
When Endocardium repolarizes first (normally epicardium repolarizes first)



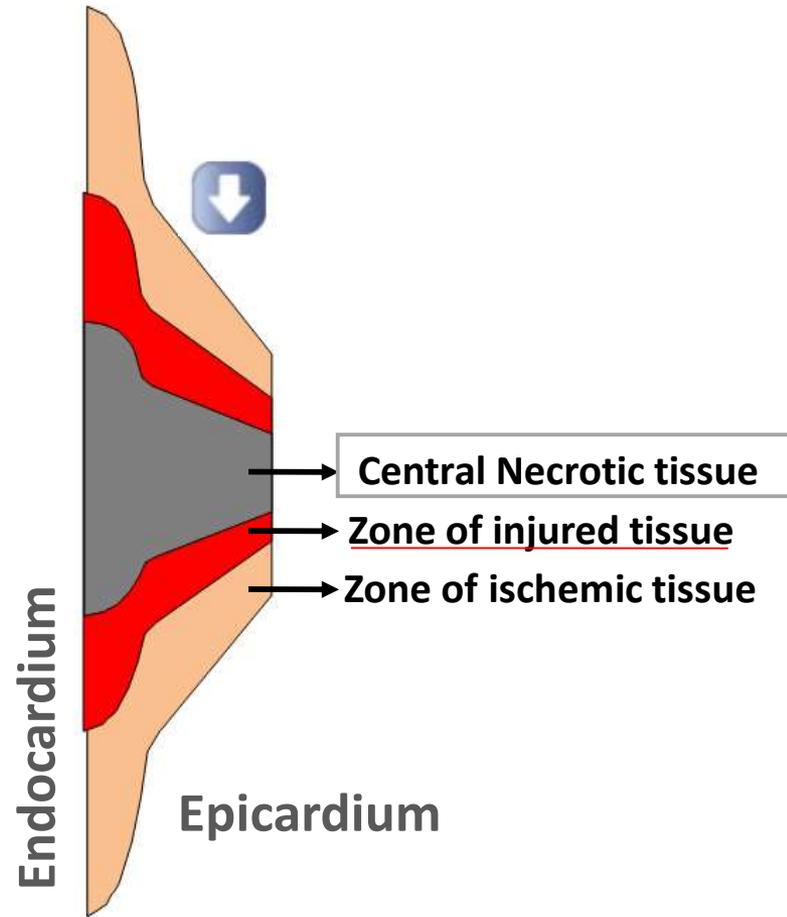
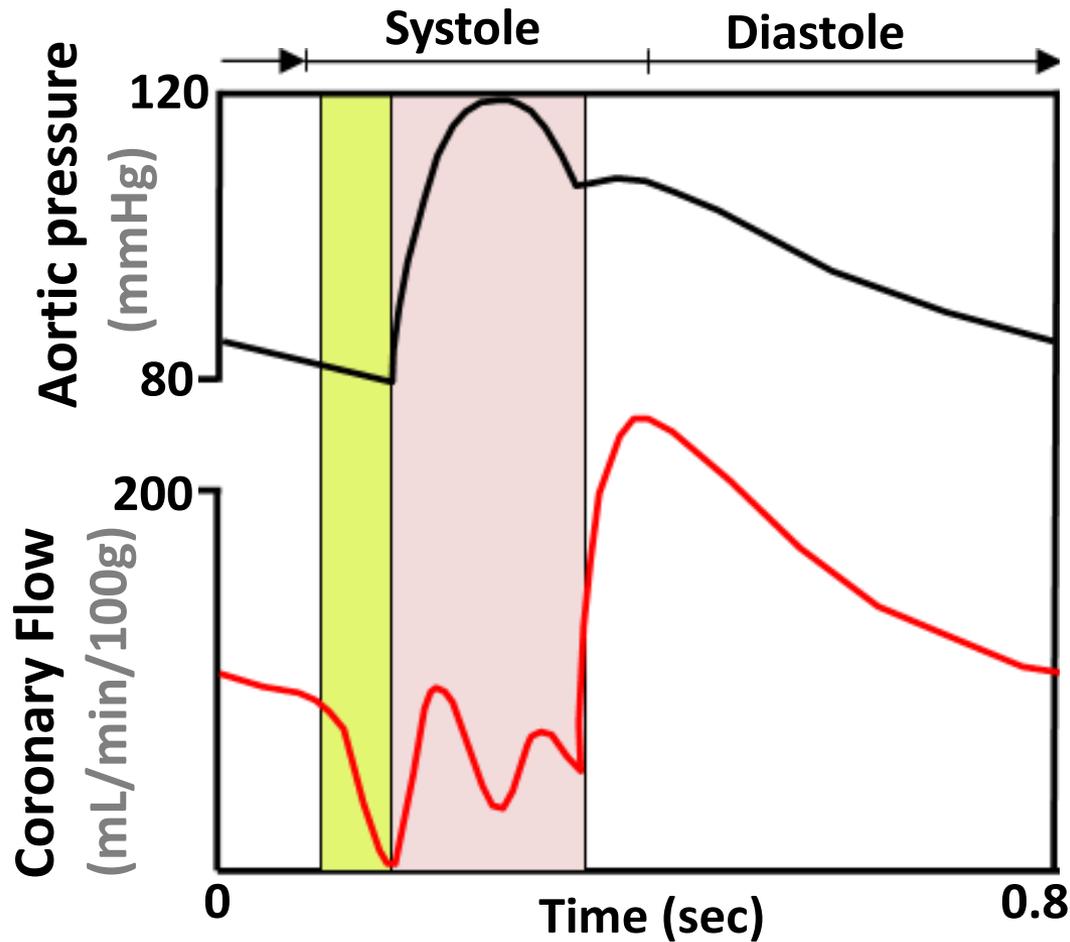
- Acute myocardial ischemia may slow the propagation of cardiac impulse with takeover by endocardium (may be global T-wave inversion)
- Reperfusion of STEMI : T-wave inversion here is not indicative of new ischemia.
- Wellen's syndrome also reperfusion injury related
- Apical hypertrophic cardiomyopathy associated angina (Yamaguchi syndrome)

ECG illustrating deep T-wave inversion

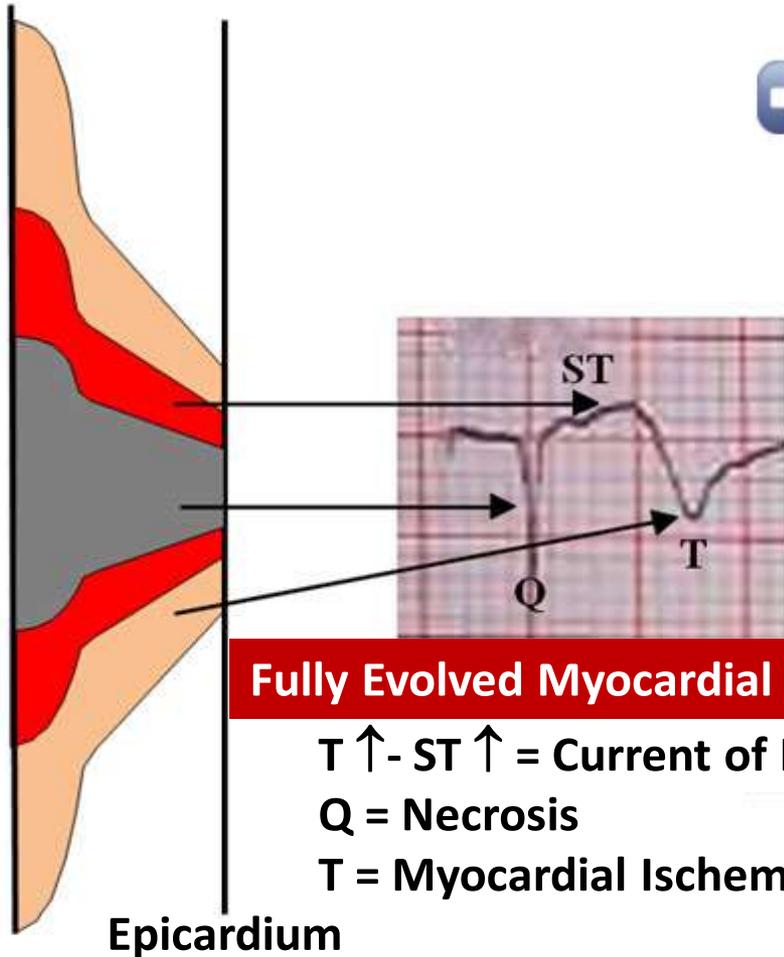
71 years male , heaviness over chest on effort known cases apical HCN



A concept of STEMI



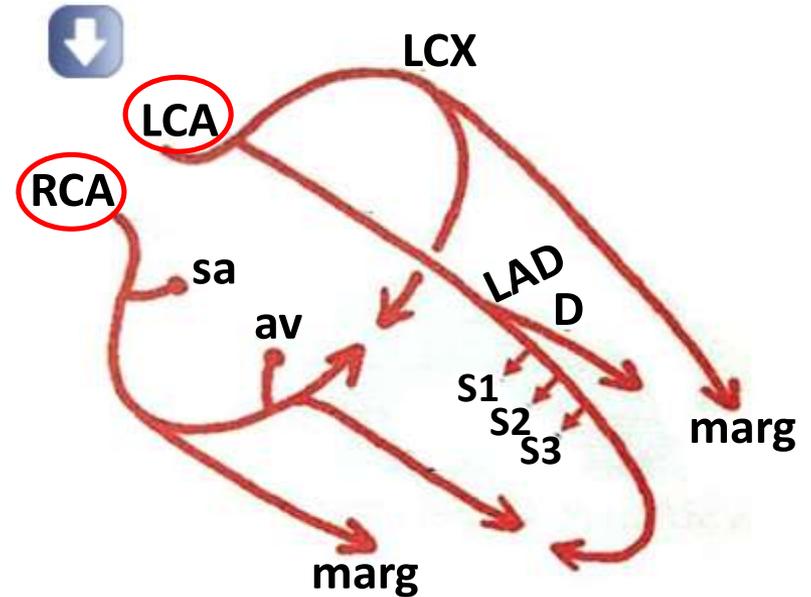
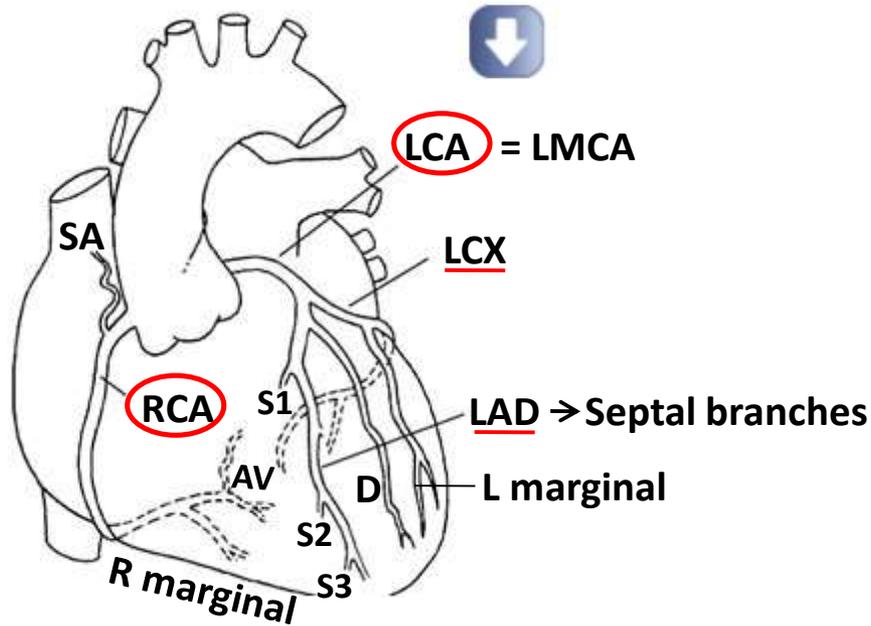
Evolution of STEMI



	1 	2 	3
Hyperacute T	mts-few hrs	few hrs-few days	few days – few weeks

- New ST-segment elevation at J-point in at least two anatomical continuous leads of ≥ 2 mm in a male or ≥ 1.5 mm in a female over leads V2-V3 and/or at least 1 mm in other continuous leads or limb leads
- A wide Q wave (>0.04 sec) that exceeds 25% of the R wave in the leads with necrosis.

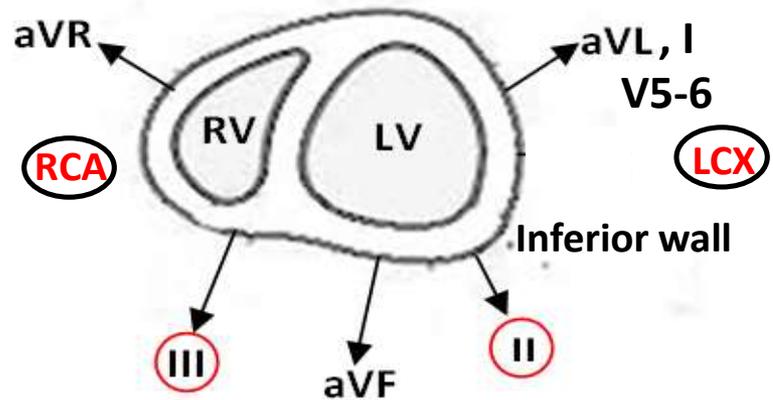
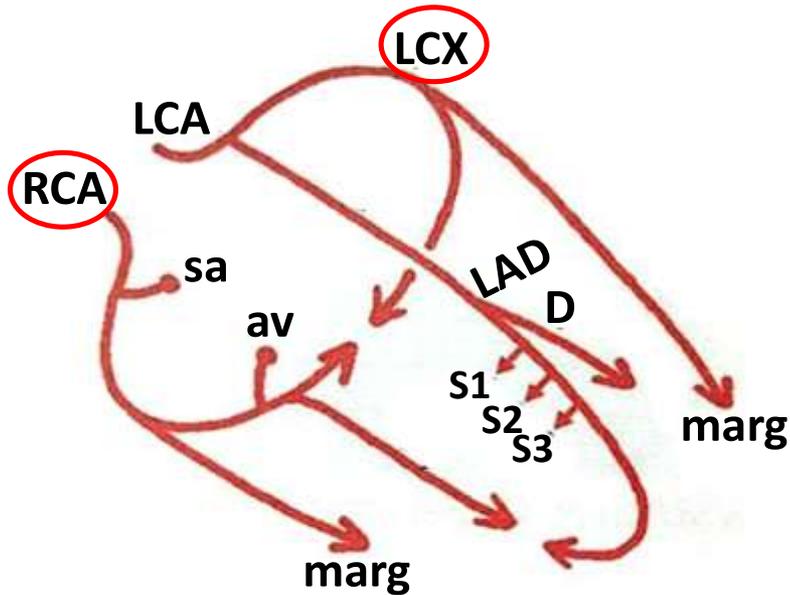
Coronary circulation



- RCA → posterior descending artery = **"right-dominant"**. (In 70% of cases).
- LCX → posterior descending artery = **"left-dominant"**. (In 20% of cases).
- Rarely by both (10%)

(A precise anatomic definition of dominance would be the artery which gives off supply to the AV node)

RCA and LCX STEMI



RCA STEMI

- ST elevation in III > II + ST elevation in V1 and ST depression and V2 (highly specific for RV infarction)
- Diagnosis is confirmed by the presence of ST elevation in the right sided leads (V3R – V6R) concentrate upon ST elevation in V4R in the diagnosis of RV infarction

LCX STEMI

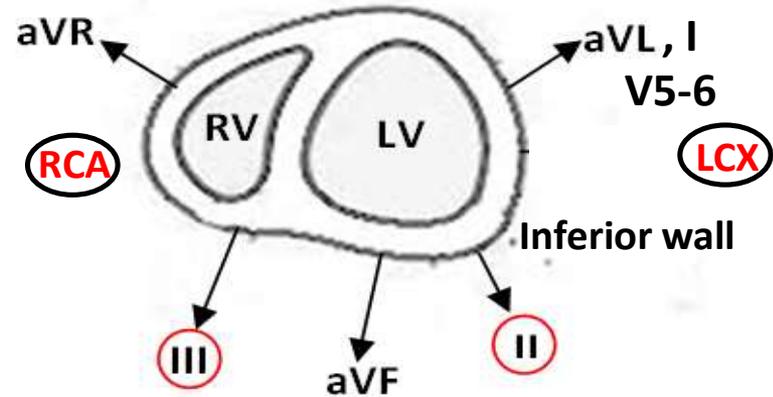
- ST elevation in II > III
- ST elevation in aVL, I, V5-6 (combination of high lateral leads aVL, I and apical leads V5-6)

STEMI in association with inferior MI

❑ Proximal left circumflex artery

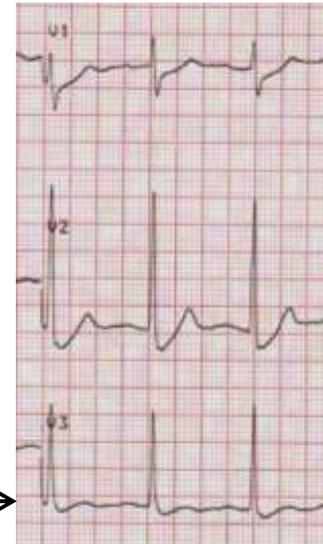
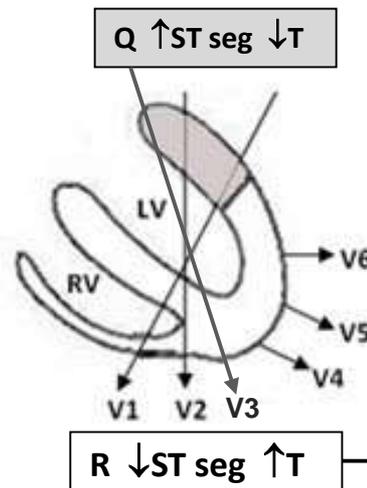
❑ **Concentrate over V1 and V2**

- Proximal RCA occlusion
(ST↑ in V1 and ST↓ in V2)



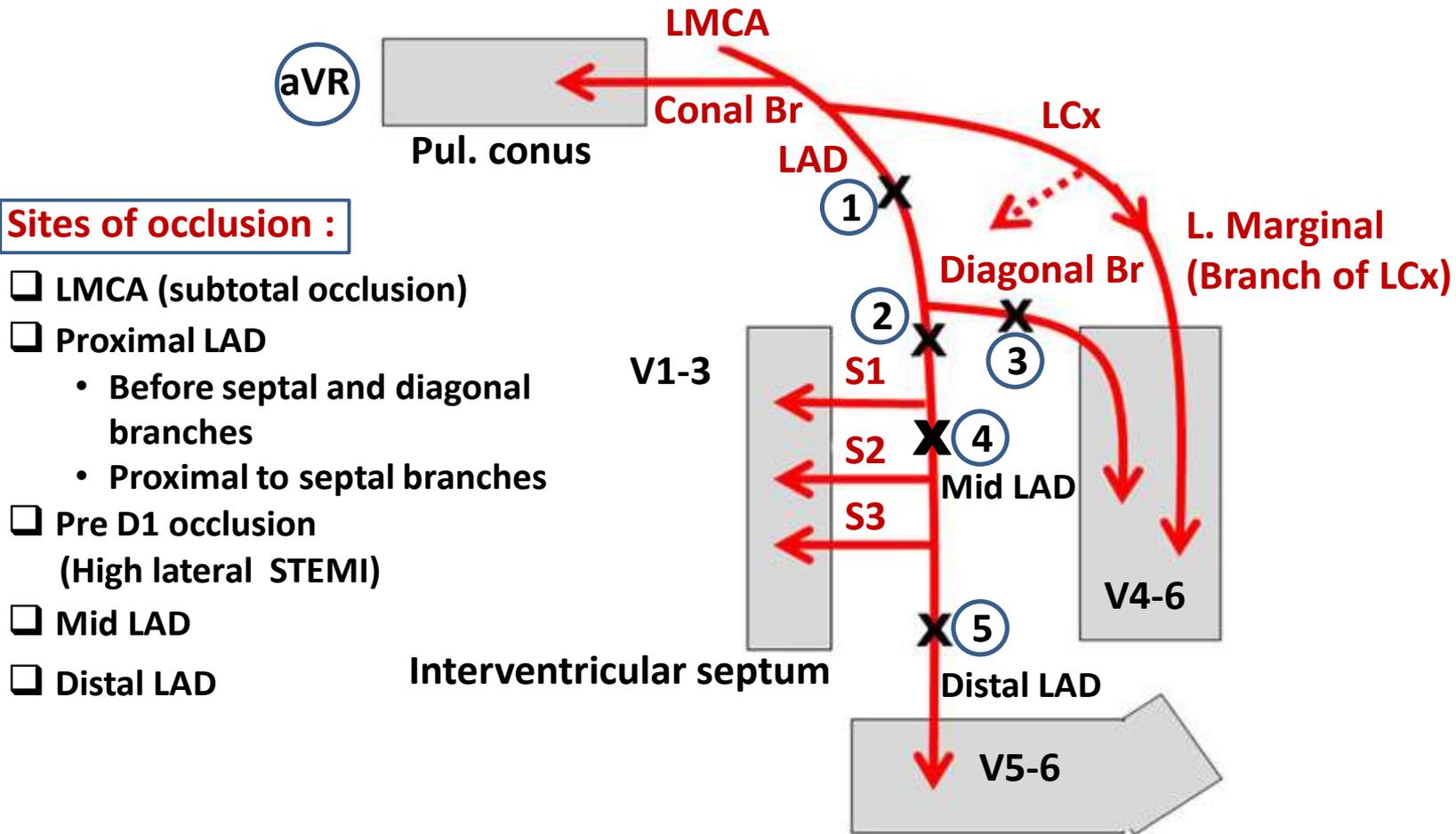
• Posterior wall MI

(Reciprocal changes in acute posterior MI on V1 V2 V3)



- Proximal LAD with a wrap artery lesion around the apex of the left ventricle
- Distal RCA dominant

Anterior wall MI



Sites of occlusion :

- LMCA (subtotal occlusion)
- Proximal LAD
 - Before septal and diagonal branches
 - Proximal to septal branches
- Pre D1 occlusion (High lateral STEMI)
- Mid LAD
- Distal LAD

An elementary sketch showing the atrial supply by LMCA and its two branches LAD and LCx

An approach to recognize sites of occlusion in CAD

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

LMCA (subtotal occlusion)

Widespread ST depression with ST elevation in aVR ≥ 1 mm

Proximal LAD

- Before septal and diagonal branches : ST \uparrow in V1-6 \pm aVL (1) / qRBBB (C) and ST elevation in lead V1 > aVR
- Proximal to septal branches : ST \uparrow in V1-3

Pre D1 occlusion

ST \uparrow in I , aVL , +/- V2

Mid LAD

ST \uparrow in V2-6 (reciprocal depression in aVL)

Distal LAD

ST \uparrow in V5-6 (reciprocal depression in aVL)

RCA STEMI

ST \uparrow in III>II + ST \uparrow in V1
and ST \downarrow in V2

LCX STEMI

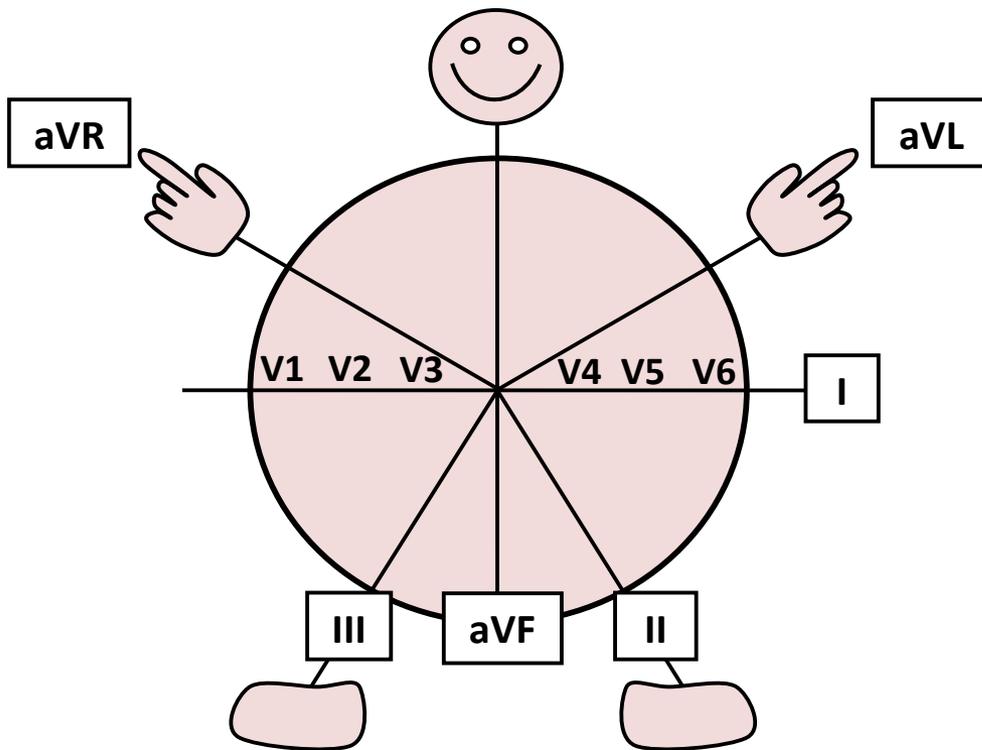
ST \uparrow in II>III , aVL , I , V5-6

Posterior wall MI

Reciprocal changes in

V1 V2 V3 : R \downarrow ST Seg \uparrow T

Reciprocal changes with CAD

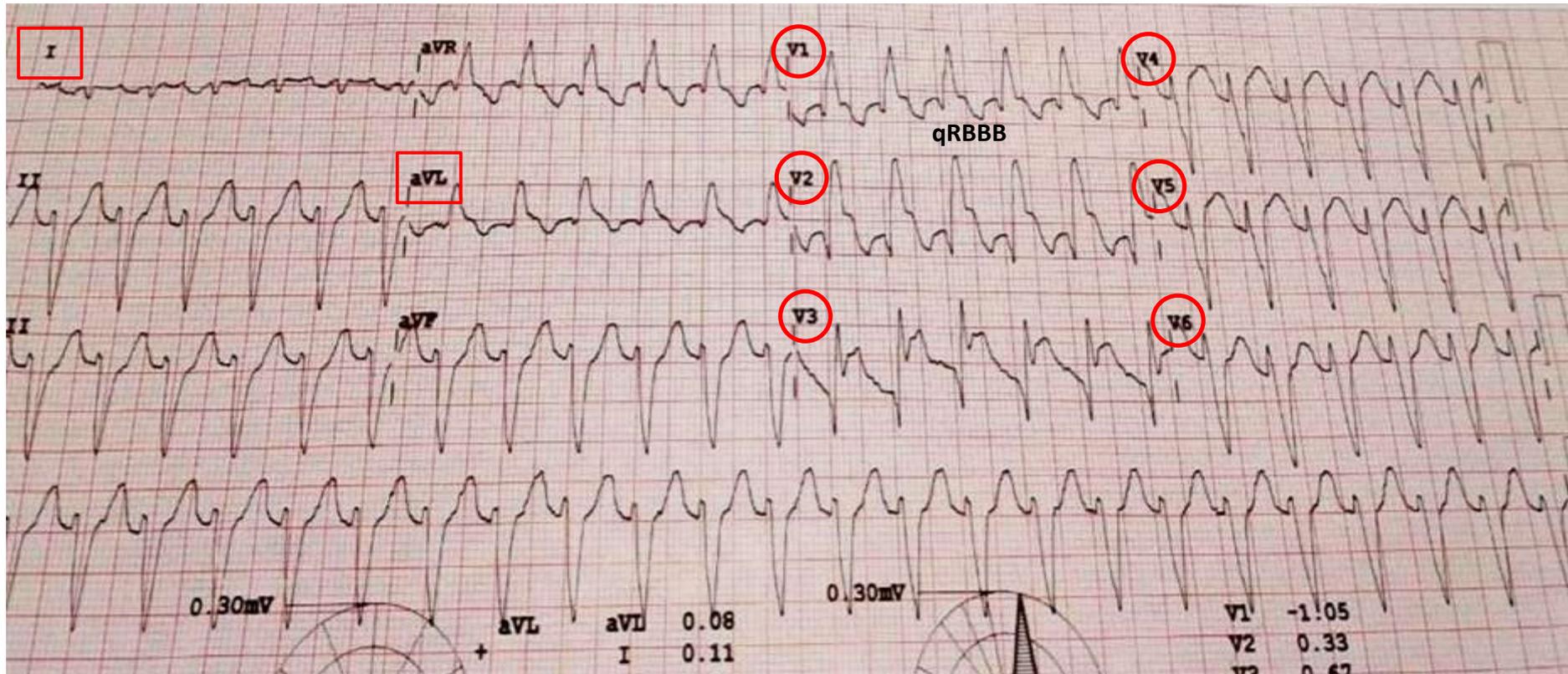


➔ Reciprocal change is defined as ST-segment depression ≥ 1 mm in at least 2 leads in a single anatomic segment, occurring onto the contralateral side, as a mirror-image effect of ST-segment elevation associated with STEMI.

➔ A basic concept
Such reciprocal ST segment depression is considered to represent mirror image - a phenomenon of electrical reflection of the transmural injury onto the opposite ventricular wall. This supports the diagnosis of STEMI with specificity and positive predictive value of 93%.

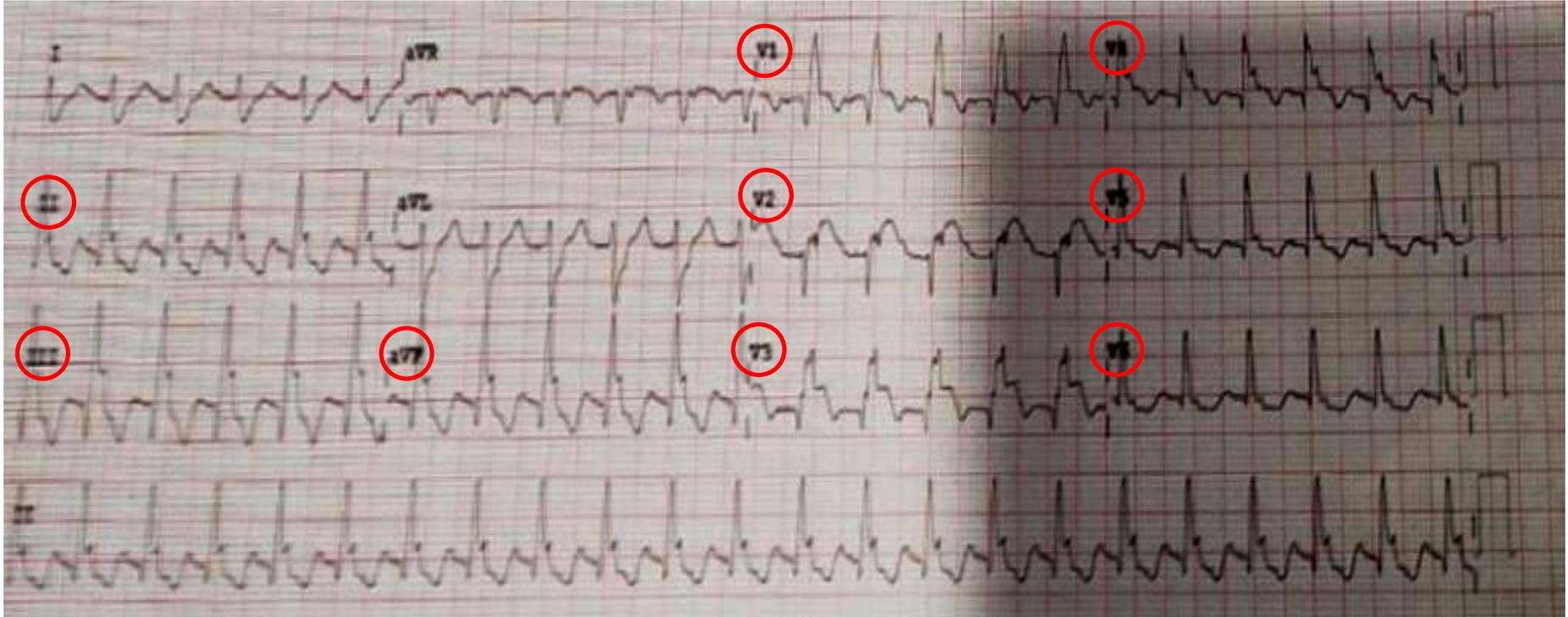
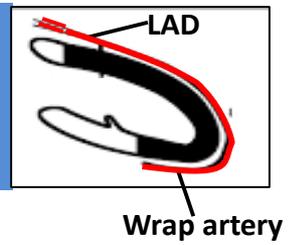
➔ Reciprocal change can be the first sign of STEMI - the dominant sign of occlusion, contralateral to subtle ST elevation.

Proximal LAD occlusion before septal and diagonal



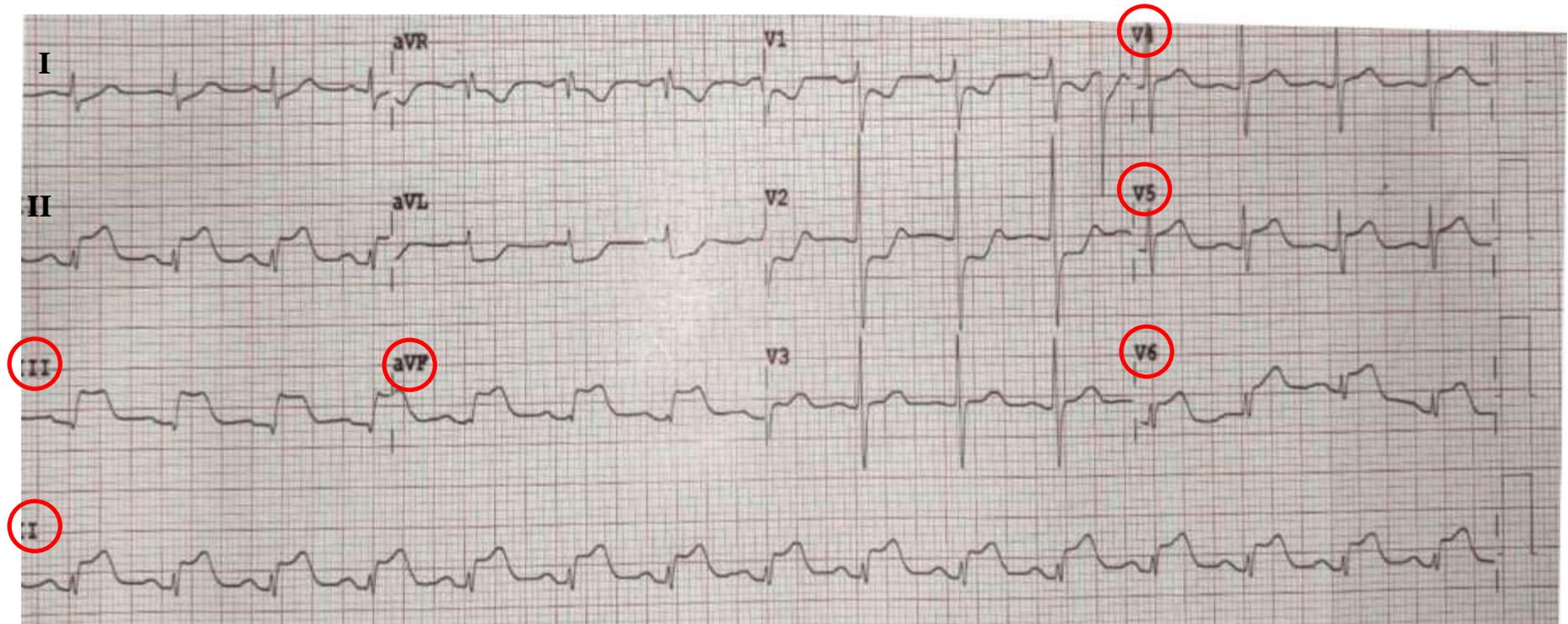
✓ I	aVR	✓ V1	✓ V4
II	✓ aVL	✓ V2	✓ V5
III	aVF	✓ V3	✓ V6

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



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

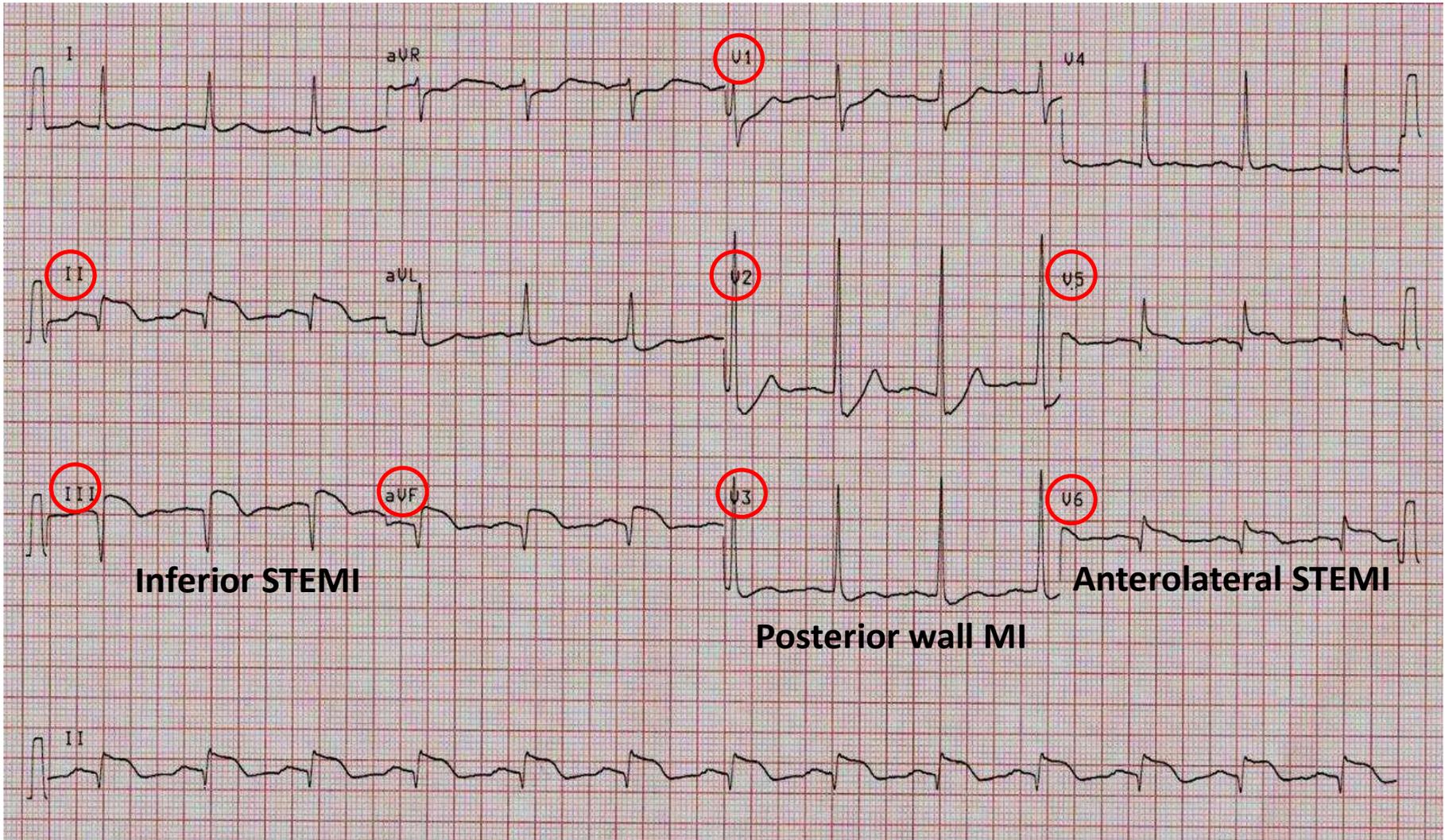
Proximal left circumflex artery occlusion



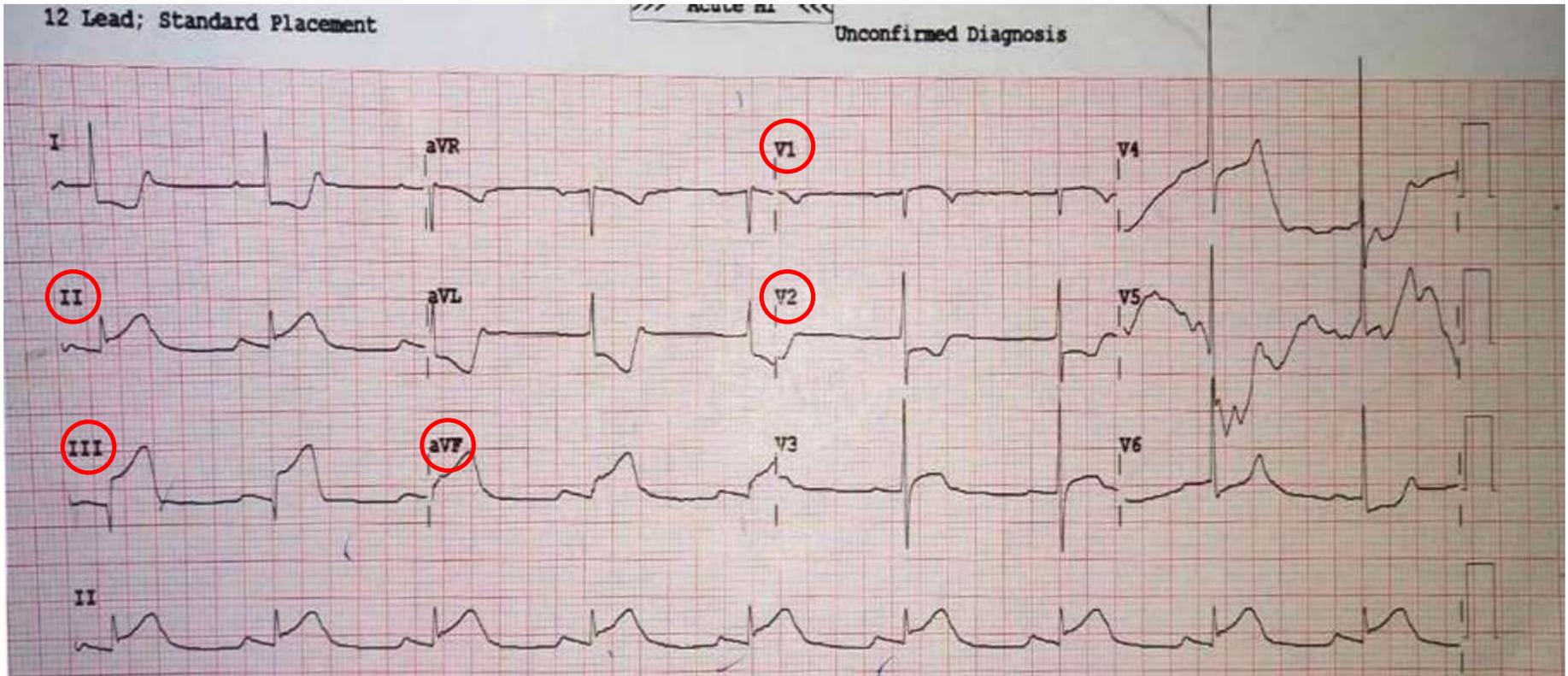
I	aVR	V1	✓ V4
✓ II	aVL	V2	✓ V5
✓ III	✓ aVF	V3	✓ V6

Dominant LCX occlusion (in 20% of cases)

Three in one



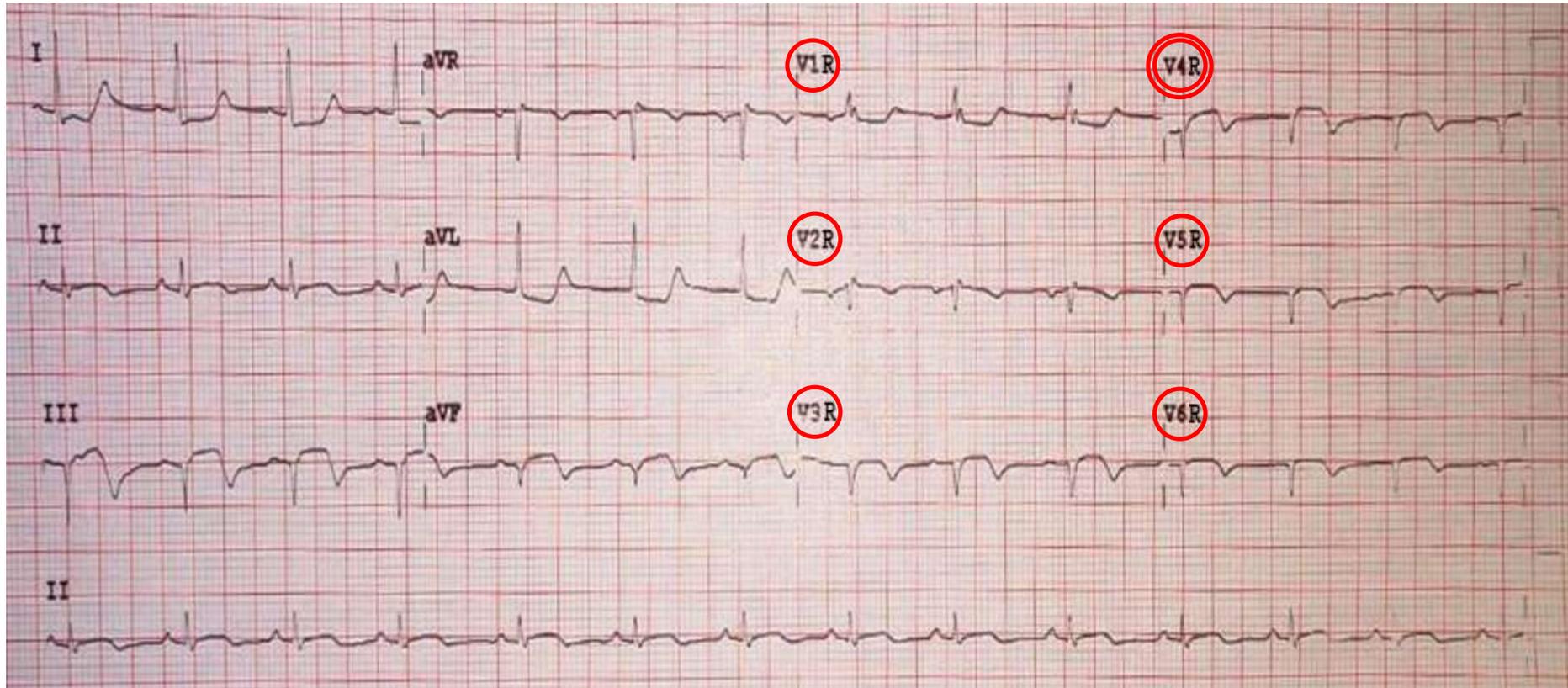
Acute right ventricular infarction



- ❑ Sinus Bradycardia with first degree AV block
(Right coronary artery supply also SA node and AV node)

I	aVR	✓ V1	V4
✓ II	aVL	✓ V2 ST↓	V5
✓ III	✓ aVF	V3	V6

By placing the precordial leads over the right chest

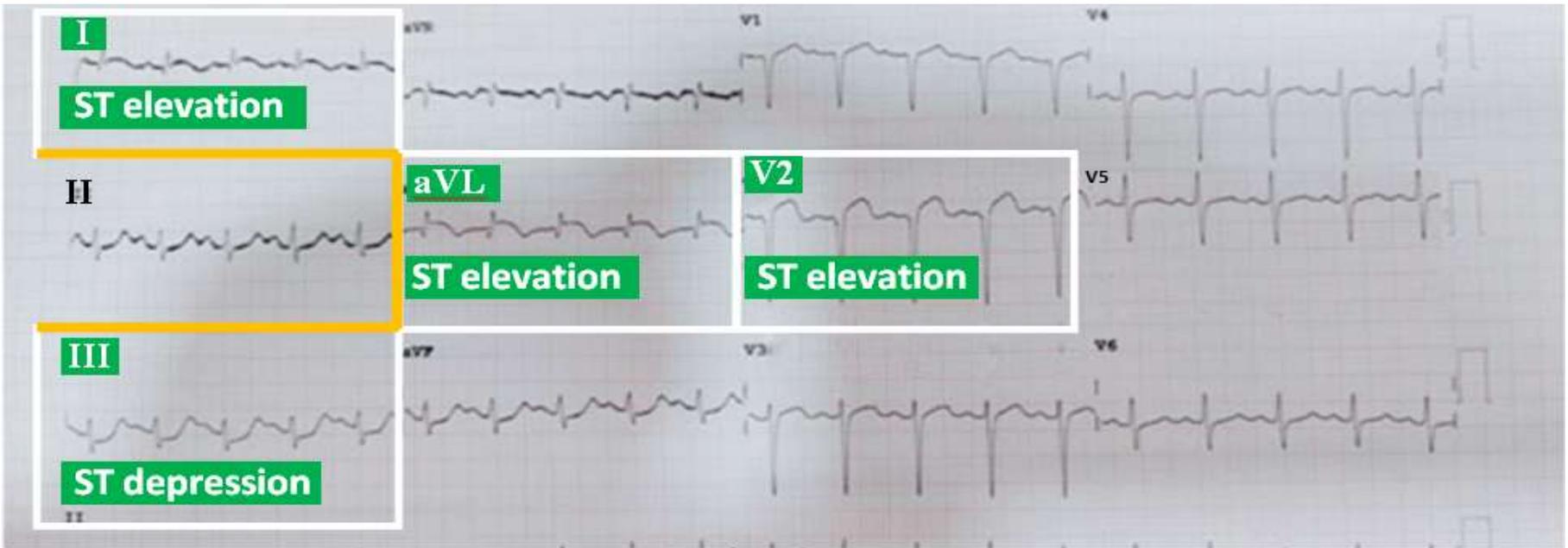
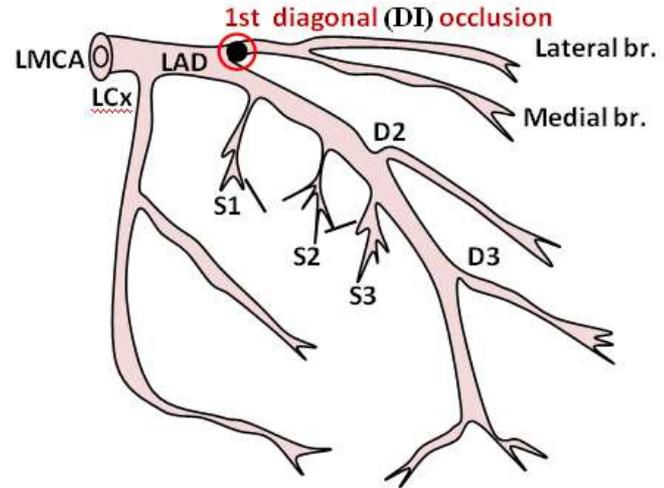


Comments :

Acute inferior MI + presence of ST elevation in the right sided chest leads (V3R –V6R) = further confirmation of acute right ventricular infarction

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

HIGH LATERAL MI – PRE D1 OCCLUSION (ST↑ in I, aVL, +/- V2 and reciprocal ST depression in inferior leads, predominantly in lead III)

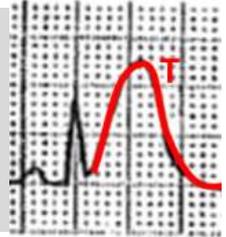


Working classification of occlusion MI

Alteration in T-wave

Hyperacute T-wave (HATW)

Hyperacute T-wave is out of the proportion of preceding R-wave , with \geq two contiguous leads may be the first sign of developing infarct often preceding STE- these are broad based often generally more symmetrical than the normal T-wave



Peaked tall T-wave (de Winter pattern)

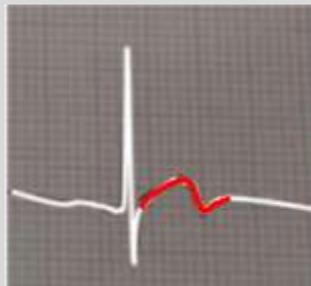
- Tall prominent symmetrical T waves in the precordial leads
- Upsloping ST segment depression $> 1\text{mm}$ at the **J point** in the precordial leads
- Absence of ST elevation in the precordial leads
- Reciprocal ST segment elevation (0.5mm – 1mm) in aVR



T-wave morphology in Wellens' syndrome (These T changes on ECG are present with a pain free state , but with the recent history of unstable anginal pain)

Type A Wellens' syndrome

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



Type B Wellens' syndrome

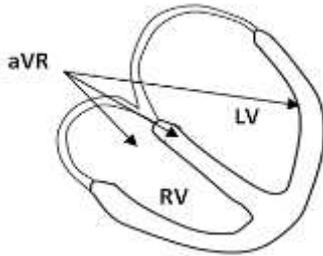
Deeply inverted and symmetrical T-wave



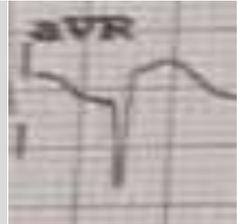
Isoelectric or minimally \uparrow ST without Q wave with preservation of precordial R-wave progression

❑ Alteration in ST segment

- ST elevation in aVR

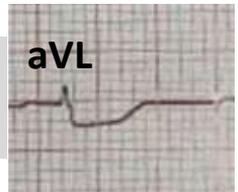


ST elevation > 1mm in lead aVR has been shown to be 80% sensitive and 93% specific for left main or triple vessel disease in patients suffering from symptoms attributed to CAD.

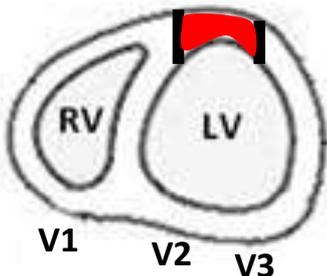


- ST depression in aVL

Any amount of ST depression in aVL with elevation of any degree with two contiguous inferior leads is highly sensitive for inferior OMI.



- Posterior MI (reciprocal changes in anterior leads V1-V3)

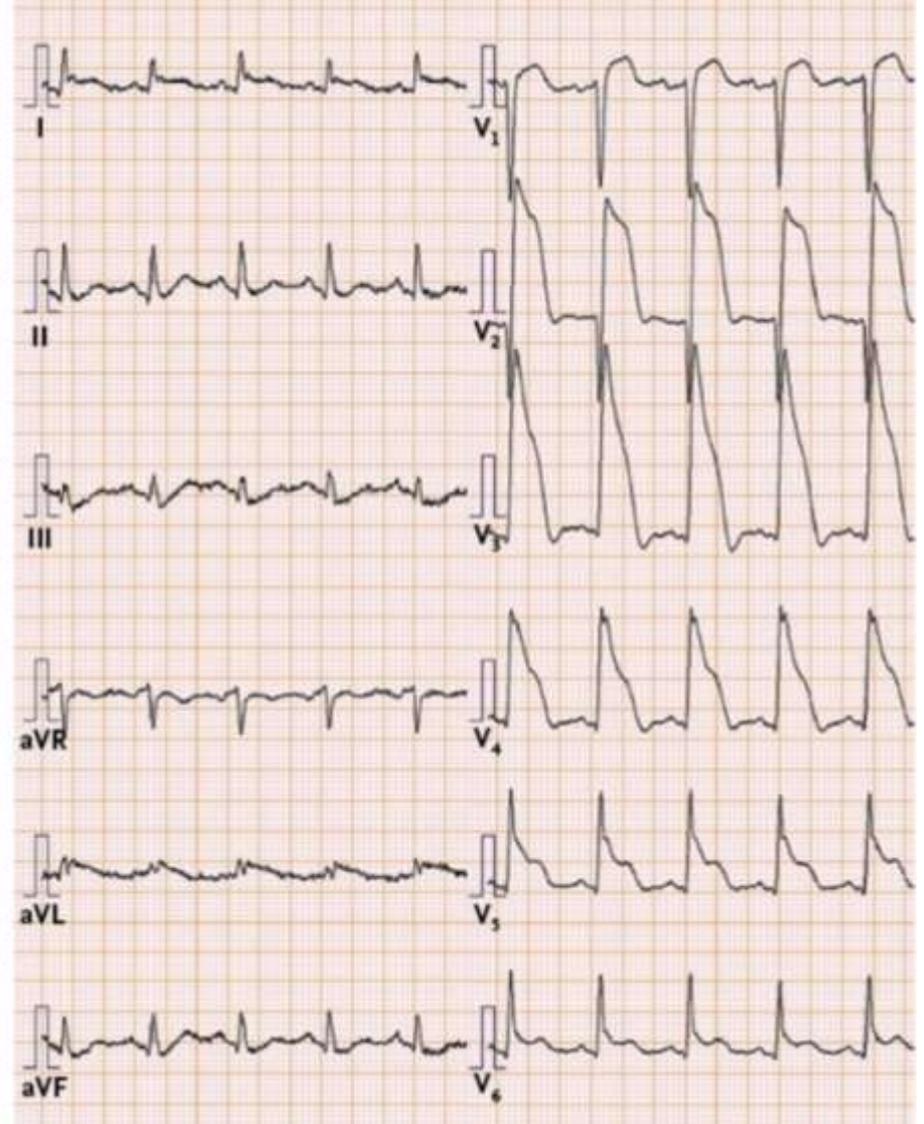
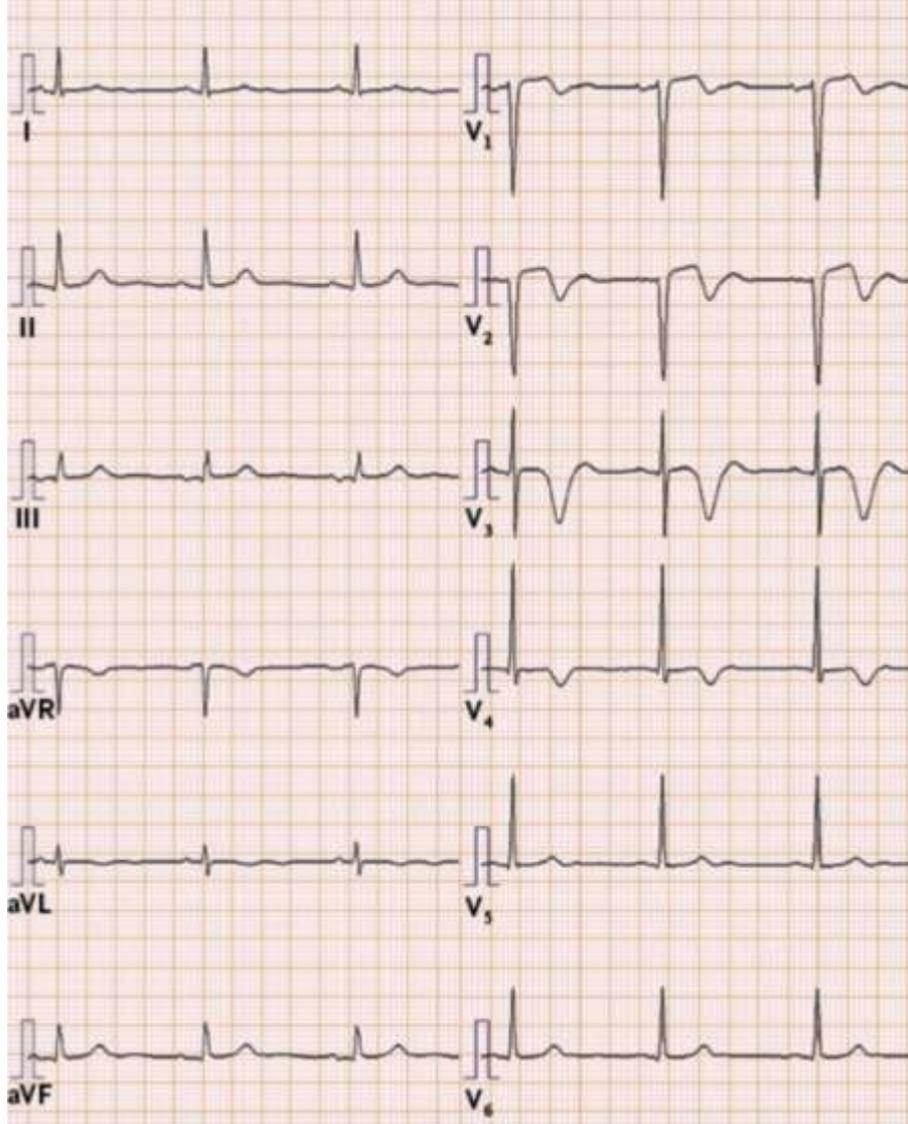


Reciprocal changes in V1 V2 V3 (R ↓ST seg ↑ T)

❑ As Bundle Branch Block Pattern

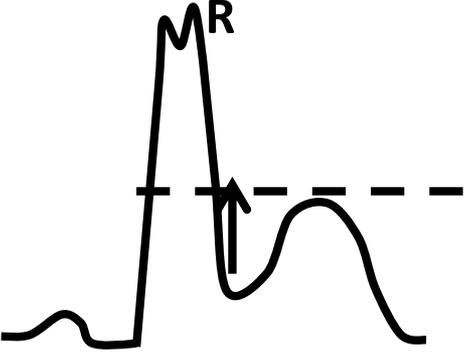
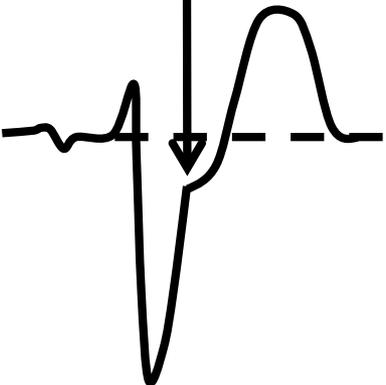
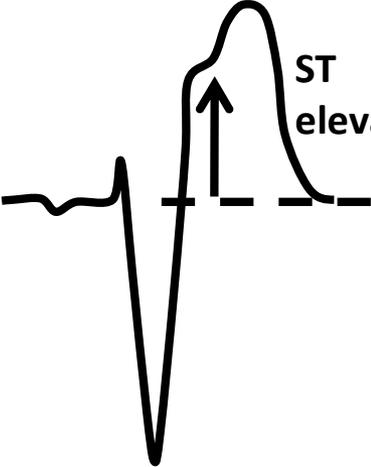
- New bifascicular block
- OMI in the presence of LBBB

Progression of Wellens' syndrome to anterior STEMI



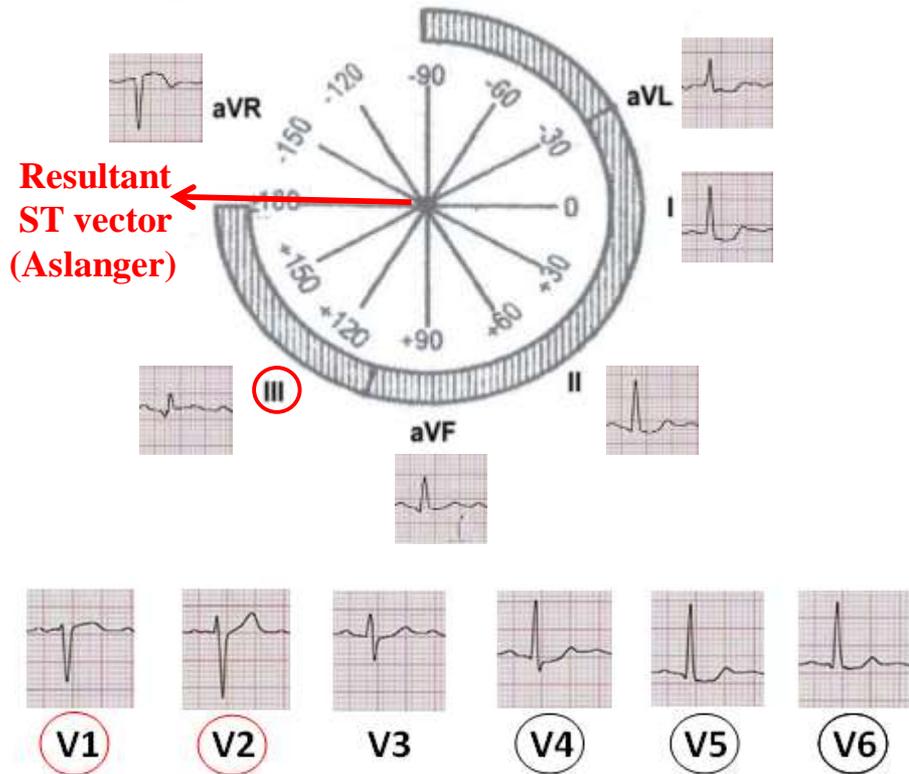
A Wellens' Syndrome → **B Anterior STEMI with "Shark Fin" sign formed by fusion of QRS, ST-seg and T wave**

Occlusion MI in the presence of LBBB (Sgarbossa criteria)

<p>Changes during repolarization</p>	<p>1. Concordant ST elevation ≥ 1mm in leads with a positive QRS complex Sgarbossa criteria Score 5.</p>	<p>2. Concordant ST depression ≥ 1 mm in V1-V3 with negative QRS complex Sgarbossa criteria Score 3.</p>	<p>3. Sgarbossa criteria Excessively discordant ST elevation > 5mm (Score 2) OR Smith-Modified Sgarbossa criteria STE $\geq 25\%$ of the depth of the preceding S-wave.</p>
<p>Illustration by concerned sketches</p>	 <p>Concordant ST elevation</p>	<p>Concordant ST depression</p> 	 <p>ST elevation</p>

- A total score of ≥ 3 is reported to have a specificity of 90% to diagnose a case of MI
- OR Smith Modified Sgarbossa criteria , being present on such ECG.

Aslanger pattern on ECG

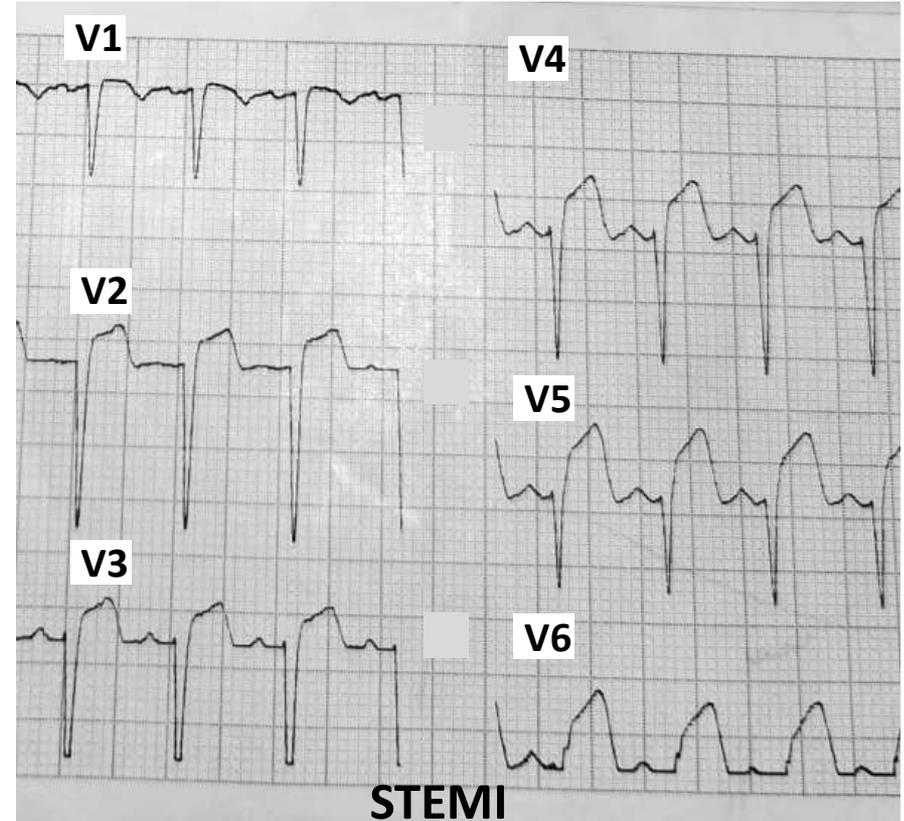
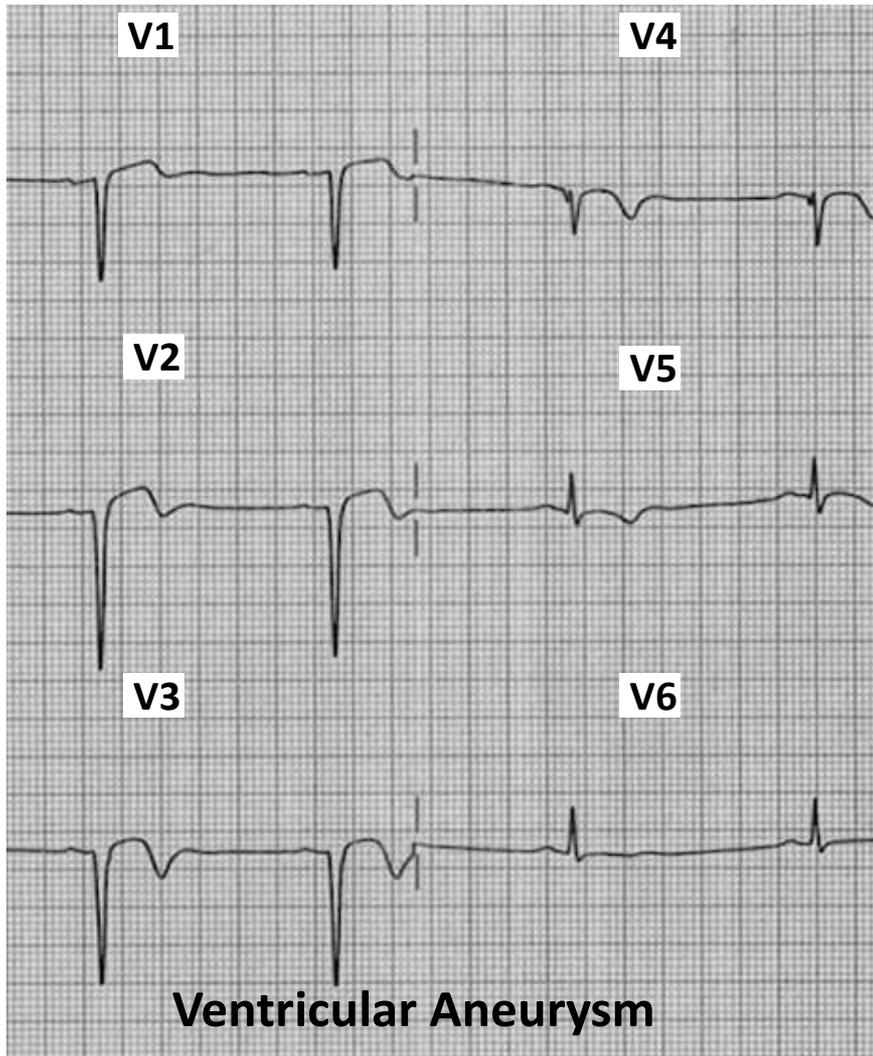


- ST \uparrow only in lead III as evidence of inferior MI
- ST \uparrow in V1>V2
- Concomitant ST \downarrow in any of V4-V6, with a positive/terminally positive T-wave

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, as indicated by **red arrow**, causing ST elevation in leads III and aVR.

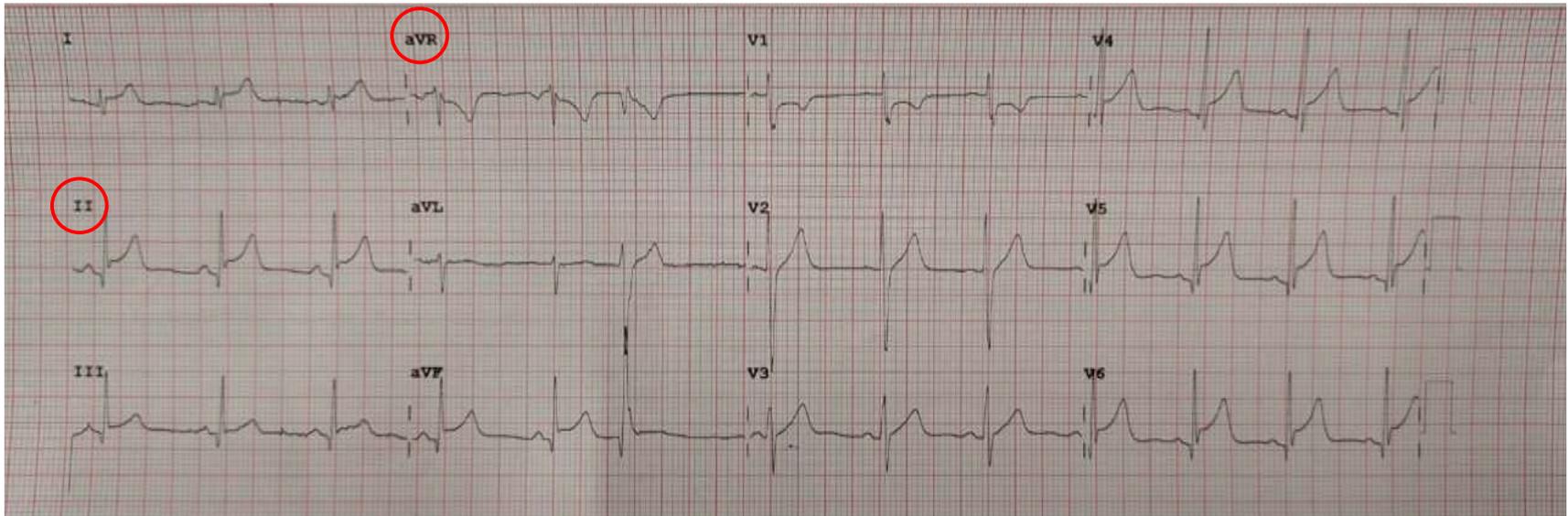
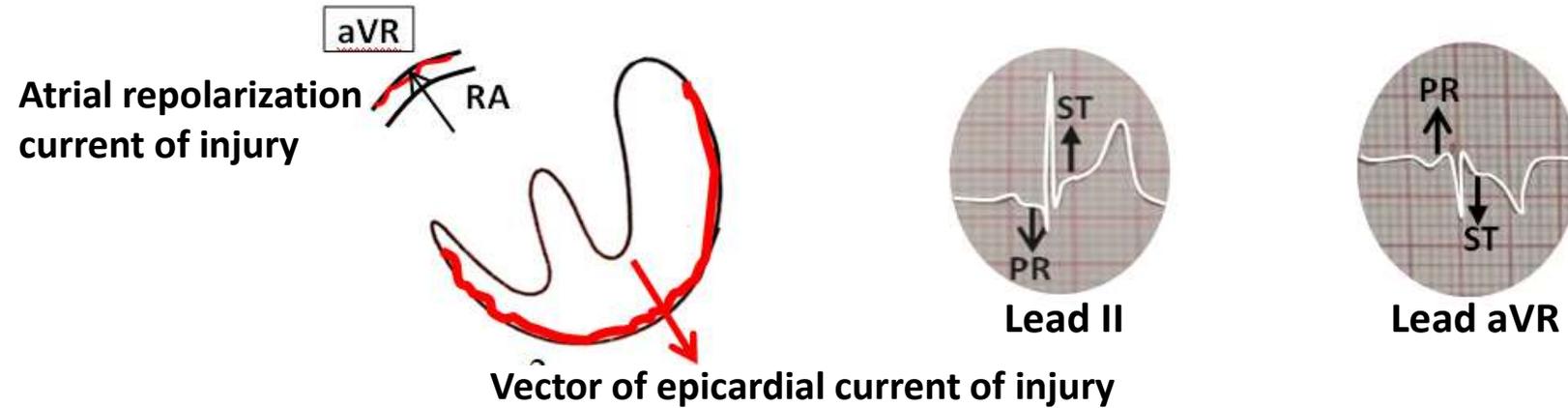
Left Ventricular Aneurysm



- ST elevation > 2 weeks following an acute STEMI commonly seen in precordial leads
- T-wave / QRS ratio < 0.36 in all precordial leads

- T-wave/QRS ratio > 0.36 in any precordial leads favours anterior STEMI

Acute pericarditis : PR-ST segment discordance sign

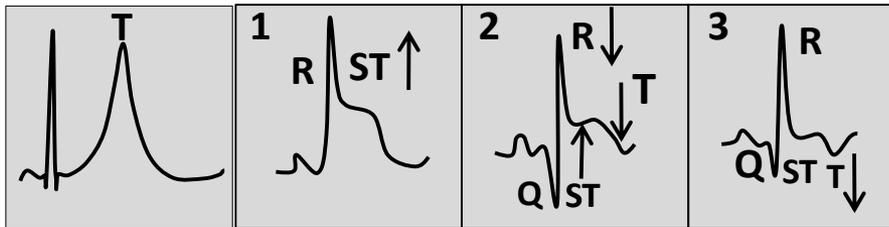


16 years old boy with chest pain of one day duration , Trop I Positive

HOME TAKE MESSAGE

- ❑ History suggestive of anginal pain on effort or at rest
- ❑ ST depression : angina pectoris on effort / chronic stable angina , unstable angina , non-STEMI

ST elevation : STEMI



- ❑ Occlusion MI without ST segment deviation
 - Alteration in T-wave
 - Hyperacute T-wave , Peaked Tall T-wave (de Winter pattern) , Wellens' Syndrome
 - Alternation in ST segment
 - ST elevation in aVR , ST depression in aVL , Posterior MI (reciprocal changes in V1-3) , Bundle branch block pattern (New Bifascicular block , OMI in the presence of LBBB)

Sometimes reverse repolarization in CAD with deep \downarrow T wave inversion

Acute myocardial ischemia , reperfusion of STEMI , Wellens' syndrome , apical HCM

Recognition of the culprit artery involved

LMCA (subtotal occlusion)

Widespread ST depression with ST elevation in aVR ≥ 1 mm

Proximal LAD

- Before septal and diagonal branches : ST \uparrow in V1-6 \pm aVL (1) / qRBBB (C) and ST elevation in lead V1 > aVR
- Proximal to septal branches : ST \uparrow in V1-3

Pre D1 occlusion

ST \uparrow in I , aVL , +/- V2

Mid LAD

ST \uparrow in V2-6 (reciprocal depression in aVL)

Distal LAD

ST \uparrow in V5-6 (reciprocal depression in aVL)

Reciprocal changes in CAD

Over the contralateral side

RCA STEMI

ST \uparrow in III > II + ST \uparrow in V1 and ST \downarrow in V2

LCX STEMI

ST \uparrow in II > III , aVL , I , V5-6

Posterior wall MI

Reciprocal changes in

V1 V2 V3 : R \downarrow ST Seg \uparrow T

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

- Occlusion MI in the presence of LBBB (Sgarbossa criteria)
- Aslanger pattern ECG
- Difference in between left ventricular aneurysm and ECG
- Acute pericarditis : PR-ST segment discordant sign

Thanks

