Localization of the occluded vessel in acute myocardial infarction

Samir Rafia¹* and Amr Kamal²

¹Emeritus Professor, Cardiology Department, Alexandria University, Egypt
²Lecturer of Cardiology, Alexandria University, Egypt

Abstract

This is a review of features in ECG to diagnose the culprit artery responsible for the infarction. Localization of the occluded vessel in acute myocardial infarction is important for many reasons: to know which artery is to dilate and stent; to assess the severity of the lesion; to compare with the echocardiographic area with hypokinesia or akinesia and to differentiate the recent from the old occluded vessel. The ST-segment changes in 12-lead ECG form the basis of diagnosis, management, and prognosis.

Introduction

Infarct related artery (culprit artery) means the coronary artery that is stenosed or occluded by thrombosis, and responsible for an acute coronary syndrome. Usually, there is one culprit artery in majority of cases [1-5].

Coronary artery dominance

The right dominant circulation is defined as RCA supplying the PDA and at least one posterolateral branch. This type is present in around 85% of patients. The non-dominant RCA is seen in 15% of patients. One half of these patients have left dominant circulation which is defined as distal LCx supplying a left PDA and left posterolateral branches. In these cases, the RCA is very small, ends before reaching the crux, and does not supply any blood to the left ventricular myocardium. The remaining patients have balanced/codominant circulation. The specificity of the ECG in acute MI is limited by large individual variations in coronary anatomy & the presence of preexisting coronary artery disease (previous MI, collateral circulation or previous CABG). ECG is limited by its inadequate representation of the posterior, lateral, and apical walls of the left ventricle.

Major steps in the diagnosis of myocardial infarction include identification of the presence of myocardial injury and its severity; and to define the location of the lesion [6-10] (Table 1).

Localization of ischemic area in NSTE-ACS/NSTEMI is more difficult because leads with ST-segment depressions do not point exactly to the ischemic area. Thus ST-segment depressions in leads V3-V4 do not necessarily mean that the ischemia is located in the anterior wall. Therefore, it is commonly stated that ST-segment depressions (as well as T-wave inversions) cannot be used to localize the ischemic area. In exception to this rule, Wellen’s syndrome and de Winter’s sign, both of which are caused by proximal occlusions in the LAD and thus cause anterior wall ischemia/infarction [11-15].

Wellens syndrome refers to specific ECG abnormalities in the precordial T-wave segment, which are associated with

Table 1: Localization of ischemic area in ST segment elevation myocardial infarction (STEMI or STE-ACS).

<table>
<thead>
<tr>
<th>Leads with ST segment elevation</th>
<th>Affected myocardial area</th>
<th>Occluded coronary artery (Culprit)</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1-V2</td>
<td>Septal</td>
<td>Proximal LAD</td>
</tr>
<tr>
<td>V3, V4</td>
<td>Anterior</td>
<td>LAD</td>
</tr>
<tr>
<td>V5, V6</td>
<td>Apical</td>
<td>Distal LAD</td>
</tr>
<tr>
<td>I, aVL</td>
<td>Lateral</td>
<td>LCx</td>
</tr>
<tr>
<td>II, aVL, III</td>
<td>Inferior</td>
<td>90% RCA. 10% LCx</td>
</tr>
<tr>
<td>V7, V8, V9 (reciprocal ST depressions are frequently evident in V1 to V3)</td>
<td>Posterolateral (also referred to as inferobasal or posterior)</td>
<td>RCA or LCx</td>
</tr>
</tbody>
</table>
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critical stenosis of the proximal left anterior descending (LAD) coronary artery [15]. Syndrome criteria include the following:

- Characteristic T-wave changes
- History of anginal chest pain
- Normal or minimally elevated cardiac enzyme levels
- ECG without Q waves, without significant ST-segment elevation, and with normal precordial R-wave progression.

Recognition of this ECG abnormality is of important because this syndrome represents a pre-infarction stage of coronary artery disease (CAD) that often progresses to a severe anterior wall MI.

Identifying myocardial injury and the involved cardiac areas:

- Anterior V1-4
- Apical V5-6
- Lateral I, AVL
- Inferior II, III, aVF

- Posterior ischemia is shown by ST depression in leads V1-3, but posterior injury may best be diagnosed by recording posterior leads V7, V8, and V9.

Right ventricle: The most sensitive sign of RV injury is ST segment elevation > 1 mm in lead V4R. A specific sign of RV injury and/or infarction is ST elevation in V1, with concomitant ST segment depression in V2 associated with ST elevation in the inferior leads.

- Primary anterior area:
  - ST elevation in V1—4
  - areas contiguous are Lateral (L1, aVL), Apical(V5,6)

- Primary inferior area:
  - ST segment elevation in two contiguous leads (II, III, aVF)
  - areas contiguous:

V5,6----------→apical
V7—9, or V1—3, ----------→posterior
V4R ----------→RV

- These contiguous leads suggest more myocardium at risk.
- Primary anterior injury: acute occlusion of the LAD coronary artery, producing changes in the anterior leads (V1-4).

- Earliest findings of occlusion:
- “Hyperacute” changes: ST elevation (injury) with loss of normal ST segment concavity, commonly with tall peaked T waves.
- Acute injury: ST elevation (Figure 2).

Why left anterior fascicle is usually blocked with the RBB? Because the left posterior fascicle receives dual blood supply but both right bundle and left anterior fascicle receive same blood supply.

**Proximal occlusion in LAD:** Results in massive infarction, the more proximal the occlusion, the more the number of leads that display ST-segment elevation. Occlusion proximal to the first septal and diagonal branch causes ST-segment elevations in V1-V4, aVL and I, as well as reciprocal ST-segment depressions in II, III, aVF, aVR. New right bundle branch block is common.

**Distal occlusion of LAD:** Occlusion distal to the first diagonal and first septal will spare the basal parts of the anterior wall. ST-segment elevations are seen in V2-V6. There are no ST-segment elevations in V1, I or aVL. And no reciprocal ST-segment depressions in II, III, aVF and aVR.

**Occlusion in a long LAD:** If the LAD is very long and supplies a significant portion of the inferior wall, occlusion may cause inferior ST-elevations. Thus, a very distal occlusion in the LAD may be deceptive.
Primary inferior process

Usually acute occlusion of the right coronary artery, producing changes in the inferior leads (II, aVF & III).

- Earliest findings:

  **Acute injury**: (ST segment elevation) The J point may “climb up the back” of the R wave (a), or the ST segment may rise up into the T wave (b).

  Evolutionary changes:

  - ST segment elevation decreases and pathologic Q waves develop.
  - T wave inversion may occur in the first 12 hours of an inferior MI, in contrast to that in anterior MI.

Inferior myocardial infarction

The culprit vessel in inferior MI: RCA (in 80%) or CX.

Greater ST-segment elevation in lead III than in lead II & ST-segment depression of more than 1 mm in leads I and aVL suggest involvement of the RCA rather than the CX.

ST-segment depression in leads V1 and V2 suggest concomitant infarction of the posterior wall of the left ventricle.[16-20].

Right ventricular myocardial infarction

ST-segment elevation of > 1 mm in lead V4R with an upright T wave in that lead: This sign is rarely present more than 12 hours after the infarction (Figure 3).

ST-segment elevation in lead V1 in association with ST-segment elevation in leads II, III, and aVF (with greater elevation in III > II) is highly correlated with the presence of right ventricular infarction (Figure 4).

Posterior injury or infarction

Acute occlusion of the CX producing changes in the posterior leads (V7, V8, V9), or reciprocal ST segment depression in leads V1-3.

Identifying the location of the lesion within the artery:

- Proximal LAD occlusion
  1- ST elevation >1 mm in either lead I, aVL, or both
  2- New right bundle branch block
  3- New left anterior fascicular block
  4- New first-degree A-V block

Identifying the location of the lesion within the artery:

- ST-segment elevation in leads V1, V2, and V3 without significant inferior ST-segment depression suggests occlusion of the LAD after the origin of the first diagonal branch.

- ST-segment elevation in leads V1, V2, and V3 with elevation in the inferior leads: Occlusion of the LAD distal to the origin of the first diagonal branch, in a vessel that wraps around to supply the inferoapical region of the left ventricle (Figure 5).

Lead aVR

Lead aVR has a frontal plane vector of −150°, which faces the inside of heart from the right shoulder and is oriented to look at the outflow area of right ventricle and basal part of interventricular septum. Lead aVR ST segment elevation has been reported in cases of acute LMCA occlusion.

New RBBB with a Q wave preceding the R wave in lead V1 is a specific but insensitive marker of proximal occlusion of the LAD in association with anteroseptal myocardial infarction.

**Defining the age of an infarction**: Acute infarction manifests ST segment elevation in a lead with a pathologic Q wave. Old or age indeterminate infarction manifests a pathologic Q wave, with or without slight ST segment elevation or T wave abnormalities [21-27].
ST segment scoring

**Anterior wall myocardial infarction:** Total amount (in millimeters) of ST elevation in the precordial leads (V1-V6). A total of >= 12 mm is a high ST segment score and indicates extensive anterior wall infarction; < 12 mm a low ST segment score.

**Inferior wall myocardial infarction:** Total amount (in millimeters) of ST segment elevation in the inferior leads (II, III, and aVF). A total of >=7 mm is a high ST segment score and indicates extensive inferior wall infarction < 7 mm a low ST segment score.

**Persistent ST segment elevation after acute MI:** Persistent ST segment elevation > 1 mm after a myocardial infarction was a sign of dyskinetic wall motion in the area of infarct. One-half of these patients had echocardiographically defined aneurysm.

**Electrocardiographic predictors of reperfusion:** Resolution of ST-segment elevation: The degree of resolution has proved to be a powerful indicator of short-term (30 day) and long-term (1-year) prognosis.

The absence of ST-segment resolution during the first 90 minutes after the administration of fibrinolytic medications should prompt consideration of rescue angioplasty.

A reduction in ST-segment elevation by more than 70 percent in the leads with maximal elevation is associated with the most favorable outcomes.

**ECG Predictors of Reperfusion:** T-wave inversion that occurs during the first few hours of reperfusion therapy are a highly specific sign of reperfusion T-wave inversion > 4 hours after the start of reperfusion therapy is consistent with the normal electrocardiographic evolution of myocardial infarction and does not indicate that reperfusion has occurred.

An accelerated idioventricular rhythm, (HR 60 to 120 beats per minute is a specific marker of reperfusion. Isolated ventricular premature depolarizations may also be seen with reperfusion. Polymorphic VT, VF can be seen with reperfusion but are rare and should raise the probability of persistent arterial occlusion.

**ECG in AMI with LBBB**

The diagnosis of MI in the presence of LBBB is not easy 50% - 70% of patients with LBBB and suspected MI are not actually having an infarct.

Scoring system for the ECG diagnosis of acute MI with LBBB [27].

* ST elevation => 1 mm discordant with the QRS complex = 5 points.
* ST depression => 1 mm in lead V1, V2 or V3 = 3 points.

A score of =>3 points is consistent with a diagnosis of MI.

### Criteria for diagnosing prior infarction in the setting of LBBB:

- Notching 0.04 seconds in duration in the ascending limb of the S wave in leads V3, V4, or V5 (Cabrera’s sign)
- Notching of the upstroke of the R wave in leads I, L, or V6 (Chapman’s sign)
- Presence of QR complexes in leads I and V5 or V6; or in II, III, and aVF
- Notching in the first 0.04 seconds of the QRS in leads II, III, and aVF

### Limitations of electrocardiogram

Assessment of the site of occlusion of a coronary vessel is most reliable in case of a first episode of MI. This is impaired by multivessel disease, an old MI, collateral circulation and when ventricular activation is altered as in pre-existent left bundle branch block, preexcitation and paced rhythm.

### Conclusion

ECG information can help to expect culprit artery involved before angiography.

### References


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