

## Recognizing electrocardiographically subtle occlusion myocardial infarction and differentiating it from mimics: Ten steps to or away from cath lab

### Elektrokardiyografik olarak belirgin olmayan oklüzyonlu miyokart enfarktüsünü tanımak ve taklitlerinden ayırt etmek: On adımda kateter laboratuvarına veya aksi yöne doğru

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**Summary**– It is increasingly evident that the ST-segment elevation (STE) myocardial infarction (MI)/non-STEMI paradigm that equates STEMI with acute coronary occlusion (ACO) is deceptive. This unfortunate paradigm, adhered to by the current guidelines, misses at least one-fourth of the ACOs, and unnecessarily over-triages a similar fraction of the patients to the catheterization laboratory. Accordingly, we have been calling for a new paradigm, the occlusion/nonocclusion MI (OMI/NOMI). Although this new OMI/NOMI paradigm is not limited to an electrocardiogram (ECG), the ECG will remain the cornerstone of this new paradigm because of its speed, repeatability, noninvasive nature, wide availability, and high diagnostic power for OMI. This review provides a step-by-step approach to ECG for the diagnosis of OMI.

**Özet**– ST-segment yükselmesi (STY) ile akut koroner tıkanıklığı (AKT) eş tutan STY miyokart enfarktüsü (ME)/ST-segment yükselmesiz (STYz) ME paradigmasının yanıltıcı olduğu giderek daha aşikâr hale gelmektedir. Güncel kılavuzlarca savunulan bu talihsiz paradigma AKT'lerin en az dördte birini kaçırmakta, benzer bir oranda hastayı da gereksiz biçimde kateterizasyon laboratuvarına yönlendirmektedir. Bu nedenle, bir süredir oklüzyonlu/oklüzyonsuz ME (OME/NOME) paradigmasına geçiş çağrısında bulunmaktayız. Bu yeni OME/NOME paradigması elektrokardiyografiye (EKG) sınırlı olmasa da EKG; hızı, tekrarlanabilirliği, girişimsel olmayan doğası, yaygın kullanılabilirliği ve OME için yüksek tanısal gücü nedeniyle yine bu paradigmanın kilit taşıını oluşturacaktır. Bu gözden geçirmede, OME tanısı için EKG'ye adım adım bir yaklaşım sunmaktayız.

The fourth universal definition of myocardial infarction (MI) dichotomizes MI as ST-elevation (STE) MI and non-STEMI, on the basis of the presence or absence of—(1) 1-mm STE in any two contiguous leads except V2 and V3; (2) STE in V2 and V3 on the basis of age and gender, where the following cutpoints apply:  $\geq 1.5$  mm elevation in women regardless of age,  $\geq 2.5$  mm in men aged less than 40 years, and  $\geq 2$  mm in men aged 40 years and above.<sup>[1]</sup> However, it is increasingly evident that the hypothesis at the root of this paradigm, which equates the presence or absence of STE at these cutoffs with the presence or absence of acute coronary occlusion (ACO), is incorrect.<sup>[2,3]</sup> This unfortunate assumption causes

the current guideline-recommended, universally-claimed paradigm to miss at least one-fourth of the ACOs, and to unnecessarily refer a similar fraction of the patients to the catheterization laboratory.<sup>[4]</sup>

Accordingly, we have been calling for a new paradigm, the occlusion/nonocclusion MI (OMI/NOMI), as discussed in detail elsewhere.<sup>[2-6]</sup> Although this new OMI/NOMI paradigm is not limited to electrocardiogram (ECG), the ECG will remain the cornerstone of this new paradigm because of its speed, repeatability, noninvasive nature, wide availability, and high diagnostic power. Nevertheless, it should always be kept in mind that the accuracy of any diagnostic test, including ECG, depends on the pretest proba-



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bility. Therefore, ECG should always be interpreted in the context of the clinical picture, according to the presence and characteristics of chest pain, especially when it shows subtle findings.

This review discusses a step-by-step approach to ECG for the diagnosis of OMI (Central Figure).

**When ECG Shows ST Elevation That Meets Universal Criteria**

Owing to the diverse etiology of STE and the relatively low frequency of ACO (only 2-5% of the patients presenting to the emergency department with chest pain actually have OMI),<sup>[7,8]</sup> only half the patients with ECG features meeting STEMI criteria have actual ACO.<sup>[5,8]</sup> To avoid false alarms, before settling on a final diagnosis of OMI, one must first quickly exclude other ECG pathologies that manifest nonischemic STE.

**Step 1: Exclude artifacts**

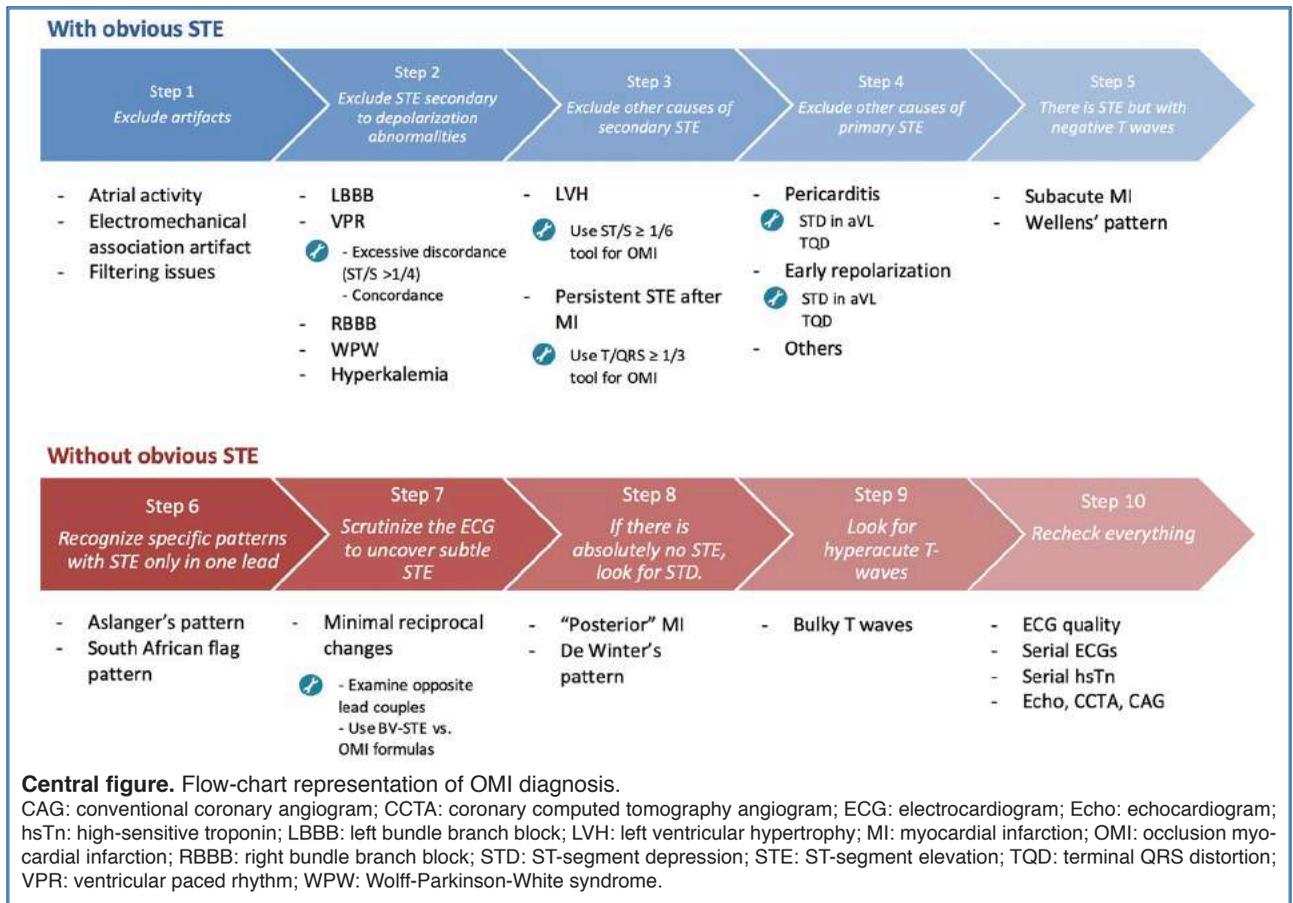
One should be certain that the STE does not have an artifactual appearance. Atrial activity, such as repolarization wave of an inverted P wave<sup>[9]</sup> (Figure 1A)

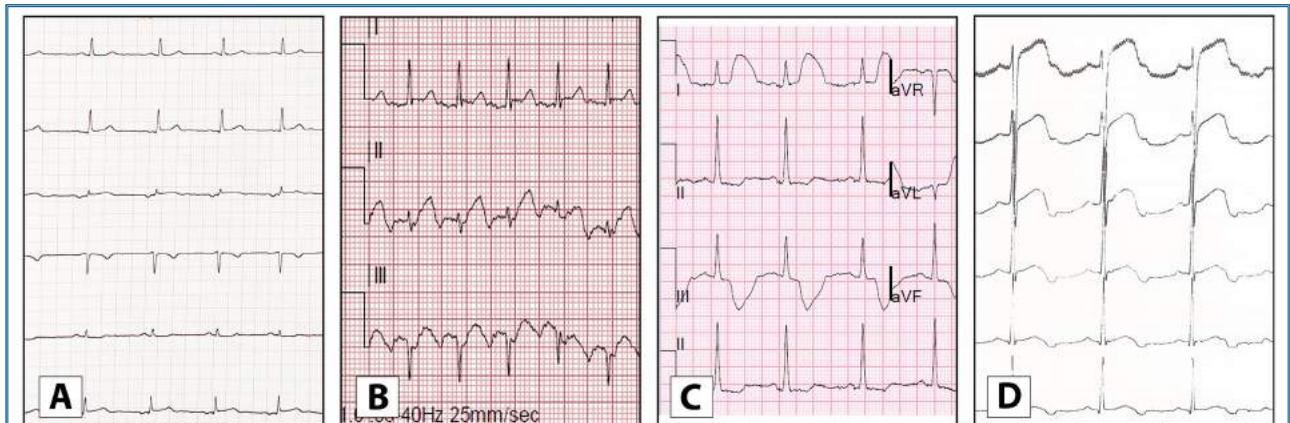
or flutter waves (Figure 1B),<sup>[10]</sup> may be superimposed on the ST-segment and mimic STE. These very frequently distort the inferior leads, resulting in a mild apparent STE, which may manifest variability in STE amplitude if beat-to-beat intervals are changing. A comparison with a previous ECG is also revealing.

**Abbreviations:**

ACO	Acute coronary occlusion
BV	Benign variant
ECG	Electrocardiogram
LAD	Left anterior descending artery
LBBB	Left bundle branch block
LCX	Left circumflex artery
LVH	Left ventricular hypertrophy
NOMI	Nonocclusion myocardial infarction
OMI	Occlusion myocardial infarction
RBBB	Right bundle branch block
STD	ST-segment depression
STE	ST-segment elevation
VPR	Ventricular pace rhythm

Electromechanical association artifact is an unusual type of artifact, which is frequently caused by the tapping of an arterial pulse to the overlying recording electrode.<sup>[11]</sup> Check if one of the limb leads is completely free of abnormal appearance, as one standard limb derivation that does not use this electrode is usually spared.<sup>[12]</sup> It may rarely affect all leads, if





**Figure 1.** Artifactual causes of STE. **(A)** Low-atrial rhythm causing apparent STE in lead II and III. **(B)** Flutter waves causing apparent STE in leads II and III. **(C)** Electromechanical association artifact caused by left radial pulse. Lead II is completely spared. **(D)** Artifactual STE due to high-pass filter set to 0.5 Hz and manual mode is used.

Source: (A and C) Reprinted from Dr. Smith's ECG blog, courtesy of Stephen W Smith, MD. (B) Reprinted from ems12lead.com, Courtesy of Tom Bouthillet.

STE: ST elevation.

both the source of artifact is the right arm electrode and the ECG machine uses leads I and II to calculate lead III instead of directly measuring left arm–left leg potential difference.<sup>[13]</sup> Therefore, another ECG should be acquired after checking electrodes, if the contour of native waves makes sudden and suspicious turns (Figure 1C).

Rarely, ECG filtering may cause artifactual STE. This is especially the case when high-pass filter (lower of the two numbers indicating filter frequencies on ECG paper) is set to 0.5 Hz and real-time mode is used (defibrillators, bedside monitors use this mode by default).<sup>[14]</sup> It always disturbs the leads with deep S waves, and never causes a concordant STE. Slight waviness of the isoelectric line may be a clue (Figure 1D). If suspected, check whether the high-pass filter is set to high, lower it to 0.05 Hz, and repeat ECG in the auto mode. In addition, ensure the same settings were used when comparing serial ECGs.

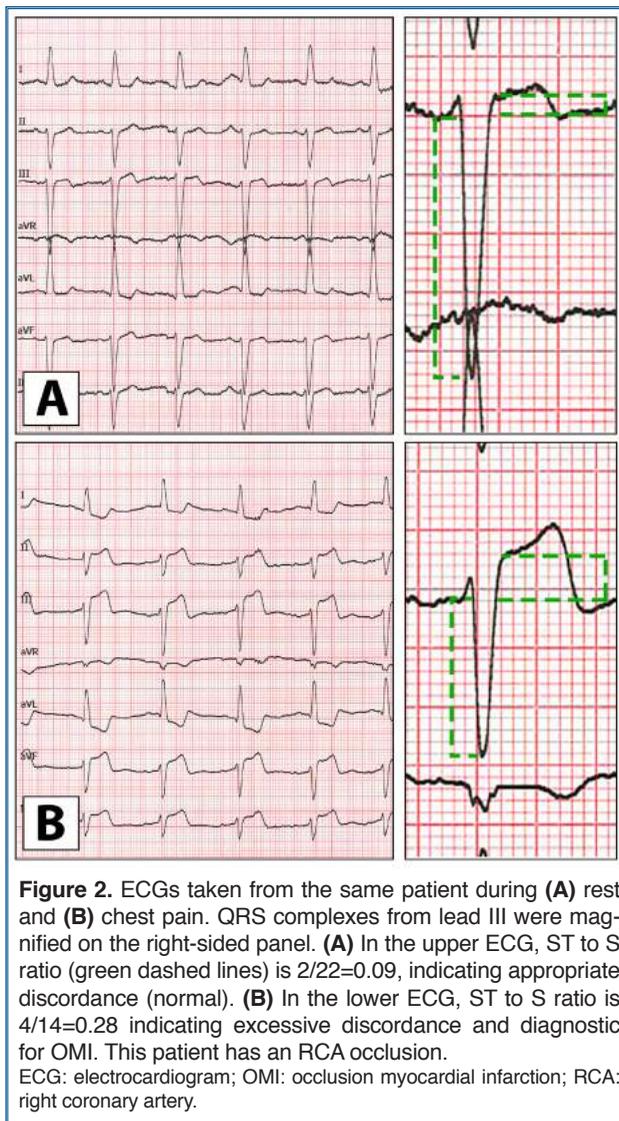
### Step 2: Exclude STE secondary to depolarization abnormalities

The next step is to determine whether the QRS complex is wide. If so, then secondary ST-T-wave abnormalities are expected, even without the presence of an OMI. In these situations, ST deviation is in the direction opposite that of the major deflection of the QRS, and it is proportional to the amplitude of the QRS complex, which is called “appropriate discordance.”

Correspondingly, some discordant STE [as well as appropriately discordant ST depression (STD)] should be anticipated in the left bundle branch block (LBBB), in which nonischemic STE is always present in leads V1–V4, where LBBB registers deep S waves. Nevertheless, if discordant STE is excessive, then concomitant anterior OMI should be considered. Although the widely known Sgarbossa criteria<sup>[15]</sup> define “excessive discordant STE” as  $\geq 5$  mm regardless of the corresponding QRS, a modified version by Smith et al.<sup>[16]</sup> uses the ratio of STE at the J-point to the preceding S-wave. Any STE higher than the one-fourth of the corresponding S-wave is highly specific for OMI (Figure 2). On the contrary, concordant STE, even if just 0.5 mm in just one lead, cannot be explained by the conduction disorder and is always considered pathologic and highly specific for OMI.<sup>[17]</sup>

Right ventricular paced rhythm (VPR) causes a similar conduction abnormality; a recent study showed that the same rules can be used for the diagnosis of OMI.<sup>[18]</sup> Therefore, in the presence of LBBB or VPR, look for—(1) excessive discordant ( $ST/S > 1/4$ ) or (2) even minimal concordant STE; 1 mm of concordant STD in any single lead of V1–V6 should also be taken as a primary change and interpreted accordingly (see below).

Right bundle branch block (RBBB) does not generally cause STE, but sometimes results in a mild discordant STD in V1–V3. This is especially import-



ant for the unfortunate coincidence of RBBB with basal inferolateral (formerly called posterior) MI.<sup>[19]</sup> Although there is no study that specifically explored this coincidence, excessively discordant STD, usually more than 1 mm and out of proportion to the preceding R' wave, should be taken as primary in this occasion. Right ventricular hypertrophy (right axis deviation and large R-wave in V1) with strain may mimic this picture, but, even then, the STD should be assessed in proportion to the excessive R-wave voltage.

The abnormal depolarization that results from Wolff-Parkinson-White syndrome can produce STE that mimics OMI. If a delta wave is present, suspect that STE may be secondary and not primary.<sup>[20]</sup>

Significant hyperkalemia can also cause STE. STE caused by hyperkalemia usually occurs after some QRS prolongation and frequently occurs in V1-V3 with a particularly recognizable pattern reminiscent of Brugada syndrome.<sup>[21]</sup> Less commonly, hyperkalemia can cause STE in the inferior or high lateral leads. In the presence of obvious QRS prolongation, large R-wave in aVR, peaked T waves, or sinoventricular conduction, hyperkalemia should be considered.

### Step 3: Exclude other causes of secondary STE

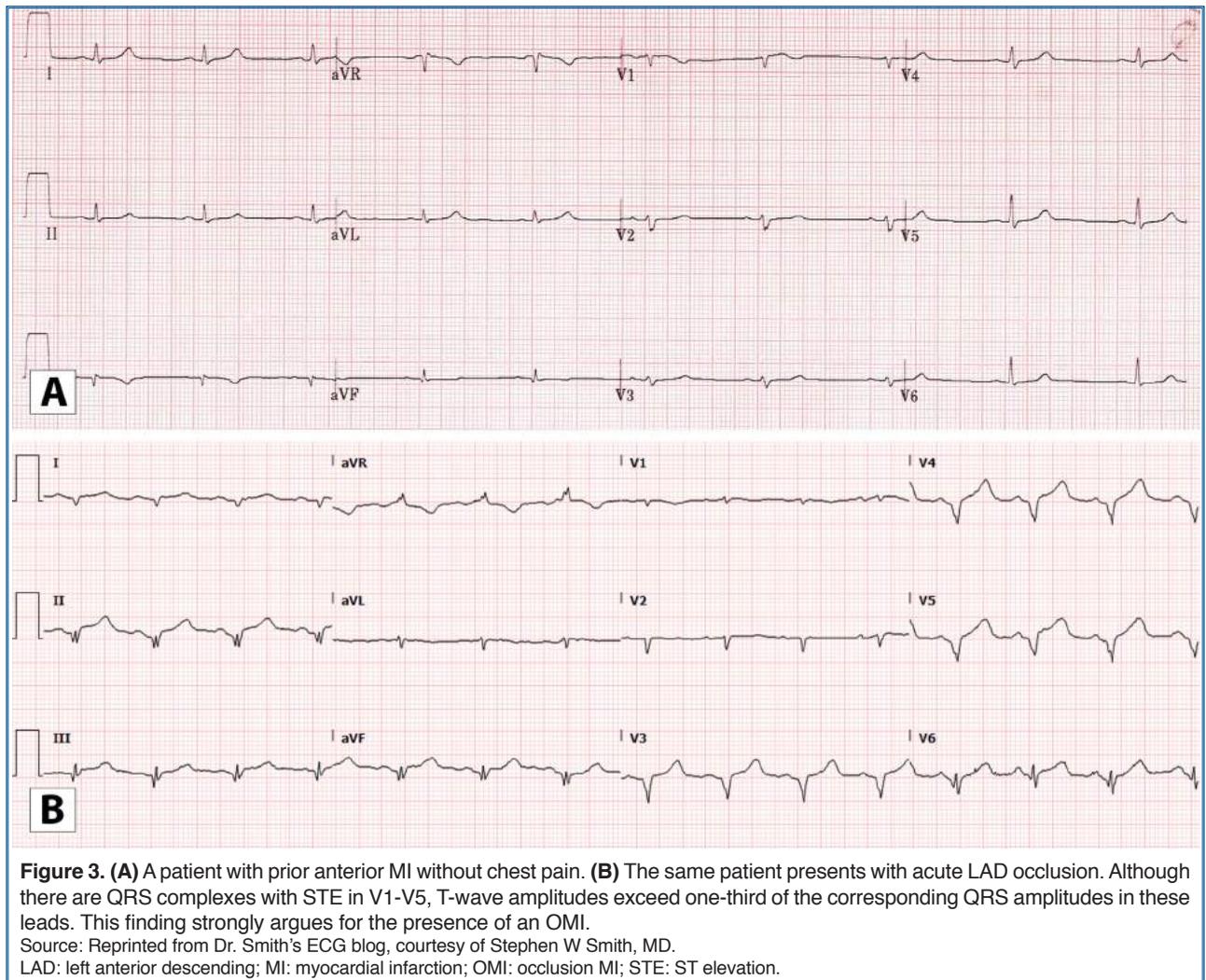
If QRS duration is normal but its amplitude or configuration is abnormal, then there may be pathology that influences both depolarization and repolarization.

Look at the amplitude of the QRS complex. If there is high voltage indicating left ventricular hypertrophy (LVH), some STE may be seen in leads with deep S waves (usually V1-V3) and may mimic STEMI. If the amplitude of STE is more than one-sixth of the amplitude of S-wave in one of these leads, it is highly suspicious for OMI. Convex morphology might be seen in LVH and does not necessarily indicate an OMI. STD in V5 and V6, with reciprocal STE in aVR, occurs frequently in LVH and might mimic ACS.<sup>[22]</sup> LVH with high voltage in the limb leads can also result in nonischemic inferior ST elevation with reciprocal STD in aVL.

In the presence of STE and well-formed Q waves, especially QS waves (complete absence of R-wave), in leads V1-V4, persistent STE after a previous MI (formerly called “ventricular aneurysm pattern”) is a possibility (Figure 3). Because both OMI and persistent STE after a previous MI have Q waves and STE, the size of the T waves provides the critical clue for the distinction, as acute OMI has relatively large T waves.<sup>[23,24]</sup> If there is a single lead among V1-V4, which has a T-wave amplitude higher than one-third of the corresponding total QRS complex, then the diagnosis of acute OMI is likely (Figure 3B).<sup>[24]</sup> False negatives may occur when chest pain duration is >6 hours because T-wave amplitude diminishes as the amount of viable myocardium diminishes. Unfortunately, this rule does not apply to the differentiation of persistent inferior STE caused by a previous inferior MI from inferior OMI, which still remains very challenging.

### Step 4: Exclude other causes of primary STE

If the QRS complex is normal, then evaluate the distribution and maximal location of STE. When there is

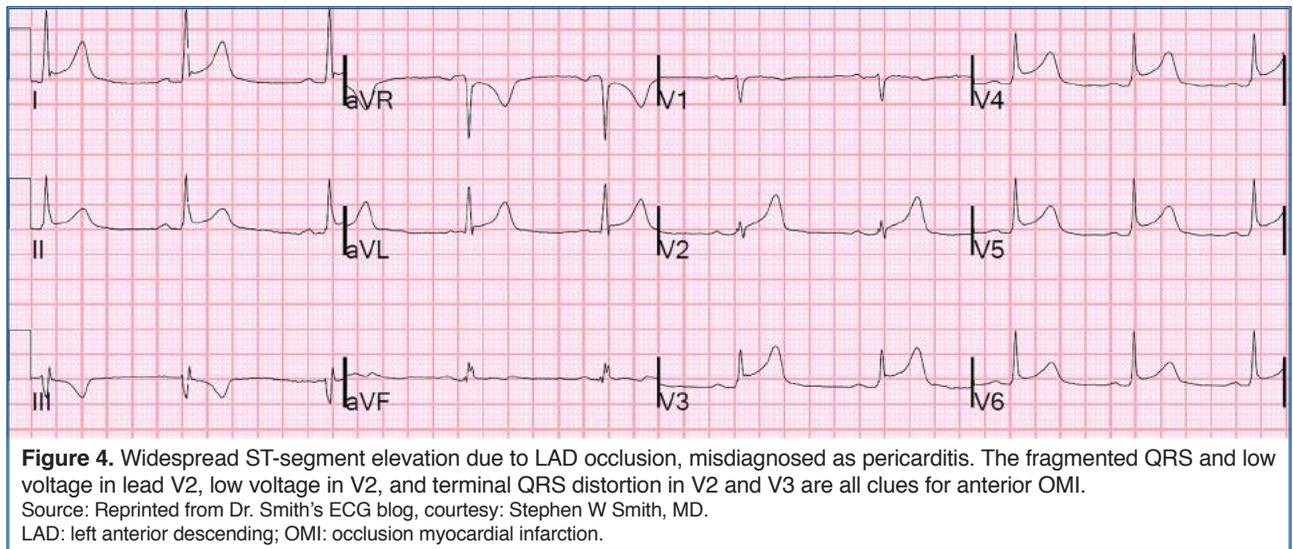


widespread STE, the possibility of pericarditis arises. Although pericarditis is far rarer than OMI, it is overdiagnosed on the ECG, partly because this low pretest probability is widely ignored. The assumption that widespread STE must be due to pericarditis unless the patient has hemodynamic instability is not correct, as this pattern of STE may be seen in patients with certain left anterior descending (LAD) artery occlusions.<sup>[25,26]</sup> Inferolateral OMI with left circumflex (LCX) or right coronary artery occlusion frequently mimics pericarditis, but the latter never has reciprocal STD in aVL.<sup>[27,28]</sup> Thus, the presence of reciprocal STD can be helpful to distinguish OMI from pericarditis. The absence of reciprocal STD is common in LAD occlusion, and should not be relied upon to rule out OMI. We warn that “you diagnose pericarditis at your peril.”

Nevertheless, some indicators that suggest what appears to be OMI might rarely be pericarditis are—(1)

Absence of any reciprocal STD except in aVR and less commonly V1; (2) STE to T-wave ratio  $>1/4$  especially in the left lateral leads (which helps to distinguish pericarditis from both early repolarization (ER) and OMI, but does not distinguish the latter two); (3) STE highest in lateral and inferior leads.<sup>[27]</sup> The presence of any STD in aVL, developing Q waves, convex STE, STE distorting terminal QRS (Figure 4), and prolonged QT interval are signs of OMI.<sup>[28-30]</sup> PR depression is a frequent finding in normal subjects, but PR depression  $>0.8$  mm is suspicious for pericarditis, especially when occurs in both, limbs and precordial leads.<sup>[27,31]</sup>

ER, which is defined as—(1) the presence of an end-QRS notch or slur that is entirely above the baseline on the downslope of a prominent R-wave; (2) the presence of J-point elevation equal to or more than 0.1 mV in two or more contiguous leads, excluding leads V1–V3; and (3) a QRS duration less



than 120 milliseconds,<sup>[32]</sup> is a quite frequent entity with an incidence of 2%-31%. As it can cause inferior and lateral STE, it is a frequent cause of false catheterization laboratory activations. To distinguish ER from inferolateral OMI, check whether lead aVL shows reciprocal STD, which indicates inferior OMI.<sup>[28]</sup> OMI should be suspected especially when there are proportionally large T waves, in addition to STE. Presence of J-wave notching is more common in ER than in OMI but may be present in both.<sup>[33]</sup> When STE is limited to midanterior leads and mild, use one of the anterior OMI versus benign variant (BV) anterior STE differentiation formulas (see below).

There are some other causes of STE, some of which can, generally, only be excluded after a normal angiogram, such as myocarditis<sup>[34]</sup> and Takotsubo cardiomyopathy.<sup>[35]</sup> Brugada syndrome, and also conduction delays caused by sodium channel blocking medications, can cause STE in V1-V3, which may be mistaken for OMI, but their morphology is distinct; type 1 Brugada has an R' wave with down-sloping STE, and type 2 has a saddleback STE appearance, which makes OMI very unlikely.<sup>[36]</sup> Massive pulmonary embolism may present with STE in V1-V3, mimicking right ventricular OMI.<sup>[37]</sup> Tumor invasion has also been reported in some studies.<sup>[38]</sup> Even with a normal angiogram, some cases may still be MI with normal coronary arteries (MINOCA), many of which are ACS due acute coronary thrombosis that undergoes complete autolysis in an artery that has only extraluminal coronary plaque.<sup>[39]</sup>

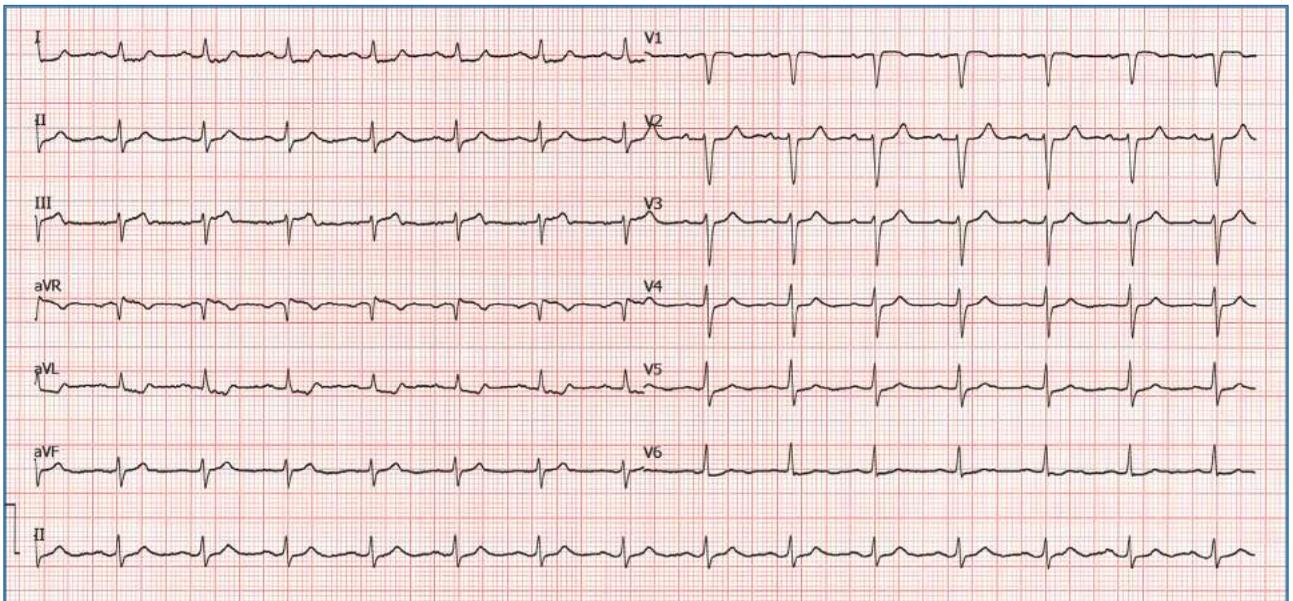
### Step 5: There is STE but with negative T waves

STE may persist in a patient with resolved pain after spontaneous reperfusion or subacute process. In this situation, one may encounter an ECG with STE and negative T waves. These changes on ECG, among others, are more important than the duration of the chest pain in determining the acuity. A larger T-wave and absence of Q waves, or smaller Q waves and absence of QS waves, is indicative of high acuteness.<sup>[40]</sup> Small or shallowly inverted T waves or well-developed Q waves indicate low acuteness. QS wave with shallow T-wave inversion is typical of completed MI. In contrast, deep symmetric T-wave inversion signifies reperfusion in the context of much remaining viable myocardium, usually with preserved R waves (see below).

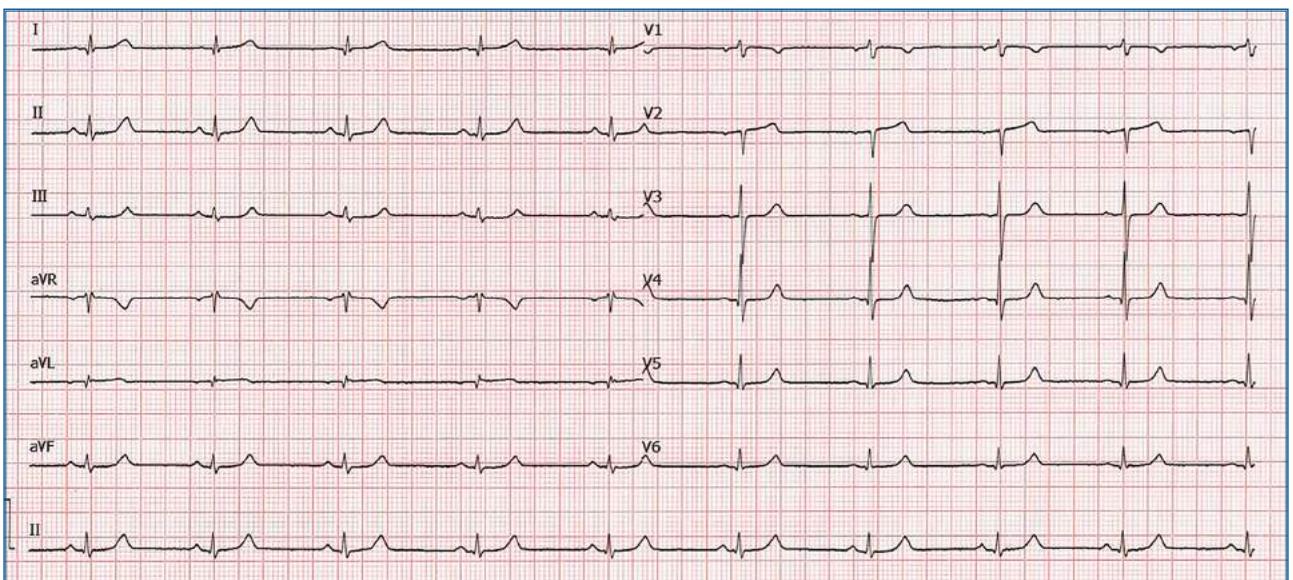
Look for terminal T-wave inversion. Fully developed Q waves and shallow T-wave inversion may indicate a completed infarction, in which urgent catheterization is controversial.<sup>[41]</sup> However, terminal (Wellens' pattern A, found early after reperfusion) or deep symmetric T-wave inversions without STE (Wellens' pattern B, which generally evolves from pattern A), with predominantly preserved QRS complexes, indicates spontaneous reperfusion with an unstable thrombotic lesion remaining in the coronary artery. In combination with preceding chest pain, that is resolved at the time of ECG recording, it is called "Wellens' syndrome."<sup>[42]</sup> In contrast to the notion that Wellens' syndrome is only a phenomenon of the LAD and anterior wall,

the ECG patterns of reperfusion of OMI are seen in all coronary artery distributions and ECG leads. These patients need urgent, although not emergent, referral to the catheterization laboratory. Until then, these patients should also be closely monitored for recurrence of pain, STE or pseudonormalization of

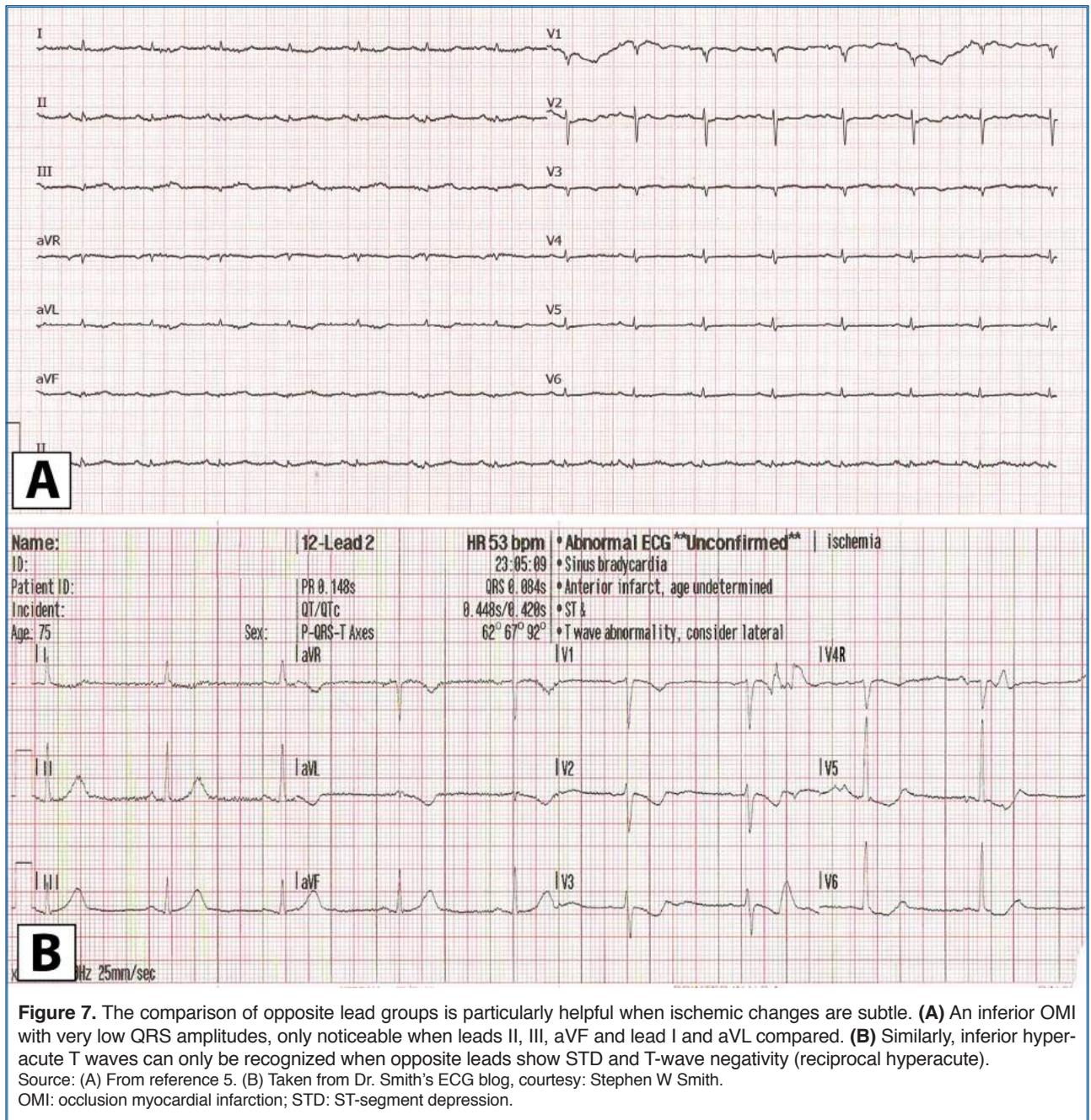
T waves, which indicates re-occlusion.<sup>[43]</sup> One rule of thumb is that a large upright T-wave in occlusion is possible only with a large amount of viable myocardium; a large-inverted T-wave occurs when there is reperfusion (if it is not reciprocal) but only in the context of a large amount of viable myocardium.



**Figure 5.** Aslanger's pattern with STE only in lead III, the ST in V2 lower than ST in V1 and isoelectric, and there is STD in V4-6. This ECG clearly does not meet STEMI criteria. Angiogram showed multivessel disease with acute RCA occlusion. Source: Used with permission from Reference 3. ECG: electrocardiogram; OMI: occlusion myocardial infarction; RCA: right coronary artery; STE: ST elevation; STEMI: ST-segment elevation myocardial infarction.



**Figure 6.** Midanterior OMI with South African flag pattern. There is very subtle STE in lead I, aVL and V2 but not in other anterior leads, which do not meet STEMI criteria. Angiogram showed acute first diagonal artery occlusion. Source: Used with permission from Reference 3. OMI: occlusion myocardial infarction; STE: ST elevation; STEMI: ST-segment elevation myocardial infarction.



**Figure 7.** The comparison of opposite lead groups is particularly helpful when ischemic changes are subtle. **(A)** An inferior OMI with very low QRS amplitudes, only noticeable when leads II, III, aVF and lead I and aVL compared. **(B)** Similarly, inferior hyperacute T waves can only be recognized when opposite leads show STD and T-wave negativity (reciprocal hyperacute). Source: (A) From reference 5. (B) Taken from Dr. Smith’s ECG blog, courtesy: Stephen W Smith. OMI: occlusion myocardial infarction; STD: ST-segment depression.

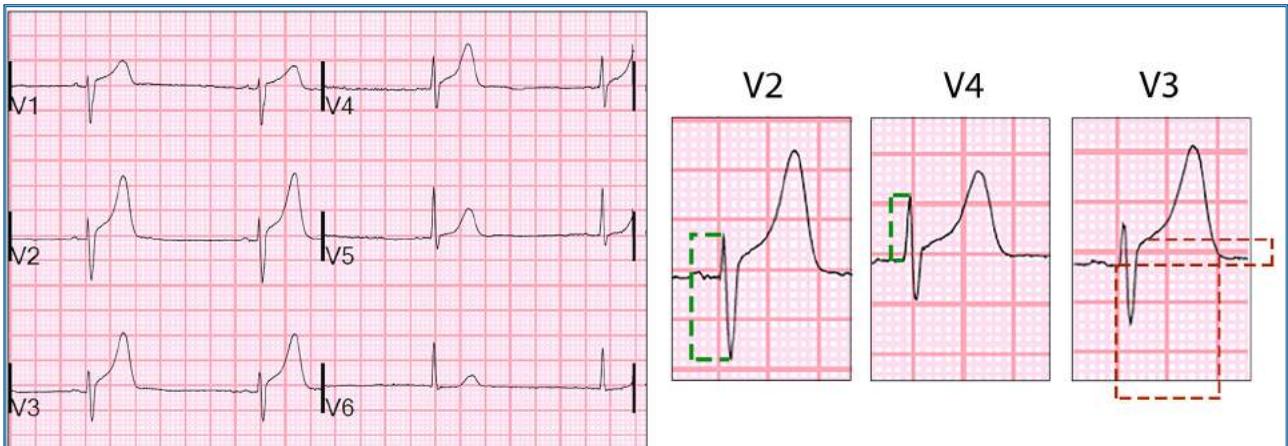
**When There Is No Obvious STE**

**Step 6: Recognize specific patterns with STE only in one lead**

A peculiar configuration with only STE in lead III, but not in lead II and aVF may sometimes be encountered, which is also known as Aslanger’s pattern.<sup>[44]</sup> In this pattern, (1) there is STE only in lead III, (2) ST in V2 <ST in V1, and (3) STD in V4–V6. This may indicate a limited inferior OMI

with multivessel disease in the right clinical context (Figure 5).

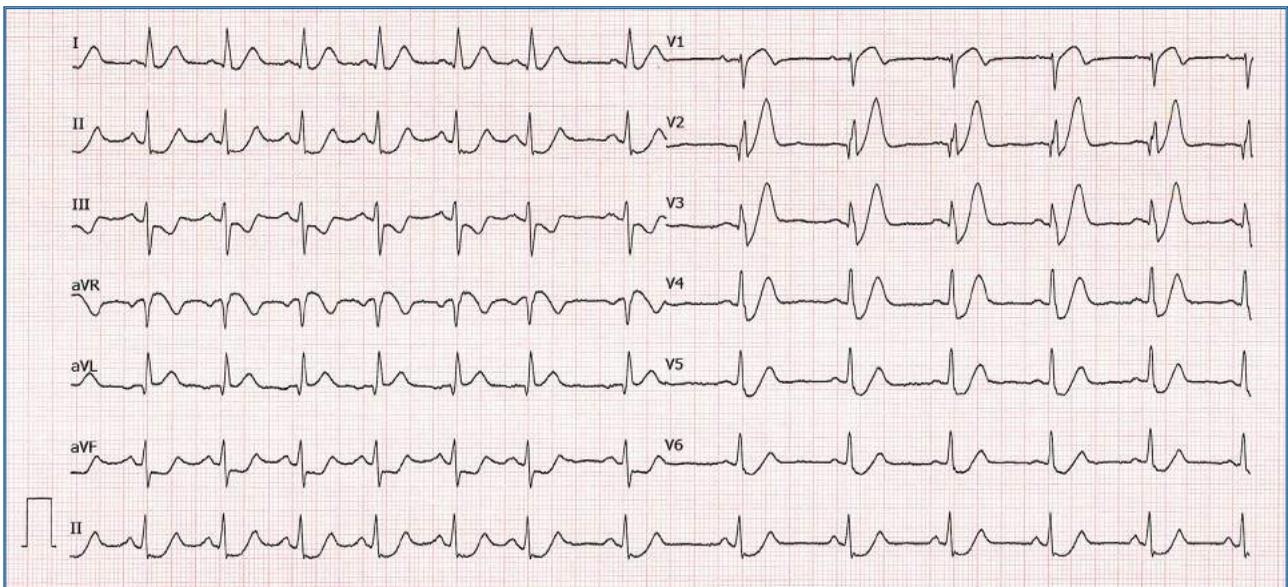
Another pattern with somewhat noncontiguous STE is midanterior OMI caused by first diagonal or intermediate artery occlusion. In this pattern, there is STE only in V2, and STD in the rest of precordial derivations (also known as South African flag sign), and there is STE in lead I and aVL (Figure 6).<sup>[45]</sup>



**Figure 8.** According to the simplified formula for differentiation of OMI from BV-STE, the higher the QRS amplitude in V2 and the R-wave amplitude in V4, and the lower the STE in V3 and the shorter QT interval, the less likely is an LAD occlusion. The sum of QRS amplitude in V2 and R-wave amplitude in V4 (green dashed lines) is 19.5 mm. The sum of STE at 60 msec after J-point and QT (red dashes) 12.5 mm. The difference is 7 mm, which is below 12 mm and indicates LAD occlusion.

Source: Modified from Dr. Smith's ECG blog, courtesy: Stephen W Smith, MD.

BV-STE: Benign variant-ST elevation; LAD: left anterior descending; OMI: occlusion myocardial infarction.



**Figure 9.** De Winter's pattern with upsloping STD, especially at J-point, and hyperacute T waves. Although coronary angiography showed proximal LAD occlusion, this case does not fulfill the requirements of the STEMI definition.

Source: Taken from reference 5.

LAD: left anterior descending; STD: ST-segment depression; STEMI: ST-segment elevation myocardial infarction.

### Step 7: Scrutinize the ECG to uncover subtle STE

One of the limitations of the STEMI approach is that it removes the STE analysis from its context within the entire QRS complex. Repolarization always has some proportional relationship to depolarization, and thus STE is often very high in LVH and very low in leads with low QRS voltage. Furthermore, patients with normal STE have a different QRS morphology than patients with ischemic STE; namely, they have higher R-wave and QRS amplitude. Large T-wave

size, by width, height, and bulk, is an important clue that any STE is ischemic; its absence is evidence against ischemia. Finally, mild QT prolongation is one of the cardinal manifestations of ischemia.<sup>[30]</sup> Therefore, any STE should always be interpreted in its QRST context.

Three-fourths of the OMIs that are missed on first glance can be recognized by subtle STE.<sup>[5]</sup> Most of the missed STEs on first glance manifest in the context of proportionally low-amplitude QRS complexes

and/or have proportionally large T waves. Sometimes the most recognizable feature is reciprocal STD, which should prompt one to scrutinize the opposite lead for any subtle STE (Figure 7A). It should also be emphasized that ECGs with an abnormal QRS, especially limb lead LVH, LBBB, Wolf-Parkinson-White, known inferior LV aneurysm, or paced rhythm, often have reciprocal STD at baseline, without ischemia. In contrast, ECGs of patients with myocarditis often manifest reciprocal STE that might be impossible to distinguish from STEMI without an angiogram.<sup>[22]</sup>

To uncover these, a good starting point is “inferior leads and aVL couple” where QRS amplitude is usually low. First, look for any STE or large T waves in inferior leads, especially lead III, and then look to aVL for any STD of any amount, as well as T-wave inversion, as signs that support the suspicion that minimal STE in inferior leads may be inferior OMI (Figure 7B). The inverted T-wave may be reciprocally large relative to the QRS (reciprocally hyperacute).<sup>[28]</sup>

Next, look for any STD in the inferior leads, as a clue to the LCX, diagonal or proximal LAD occlusion. These may manifest as an extremely minimal STE in aVL, or only a T-wave that is larger relatively to the QRS. There may be inverted T waves in inferior leads, which may also be reciprocally large relative to the QRS (reciprocally hyperacute). To differentiate the culprit artery, look at the precordial leads. If there is STD or an impression of it in V2, then it is LCX occlusion with basal inferolateral (“posterior”) extension. If there is STE only in V2, and STD in the rest of precordial derivations, this indicates mid-anterolateral MI (see above). If there is anterior STE, then it is anterior MI probably caused by the LAD occlusion proximal to the first diagonal artery. However, localization of the culprit lesion may be spurious when ischemic changes are very minimal.

If one is convinced that there is no reciprocal ST deviation or hyperacute T waves in limb leads, check if there is reciprocal STD in V5-V6. This is not expected in BV-STE and makes any STE in the right precordial leads highly suspicious for anterior OMI.

If not, there may still be an anterior OMI without reciprocal changes. Without confirming reciprocal STD, this is a hard task as anterior leads V2-V4 normally show some degree of STE. Guidelines ac-

cordingly increased STE limit for V2-V3 according to age and sex, but this is not always helpful because ischemic STE is often not so elevated. Although upward convexity or a straight ST segment is specific for ischemic STE, it is absent in approximately 40% of the anterior OMI.<sup>[33,46]</sup> Normal STE is virtually always upwardly concave in all of the leads V2-V6. If there is at least 1 mm STE in one of the leads V2-V4, formulas help in distinguishing BV-STE from anterior OMI, using these four variables—STE at 60 ms after the J-point in V3, total QRS amplitude in V2, R-wave amplitude in V4, and the QT interval.<sup>[33,47-49]</sup> The higher the QRS amplitude in V2 and the R-wave amplitude in V4, and the lower the STE in V3 and the shorter the QT interval, the lesser are the chances of an LAD occlusion. Start with the simple rule, subtract the latter two from the former two. If the result is lower than 12, LAD OMI is highly likely (Figure 8).<sup>[49]</sup> When the result is borderline, use a more sophisticated 4-variable formula,<sup>[47]</sup> which is externally validated<sup>[48]</sup> and has been made into online calculators and applications for smartphones (iPhone: subtleSTEMI; Android: ECG SMITH). Keep in mind that formulas may also be false positive when the variables are influenced by other conditions (low QRS voltage due to obesity, pericardial fluid, myocarditis; or excessively long QT segment due to various causes).

### Step 8: If there is absolutely no STE, look for STD

If there is any inferior STD, look for any hyperacute T-wave in precordial leads, as sometimes reciprocal inferior STD precedes significant STE in anterior leads (see below), or is more apparent.

Another special pattern with some STD is de Winter’s pattern,<sup>[50]</sup> which consists of STD (especially J-point depression) preceding large, hyperacute T waves in the precordial leads (Figure 9). Such T waves are not necessarily tall, but they are wide and “bulky” like typical hyperacute T waves. The presence of de Winter’s T-waves indicates a proximal LAD complete or near complete ACO, and should be treated immediately without waiting for STE to appear, as it may or may not evolve into STE before substantial loss of myocardium.<sup>[51]</sup>

If there is only STD, especially when maximal in the leads V2-V4, basal inferolateral (formerly posterior) OMI is most likely.<sup>[52]</sup> Posterior leads may be helpful. If there is STD in multiple leads, especially if

maximal out to V5 and V6, and including limb leads I and II, and which may be associated with reciprocal STE in leads aVR or V1, this may indicate critical multivessel or left main ACS, but without full occlusion, in the appropriate clinical situations, especially when a relatively normal prior ECG is present.<sup>[53]</sup> Although these patterns do not indicate OMI, they suggest an extensive area at risk and correspondingly need early intervention. It should be remembered that STE in aVR is a result of simple mathematical relationship among limb leads; therefore, do not overstate its importance in isolation.<sup>[54]</sup>

### Step 9: Look for hyperacute T-waves

Although hyperacute T-waves lack a formal definition, they are often insufficiently described by their amplitude. We believe that they are better defined by their “bulk,” and this bulk must be relative to the QRS size. Bulk is measured by the area under the curve, which takes into account the height, width, and amount of upward ST concavity (the straighter the ST segment, the more the area, and the “bulkier” the T-wave). They may have reciprocal findings as well manifesting as reciprocal hyperacute T waves (negative and bulky). Always interpret T waves within their QRS and ST-segment context. If there is a suspicion for anterior hyperacute T waves, use BV-STE versus anterior OMI formulas, but especially use serial ECG tracings.<sup>[33,47-49]</sup>

### Step 10: When everything looks normal, recheck everything if the clinical suspicion is high

Ensure that all leads are recorded appropriately. Many ECG devices calculate other limb leads from lead I and II,<sup>[54]</sup> a blank lead II may completely eclipse an inferior MI. Additionally, baseline artifacts may obscure many subtle findings discussed above.<sup>[13]</sup> Repeat ECG if any lead is missing or uninterpretable because of the artifacts.

Consider additional leads. Right-sided leads are not necessary, and may cause false positives, but posterior leads may uncover or clear up the suspicion in an additional 5-10% of the OMIs.<sup>[55]</sup> However, in a patient at high suspicion of OMI, STD maximal in V2-V4 is OMI until proven otherwise, and negative posterior leads should not dissuade from that decision, as STD in anterior leads must always manifest as STE in posterior leads, except that the magnitude of the various vectors is changed owing to interven-

ing air (lung) and distance.<sup>[56]</sup> When additional leads are also not revealing, a feasible method may be to acquire a standard 12-lead ECG every 15 minutes, more frequent if pain has a crescendo pattern, for the first hour.

Serial hs-troponin, bedside echocardiogram, coronary computed tomography, and even coronary angiogram may be used if clinical suspicion is very high. However, it should be noted that any ECG sign is dependent on pretest probability of the patient, and as the ECG sign gets more subtle, one needs higher pretest probability. Therefore, clinicians must be aware of the false alarms, which are easily generated in inappropriate clinical contexts, and eloquently advocate their opinion when the clinical situation and ECG are compatible.

### Conclusion

There are many ECG tools available beyond the STE criteria for recognizing OMI, and for differentiating OMI from OMI mimics. The entirety of the ECG must be assessed, including Q waves, R waves, S waves, ST segments, T waves, and the QT interval. The amplitude and width of all waves contribute to a nuanced interpretation of the ECG. This requires a significant investment in training, but in the future may be taught to deep convolutional neural networks.<sup>[57]</sup> The diagnosis of OMI may be missed in the ECG especially in the hands of those who lack expertise, and even by experts at times. Thus, it is essential to remain humble when confronted with a patient who may be actively infarcting viable myocardium, and to use all the methods at one's disposal to identify these patients expeditiously.

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### REFERENCES

1. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College

- of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal Definition of Myocardial Infarction. *J Am Coll Cardiol* 2018;72:2231-64. [\[Crossref\]](#)
2. Aslanger EK, Meyers PH, Smith SW. Time for a new paradigm shift in myocardial infarction. *Anatol J Cardiol* 2021;25:156-62. [\[Crossref\]](#)
  3. Aslanger EK, Meyers PH, Smith SW. STEMI: a transitional fossil in MI classification? *J Electrocardiol* 2021;65:163-9. [\[Crossref\]](#)
  4. Khan AR, Golwala H, Tripathi A, Bin Abdulhak AA, Bavishi C, Riaz H, et al. Impact of total occlusion of culprit artery in acute non-ST elevation myocardial infarction: a systematic review and meta-analysis. *Eur Heart J* 2017;38:3082-9. [\[Crossref\]](#)
  5. Aslanger EK, Yıldırım Türk Ö, Şimşek B, Bozbeyoğlu E, Şimşek MA, Karabay CY, et al. Diagnostic accuracy of electrocardiogram for acute coronary occlusion in myocardial infarction (DIFOCCULT Study). *Int J Cardiol Heart Vasc* 2020;30:100603. [\[Crossref\]](#)
  6. Meyers HP, Smith SW. Prospective, real-world evidence showing the gap between ST elevation myocardial infarction (STEMI) and occlusion MI (OMI). *Int J Cardiol* 2019;293:48-9. [\[Crossref\]](#)
  7. Sandoval Y, Smith SW, Thorsden SE, Bruen CA, Carlson MD, Dodd KW, et al. Diagnostic performance of high sensitivity compared with contemporary cardiac troponin I for the diagnosis of acute myocardial infarction. *Clin Chem* 2017;63:1594-604. [\[Crossref\]](#)
  8. Hillinger P, Strebel I, Abächerli R, Twerenbold R, Wildi K, Bernhard D, et al. Prospective validation of current quantitative electrocardiographic criteria for ST-elevation myocardial infarction. *Int J Cardiol* 2019;292:1-12. [\[Crossref\]](#)
  9. Manne JRR. Atrial repolarization waves (Ta) mimicking inferior wall ST segment elevation myocardial infarction in a patient with ectopic atrial rhythm. *Case Rep Med* 2018;2018:1015730. [\[Crossref\]](#)
  10. Antonis Manolis IK. Atrial flutter mimicking acute myocardial infarction. *Hospital Chronicles* 2015;10:1-3.
  11. Aslanger E, Yalin K. Electromechanical association: a subtle electrocardiogram artifact. *J Electrocardiol* 2012;45:15-7. [\[Crossref\]](#)
  12. Aslanger E. An unusual electrocardiogram artifact in a patient with near syncope. *J Electrocardiol* 2010;43:686-8. [\[Crossref\]](#)
  13. Aslanger E, Yalin K, Golcuk E, Oncul A. An example of apparently normal electrocardiogram originating from incorrect electrocardiographic acquisition in a patient with ST-segment elevation myocardial infarction. *J Electrocardiol* 2010;43:222-3. [\[Crossref\]](#)
  14. Aslanger E, Ohtaroglu Tokdil K, Tokdil H, Yalin K. Filtering electrocardiogram: Music, math and ST-elevation myocardial infarction. *Arch Turk Soc Cardiol* 2021;49(6):509-11.
  15. Sgarbossa EB, Pinski SL, Barbanelata A, Underwood DA, Gates KB, Topol EJ, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. *N Engl J Med* 1996;334:481-7. [\[Crossref\]](#)
  16. Smith SW, Dodd KW, Henry TD, Dvorak DM, Pearce LA. Diagnosis of ST-elevation myocardial infarction in the presence of left bundle branch block with the ST-elevation to S-wave ratio in a modified Sgarbossa rule. *Ann Emerg Med* 2012;60:766-76. [\[Crossref\]](#)
  17. Dodd KW, Elm KD, Smith SW. Comparison of the QRS complex, ST-segment, and T-wave among patients with left bundle branch block with and without acute myocardial infarction. *J Emerg Med* 2016;51:1-8. [\[Crossref\]](#)
  18. Dodd KW, Zvosec DL, Hart MA, Glass 3rd G, Bannister LE, Body RM, et al. Electrocardiographic diagnosis of acute coronary occlusion myocardial infarction in ventricular paced rhythm using the modified sgarbossa criteria. *Ann Emerg Med* 2021;S0196-0644(21)00249-3. [\[Crossref\]](#)
  19. Okabe M, Nomoto J, Fukuda K, Arakawa K, Kikuchi M. Right bundle block in a patient with acute posterior myocardial infarction. *Jpn Circ J* 1997;61:78-81. [\[Crossref\]](#)
  20. Güler N, Eryonucu B, Bilge M, Erkoç R, Türkoğlu C. Wolff-Parkinson-White syndrome mimicking acute anterior myocardial infarction in a young male patient--a case report. *Angiology* 2001;52:293-5. [\[Crossref\]](#)
  21. Chawla KK, Cruz J, Kramer NE, Towne WD. Electrocardiographic changes simulating acute myocardial infarction caused by hyperkalemia: report of a patient with normal coronary arteriograms. *Am Heart J* 1978;95:637-40. [\[Crossref\]](#)
  22. Miranda DF, Lobo AS, Walsh B, Sandoval Y, Smith SW. New insights into the use of the 12-lead electrocardiogram for diagnosing acute myocardial infarction in the emergency department. *Can J Cardiol* 2018;34:132-45. [\[Crossref\]](#)
  23. Klein LR, Shroff GR, Beeman W, Smith SW. Electrocardiographic criteria to differentiate acute anterior ST-elevation myocardial infarction from left ventricular aneurysm. *Am J Emerg Med* 2015;33:786-90. [\[Crossref\]](#)
  24. Smith SW. T/QRS ratio best distinguishes ventricular aneurysm from anterior myocardial infarction. *Am J Emerg Med* 2005;23:279-87. [\[Crossref\]](#)
  25. Bozbeyoğlu E, Yıldırım Türk Ö, Aslanger E, Şimşek B, Karabay CY, Özveren O, et al. Is the inferior ST-segment elevation in anterior myocardial infarction reliable in prediction of wrap-around left anterior descending artery occlusion? *Anatol J Cardiol* 2019;21:253-8.
  26. Bozbeyoğlu E, Aslanger E, Yıldırım Türk Ö, Şimşek B, Karabay CY, Türer A, et al. An algorithm for the differentiation of the infarct territory in difficult to discern electrocardiograms. *J Electrocardiol* 2018;51:1055-60. [\[Crossref\]](#)
  27. Spodick DH. Differential characteristics of the electrocardiogram in early repolarization and acute pericarditis. *N Engl J Med* 1976;295:523-6. [\[Crossref\]](#)
  28. Bischof JE, Worrall C, Thompson P, Marti D, Smith SW. ST depression in lead aVL differentiates inferior ST-elevation myocardial infarction from pericarditis. *Am J Emerg Med* 2016;34:149-54. [\[Crossref\]](#)
  29. Lee DH, Walsh B, Smith SW. Terminal QRS distortion is present in anterior myocardial infarction but absent in early repolarization. *Am J Emerg Med* 2016;34:2182-5. [\[Crossref\]](#)
  30. Kenigsberg DN, Khanal S, Kowalski M, Krishnan SC. Prolongation of the QTc interval is seen uniformly during early transmural ischemia. *J Am Coll Cardiol* 2007;49:1299-305. [\[Crossref\]](#)

31. Charles MA, Bensing TA, Glasser SP. Atrial injury current in pericarditis. *Arch Intern Med* 1973;131:657-62. [\[Crossref\]](#)
32. Macfarlane PW, Antzelevitch C, Haissaguerre M, Huikuri HV, Potse M, Rosso R, et al. The early repolarization pattern: a consensus paper. *J Am Coll Cardiol* 2015;66:470-7. [\[Crossref\]](#)
33. Smith SW, Khalil A, Henry TD, Rosas M, Chang RJ, Heller K, et al. Electrocardiographic differentiation of early repolarization from subtle anterior ST-segment elevation myocardial infarction. *Ann Emerg Med* 2012;60:45-56. [\[Crossref\]](#)
34. Dec GW Jr, Waldman H, Southern J, Fallon JT, Hutter AM Jr, Palacios I. Viral myocarditis mimicking acute myocardial infarction. *J Am Coll Cardiol* 1992;20:85-9. [\[Crossref\]](#)
35. Abdulla I, Ward MR. Tako-tsubo cardiomyopathy: how stress can mimic acute coronary occlusion. *Med J Aust* 2007;187:357-60. [\[Crossref\]](#)
36. Kaur P, Bendaram SR, Singh B, Elkattawy S, Sah A, Noori M, et al. A STEMI mimic (Brugada ECG pattern) unveiled by influenza fever. *J Community Hosp Intern Med Perspect* 2020;10:488-90. [\[Crossref\]](#)
37. Lu YW, Tsai YL, Chang CC, Huang PH. A potential diagnostic pitfall in acute chest pain: massive pulmonary embolism mimicking acute STEMI. *Am J Emerg Med* 2018;36:461-3. [\[Crossref\]](#)
38. Akgun T, Gulsen K, Cinier G, Pay L, Uslu A, Kup A, et al. Electrocardiographic characteristics of metastatic cardiac tumors presenting with ST-segment elevation. *J Electrocardiol* 2020;59:93-9. [\[Crossref\]](#)
39. Gue YX, Corballis N, Ryding A, Kaski JC, Gorog DA. MINOCA presenting with STEMI: incidence, aetiology and outcome in a contemporaneous cohort. *J Thromb Thrombolysis* 2019;48:533-8. [\[Crossref\]](#)
40. Birnbaum Y, Bayés de Luna A, Fiol M, Nikus K, Macfarlane P, Gorgels A, et al. Common pitfalls in the interpretation of electrocardiograms from patients with acute coronary syndromes with narrow QRS: a consensus report. *J Electrocardiol* 2012;45:463-75. [\[Crossref\]](#)
41. Hochman JS, Lamas GA, Buller CE, Dzavik V, Reynolds HR, Abramsky SJ, et al. Coronary intervention for persistent occlusion after myocardial infarction. *N Engl J Med* 2006;355:2395-407. [\[Crossref\]](#)
42. de Zwaan C, Bär FW, Wellens HJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. *Am Heart J* 1982;103:730-6. [\[Crossref\]](#)
43. Ohman EM, Califf RM, Topol EJ, Abbottsmith C, Ellis S, Sigmon KN, et al. Consequences of reocclusion after successful reperfusion therapy in acute myocardial infarction. TAMI Study Group. *Circulation* 1990;82:781-91. [\[Crossref\]](#)
44. Aslanger E, Yıldırım Türk Ö, Şimşek B, Sungur A, Türer Cabbar A, Bozbeyoğlu E, et al. A new electrocardiographic pattern indicating inferior myocardial infarction. *J Electrocardiol* 2020;61:41-6. [\[Crossref\]](#)
45. Durant E, Singh A. Acute first diagonal artery occlusion: a characteristic pattern of ST elevation in noncontiguous leads. *Am J Emerg Med* 2015;33:1326.e3-5. [\[Crossref\]](#)
46. Smith SW. Upwardly concave ST segment morphology is common in acute left anterior descending coronary occlusion. *J Emerg Med* 2006;3:69-77. [\[Crossref\]](#)
47. Driver BE, Khalil A, Henry T, Kazmi F, Adil A, Smith SW. A new 4-variable formula to differentiate normal variant ST segment elevation in V2-V4 (early repolarization) from subtle left anterior descending coronary occlusion-Adding QRS amplitude of V2 improves the model. *J Electrocardiol* 2017;50:561-9. [\[Crossref\]](#)
48. Bozbeyoğlu E, Aslanger E, Yıldırım Türk Ö, Şimşek B, Karabay CY, Şimşek MA, et al. A tale of two formulas: Differentiation of subtle anterior MI from benign ST segment elevation. *Ann Noninvasive Electrocardiol* 2018;23:e12568. [\[Crossref\]](#)
49. Aslanger E, Yıldırım Türk Ö, Bozbeyoğlu E, Şimşek B, Karabay CY, Türer Cabbar A, et al. A simplified formula discriminating subtle anterior wall myocardial infarction from normal variant ST-segment elevation. *Am J Cardiol* 2018;122:1303-9. [\[Crossref\]](#)
50. de Winter RJ, Verouden NJ, Wellens HJ, Wilde AA; Interventional Cardiology Group of the Academic Medical Center. A new ECG sign of proximal LAD occlusion. *N Engl J Med* 2008;359:2071-3. [\[Crossref\]](#)
51. Zorzi A, Perazzolo Marra M, Migliore F, Tarantini G, Iliceto S, Corrado D. Interpretation of acute myocardial infarction with persistent 'hyperacute T waves' by cardiac magnetic resonance. *Eur Heart J Acute Cardiovasc Care* 2012;1:344-8. [\[Crossref\]](#)
52. Birnbaum Y, Fiol M, Nikus K, Niebla JG, Bacharova L, Dubner S, et al. A counterpoint paper: comments on the electrocardiographic part of the 2018 Fourth Universal Definition of Myocardial Infarction endorsed by the International Society of Electrocardiology and the International Society for Holter and Noninvasive Electrocardiology. *Ann Noninvasive Electrocardiol* 2020;25:e12786. [\[Crossref\]](#)
53. Kosuge M, Ebina T, Hibi K, Morita S, Endo M, Maejima N, et al. An early and simple predictor of severe left main and/or three-vessel disease in patients with non-ST-segment elevation acute coronary syndrome. *Am J Cardiol* 2011;107:495-500. [\[Crossref\]](#)
54. Aslanger E. Maybe a dazzle but not puzzle. *J Electrocardiol* 2010;43:682-4. [\[Crossref\]](#)
55. Schmitt C, Lehmann G, Schmieder S, Karch M, Neumann FJ, Schömig A. Diagnosis of acute myocardial infarction in angiographically documented occluded infarct vessel: limitations of ST-segment elevation in standard and extended ECG leads. *Chest* 2001;120:1540-6. [\[Crossref\]](#)
56. Meyers HP, Bracey A, Smith SW. ST depression maximal in V2-V4 is specific for Occlusion Myocardial Infarction [Abstract]. *Acad Emerg Med* 2021 (In Press).
57. Smith SW, Rapin J, Li J, Fleureau Y, Fennell W, Walsh BM, et al. A deep neural network for 12-lead electrocardiogram interpretation outperforms a conventional algorithm, and its physician overread, in the diagnosis of atrial fibrillation. *Int J Cardiol Heart Vasc* 2019;25:100423. [\[Crossref\]](#)

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