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High-risk electrocardiogram presentations in the acute coronary syndrome patient – Beyond ST-segment elevation myocardial infarction

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Abstract:

This review considers high-risk electrocardiographic patterns in the acute coronary syndrome (ACS) patient; we review 7 electrocardiogram presentations lacking diagnostic criteria for ST-segment elevation myocardial infarction (STEMI) yet likely representing either STEMI equivalent syndromes or ACS presentations with significant short-and long-term risk. The STEMI equivalent presentations include acute posterior wall myocardial infarction, the hyperacute T-wave of early STEMI, de Winter syndrome, first diagonal of the left anterior descending artery occlusion, and left bundle branch block modified Sgarbossa positive findings. High-risk presentation, not felt to be STEMI equivalent entities yet still possessing significant risk of short-and long-term adverse outcome, include lead aVR ST-segment elevation and Wellens syndrome. The features of each presentation, highlighting the electrocardiographic abnormalities, are presented and discussed.

Keywords:

Acute coronary syndrome, electrocardiography, high risk, myocardial infarction, ST-segment, T-wave

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Introduction

The approach to the adult patient with chest pain focuses on the recognition and treatment of life-threatening conditions, such as ST-segment elevation myocardial infarction (STEMI), other high-risk acute coronary syndrome (ACS) presentations, and a range of noncoronary ailments. Considering STEMI and other high-risk ACS presentations, the core emergency department evaluation includes a focused history and physical examination, serum markers, and the 12-lead electrocardiogram (ECG). The ECG plays a pivotal role in the early diagnosis and management of STEMI. In fact, the presence of anatomically oriented ST-segment elevation is required for the diagnosis of STEMI and also provides an indication for emergent reperfusion therapy, such as fibrinolysis or percutaneous coronary intervention (PCI).

Assuming the correct clinical presentation, including an uncomplicated electrocardiogram (ECG), management decisions are usually straightforward with fibrinolysis or PCI likely offered to the patient in a timely fashion for the patient with STEMI. An "uncomplicated electrocardiogram" indicates that the patient's ECG meets the following criteria, including newly noted ST-segment

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elevation, at the J point, in two anatomically contiguous leads, with the following features:

- In men, <40 years of age-ST-segment elevation ≥2.5 mm in leads V2 or V3 or ≥1 mm in any other lead;
- In men, ≥40 years of age-ST-segment elevation ≥2.0 mm in leads V2 or V3 or ≥1 mm in any other leads; and
- In women, regardless of age-ST-segment elevation ≥1.5 mm in leads V2 or V3 or ≥1.0 mm in any other leads.

These criteria were developed by the American College of Cardiology (ACC), the American Heart Association (AHA), the European Society of Cardiology (ESC), and the World Heart Federation (WHF).[1]

Other electrocardiographic presentations in the ACS patient are associated with similar cardiovascular risk yet do not present with ST-segment elevation meeting the ESC/ACC/AHA/WHF definition of STEMI.[1] These patterns are high risk, with the potential for adverse outcomes with short-term complications as well as more frequent death; they include patterns which are either STEMI equivalent presentations or electrocardiographic entities considered a manifestation of acute myocardial ischemia.[2] In certain instances, these ECG presentations may warrant urgent reperfusion therapy using fibrinolysis or PCI, considering resource availability; in other situations, "aggressive" and rapidly delivered adjunctive therapy along with cardiology involvement is likely needed. STEMI equivalent patterns represent ECG presentations which likely represent acute coronary occlusion with associated myocardial death and adverse outcome, yet lack the traditional electrocardiographic features of a STEMI; these patterns, as defined by the ACC, include the following: acute posterior myocardial infarction, hyperacute T-wave, de Winter syndrome, and left bundle branch block and right ventricular paced rhythm ECGs with abnormalities defined by the modified Sgarbossa criteria.[2-4] Another STEMI equivalent presentation, not mentioned in the ACC Pathway, includes acute occlusion of the first diagonal branch of the left anterior descending (LAD) artery which presents with "nonanatomic" ST-segment elevation in leads aVL and V2. Additional high-risk ECG presentations include those likely manifesting acute myocardial ischemia with associated short-term risk of STEMI with attendant increased rates of cardiovascular complications and death, such as lead aVR ST-segment elevation and Wellen's syndrome, among others.[2]

Acute Posterior Wall Myocardial Infarction

Acute posterior wall myocardial infarction (APMI) refers to an acute myocardial infarction (AMI) of the posterior wall of the left ventricle, not unlike STEMI of the inferior or lateral walls. The majority of APMI cases occur in the setting of inferior or lateral STEMI.[5,6] While less common, the so-called "isolated" APMI does occur; this AMI presentation refers to an APMI occurring without concurrent inferior or lateral STEMI.[5] Importantly, the isolated APMI represents a STEMI equivalent, as defined by the ACC in their Expert Consensus Pathway; thus, the recognition of the electrocardiographic presentation of isolated APMI is necessary in that it potentially allows for urgent management, including reperfusion therapy.[2] Unfortunately, clinicians not infrequently fail to recognize the electrocardiographic features of isolated APMI and thus manage the patient as if a larger AMI is not occurring; this failure to recognize APMI results in longer times to urgent reperfusion therapy as compared to patients with inferior, lateral, or anterior STEMI[7,8] and related adverse outcomes.

The posterior wall of the left ventricle is not directly imaged by the 12-lead electrocardiogram, making the diagnosis more challenging. Indirect imaging of the posterior wall is performed through standard leads V1 to V4 from the 12-lead ECG. The electrocardiographic criteria for APMI focus on ST segment depression in leads V1 to V4 along with the following ECG features [Figure 1]:

- Prominent R-wave in lead V1
- R/S-wave ratio >1 in lead V2

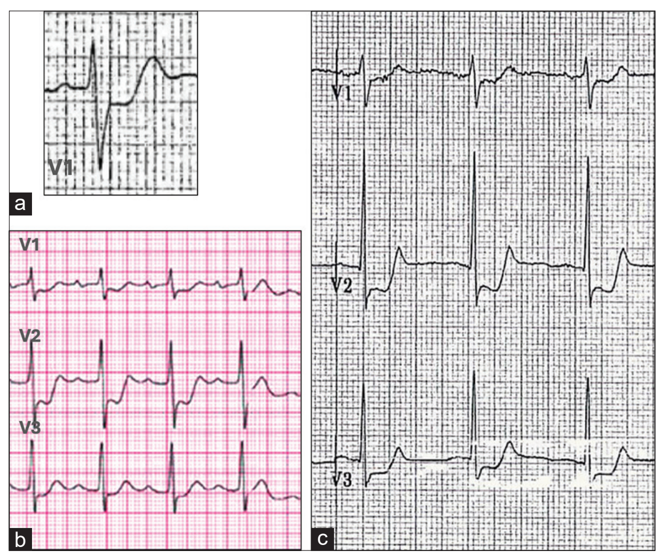

Figure 1: Acute posterior wall myocardial infarction (APMI). Recall that the posterior wall of the left ventricular is indirectly imagined by the 12-lead electrocardiogram, using leads V1 to V4. When viewing the posterior wall from this perspective, the abnormalities consistent with APMI are reversed. (a) Large R-wave, flat (horizontal) ST segment depression, and upright T-wave, findings very suggestive of APMI. (b) Leads V1 to V3, indirectly imaging the posterior wall of the left ventricle with Large R-wave, flat (horizontal) ST segment depression, and upright T-wave. (c) Leads V1 to V3, indirectly imaging the posterior wall of the left ventricle with Large R-wave, flat (horizontal) ST-segment depression, and upright T-wave– when all three findings are present, the likelihood of APMI is extremely high

- Upright (not inverted) T-waves.

If one considers all patients with suspected ACS who demonstrate ST-segment depression in leads V1 to V3 and/or V4, approximately half of these patients will be diagnosed with anterior wall ischemia while the remaining half will be found to have an APMI. To increase the rate of accurately diagnosing APMI among these patients with precordial lead ST-segment depression, the grouping of these three findings significantly increases the likelihood of acute posterior myocardial infarction [Figure 1]:
- Horizontal or flat ST-segment depression
- Prominent positive QRS complex in leads V1 and/or V2
- Upright T-wave.

If these three findings are present, then the positive predictive value for APMI approaches 95%. If needed, additional electrocardiographic leads (i.e., beyond the 12-lead ECG) can be used to directly image the posterior wall of the left ventricle; these posterior leads are termed leads V7, V8, and V9; current diagnostic criteria are ST-segment elevation >0.05 mV in one or more posterior leads or >0.10 mV for younger men <40 years old.[7]

As noted, these patients with isolated APMI are potential candidates for urgent reperfusion therapy with either fibrinolysis or PCI. The clinician should consider APMI among those ACS patients with precordial ST-segment depression, particularly those individuals who demonstrate horizontal ST-segment depression, prominent positive QRS complexes in leads V1 and/or V2, and upright T-waves in leads V1 to V3 and/or V4.[2]

Hyperacute T-Wave

The T-wave, the electrocardiographic representation of the ventricular repolarization, can be the manifestation of a range of acute and chronic myocardial and systemic disease processes, with changes either in amplitude (prominent T-wave) or polarity (inverted T-wave); considering the prominent T-wave, the electrocardiographic differential diagnosis is extensive, ranging from acute issues such as STEMI and hyperkalemia to chronic conditions, including benign early repolarization, left ventricular hypertrophy, and left bundle branch block. The most concerning cause of the prominent T-wave is early STEMI; in this instance, the prominent T-wave is termed a hyperacute T-wave.

The earliest electrocardiographic manifestation of STEMI is the hyperacute T-wave. The hyperacute T-wave can appear as early as 5 min after coronary occlusion and initiation of the acute infarction; it is transient in nature and frequently evolves into typical ST-segment elevation of STEMI within 30 min. These T-waves [Figure 2] are very large, relative to the QRS complex; in addition, they are broad-based and asymmetric in structure with a more gradual upstroke and rapid return to the baseline. The J point is frequently elevated as well. Over relatively short periods of time, the J point continues to elevate and reaches a similar height as the T-wave, resulting in the tombstone-like ST-segment elevation of early STEMI. As the acute infarction continues to evolve, the ST segment will assume a more typical contour of STEMI. As noted in the ACC Pathway, the hyperacute T-wave is considered a STEMI equivalent presentation; with a clinical presentation suggestive of AMI, the patient should be considered for cardiology consultation with urgent reperfusion therapy.[2]

de Winter Syndrome

Another STEMI equivalent presentation is the de Winter syndrome.[9-11] This ECG presentation is an extremely high-risk entity, most often with progression to typical STEMI within minutes to hours of presentation. It is a constellation of ECG findings coupled with proximal LAD artery occlusion. The electrocardiographic features [Figure 3] are most often encountered in the anterior (leads V1 to V4) and/or anterolateral leads (leads V1 to V6) and are as follows:[9]
1. ST-segment depression at the J point with upsloping ST segments continuing into prominent, symmetric T-waves;
2. The absence of ST-segment elevation in the affected leads;
3. Frequent prominent J-point elevation producing ST-segment elevation of approximately 0.5–1.0 mm in lead aVR.

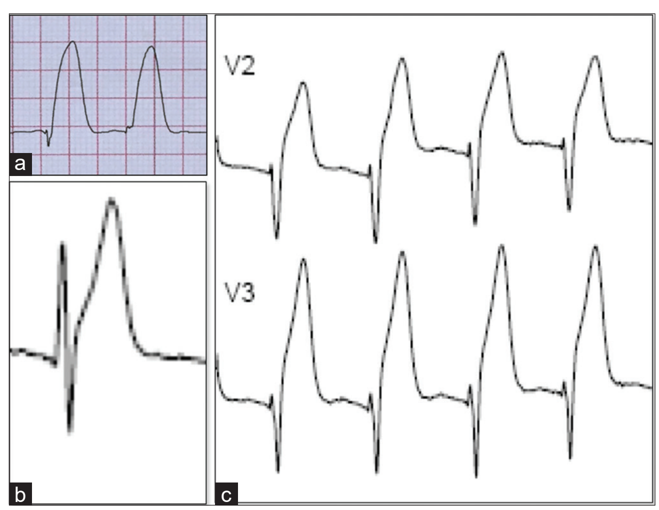

Figure 2: (a-c) Hyperacute T-wave of early STEMI. Note the prominent T-wave with a broad base, asymmetric morphology, and J-point elevation

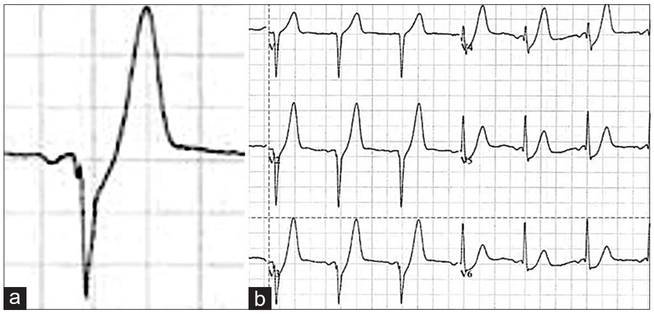

Figure 3: De Winter syndrome. (a) Lead V3 with prominent, hyperacute T-wave and J-point depression with accompanying T-segment depression. (b) Leads V1 to V4 with prominent, hyperacute T-wave and J-point depression with accompanying T-segment depression; similar ST-segment depression is also noted in leads V5 and V6

This ECG pattern is quite striking morphologically [Figure 3] – with the combination of ST segment depression and prominent T-wave – and is usually found in patients that are acutely ill with the typical presentation of STEMI… chest discomfort coupled with a pale, diaphoretic, anxious patient. With the culprit artery being the LAD artery, the ECG leads involved are most often leads V1 to V4, with occasional extension to leads V5 and V6. The natural history of de Winter syndrome is progression to a "typical" anterior or anterolateral STEMI, usually within minutes to hours of presentation. As with the hyperacute T-wave presentation of early STEMI, de Winter syndrome is considered a STEMI equivalent presentation; with a clinical presentation suggestive of AMI, the patient should be considered for cardiology consultation with urgent reperfusion therapy.[2]

Acute Occlusion of the First Diagonal of the Left Anterior Descending Artery

Occlusion of the first diagonal branch (D1) of the LAD coronary artery can result in STEMI with a somewhat unusual electrocardiographic presentation [Figure 4]; by "unusual," we are noting that the ST-segment elevation is located in a noncontiguous distribution, leads aVL and V2.[12] In some patients, the D1 branch of the LAD is a rather prominent artery, supplying perfusion to a significant portion of the anterior and/or lateral walls of the left ventricle. When obstructed and producing AMI, these patients will present with ST-segment elevation in a nontraditional, or noncontiguous, distribution, including elevation in leads aVL and V2, along with ST-segment depression in leads III, aVF, V4, and V5 [Figure 4]. It is important to recognize that this so-called nonanatomic distribution of ST-segment elevation represents an acute myocardial infarction with a large amount of myocardium in jeopardy. In the appropriate clinical setting, these patients should be considered for emergent reperfusion therapy.[12]

Left Bundle Branch Block (and Ventricular Paced Rhythm) with Modified Sgarbossa Criteria-related Abnormality

The electrocardiographic diagnosis of AMI in the setting of the left bundle branch block is challenging; in fact, the left bundle branch block (LBBB) pattern itself confounds the ECG's ability to detect and demonstrate acute myocardial infarction. Furthermore, LBBB is a marker of cardiovascular risk among patients with AMI; patients with acute myocardial infarction and LBBB, either newly occurring or previously present, have higher rates of cardiovascular complications and death than AMI patients without left bundle branch block. Finally, LBBB AMI patients benefit significantly from reperfusion therapy, likely more than the typical patient with AMI without LBBB. Thus, these patients with this challenging diagnosis are at higher risk of adverse outcomes and, if appropriately diagnosed and expeditiously managed, can benefit markedly from therapy.

The original Sgarbossa criteria gave the emergency clinician an ECG tool to assist in the diagnosis of AMI. The third criterion from the original rule, discordant ST-segment elevation >5.0 mm, demonstrated very weak test characteristics and was widely criticized.[13] Smith *et al*. modified the Sgarbossa criteria, addressing this limitation in the original clinical decision rule. The resultant clinical decision rule, termed the modified Sgarbossa criteria, improved the sensitivity for the ECG diagnosis of AMI from 52% with the original rule to 91% in identifying angiographically proven STEMI; positive and negative likelihood ratios for the revised rule were 9.0 (95% confidence interval [CI] 8.0–10) and 0.1 (95% CI 0.03–0.3), respectively.[3,4]

The modified Sgarbossa criteria are useful in the ECG evaluation of potential AMI in patient with LBBB. Using the modified Sgarbossa criteria requires an understanding of the anticipated ST-segment and T-wave configurations of uncomplicated LBBB. In general, the major terminal portion of the QRS complex is located on the opposite side (i.e. discordant) of the isoelectric baseline from the ST-segment and T-wave; this relationship is termed the concept of appropriate discordance [Figure 5a]. Thus, with a primarily negatively oriented QRS complex, ST-segment elevation with an upright T-wave is anticipated; this ST-segment elevation is termed discordant ST-segment elevation. Conversely, when a primarily positively oriented QRS complex, ST-segment depression with T-wave inversion is anticipated; this ST-segment depression is termed discordant ST-segment depression. Importantly, the amplitude of the QRS complex is proportional to the magnitude of the ST-segment deviation.

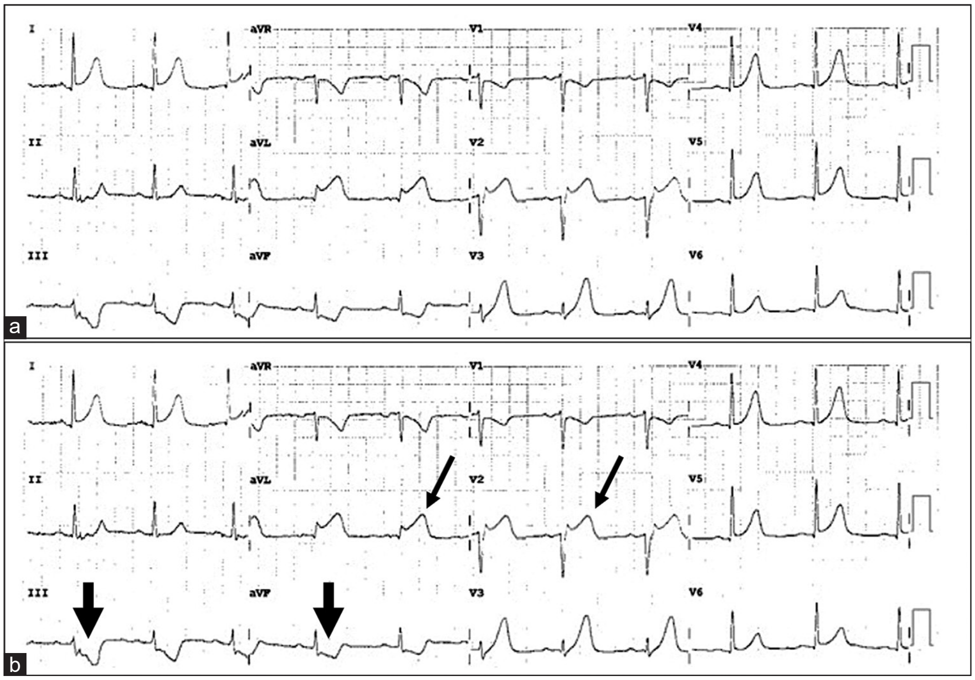

Figure 4: First diagonal branch of left anterior descending artery acute occlusion. (a) ST-segment elevation in leads aVL and V2 along with ST-segment depression in the inferior leads. (b) Annotated electrocardiogram from "A" with ST-segment elevation in leads aVL and V2 (small arrows) along with ST-segment depression in the inferior leads (large arrows)

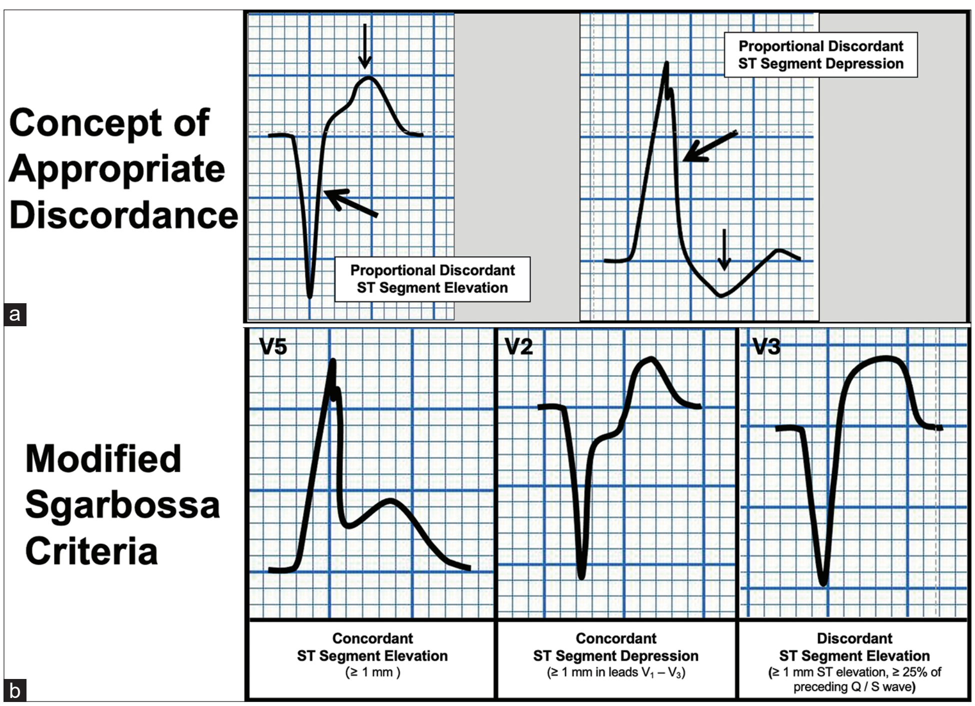

Figure 5: LBBB with anticipated and abnormal ST-Segment-T-wave Configurations. (a) Concept of Appropriate Discordance– note that the terminal portion of the QRS complex and ST-segment are located on opposite sides of the isoelectric baseline; this relationship is the anticipated electrocardiogram (ECG) appearance of an uncomplicated LBBB. (b) Modified Sgarbossa Criteria for AMI diagnosis in LBBB– 3 ECG findings which are strongly suggestive of AMI in the setting of LBBB and appropriate clinical presentation, including concordant ST-segment elevation, concordant ST-segment depression, and excessive (disproportionate) discordant ST-segment elevation

The modified Sgarbossa criteria [Figure 5b] include the three findings, all of which are suggestive of AMI from the electrocardiographic perspective; note that the appropriate clinical presentation consistent with AMI is necessary. The modified Sgarbossa criteria include the following three ECG abnormalities:[3]

- ST-segment elevation of at least 1.0 mm that is concordant with the QRS complex (concordant ST-segment elevation);
- ST-segment depression of at least 1.0 mm in leads V1, V2, or V3 (concordant ST-segment depression); and/or
- Excessive discordant ST-segment elevation, defined with an ST-segment to S-wave ratio of ≥0.25.

Any of these criteria, if found, suggests the ECG presence of AMI; reperfusion therapy is likely indicated in such patients. Furthermore, these findings need only be present in a single ECG lead to support the diagnosis of AMI.[3]

It is important to note that a significant portion of AMI patients with LBBB will not demonstrate ECG abnormalities as described in the modified Sgarbossa criteria; thus, in a patient with a concerning clinical picture yet who lack positive Sgarbossa criteria, these individuals can still be experiencing an AMI. Management decisions must be made with this limitation in mind.

Right ventricular paced rhythms similarly confound the ECG's ability to electrocardiographically detect AMI. While such patients have an array of cardiovascular comorbid issues, they are generally not as chronically ill as patients with LBBB. The concept of appropriate discordance, as discussed with LBBB, applies in the case of right ventricular paced rhythm with respect to recognizing the anticipated ST-segment and T-wave configurations of the ventricular paced rhythm. Furthermore, the modified Sgarbossa criteria as applied to LBBB were found to be similarly useful in the electrocardiographic diagnosis of AMI in the setting of right ventricular paced rhythms.[4] As with LBBB application of the modified Sgarbossa criteria, patients with right ventricular paced rhythms can still be experiencing AMI, despite the absence of abnormality noted through the modified Sgarbossa criteria; management decisions must be made with consideration of this caveat.[4]

Lead aVR ST-Segment Elevation

STEMI resulting from left main coronary artery (LMCA) near-complete obstruction is associated with a very high mortality rate and is most appropriately managed with reperfusion strategies other than fibrinolysis, namely, PCI or emergent coronary artery bypass grafting. Thus, the ability to identify left main STEMI presentations early in ED care is important. One such electrocardiographic pattern involves ST-segment elevation in lead aVR >0.5 mm [Figure 6] and is associated with LMCA near-complete obstruction; in some patients, additional ST-segment elevation can be seen in lead aVL [Figure 6].

Yamaji has noted that ST-segment elevation in lead aVR >0.5 mm has a modest sensitivity and specificity for predicting LMCA obstruction with values of 81% and 80%, respectively.[14] Rostoff determined that lead aVR ST-segment elevation was twice as frequent in the patient population with LMCA involvement with approximately 70% involvement as opposed to those with involvement of a different vessel (35%).[15] Kosuge found modest test characteristics supporting lead aVR ST-segment elevation as a predictor of LMCA as the culprit vessel in ACS presentations with a sensitivity of 78%, a specificity of 86%, a positive predictive value of 57%, and a negative predictive value of 95%.[16]

Left main coronary obstructive issues, in other words, when the culprit vessel is the LMCA in ACS presentations, is a marker of significant risk. For example, Barrabes examined the prognostic significance of this finding by studying the initial ECG in 775 consecutive patients admitted with their first non-AMI ACS event. In the ACS patient with LMCA obstruction, Barrabes found that inhospital mortality increased proportionately with increasing amounts of lead aVR ST-segment elevation. Mortality rates relative to the magnitude of lead aVR ST-segment elevation noted this increase in adverse outcomes with only 1% deaths-elevation <0.5 mm, 9% deaths - 0.5–1.0 mm, and 19% deaths - >1.0 mm. The odds ratio for death increased from 4.2 to 6.6 with minimal to maximal lead aVR ST-segment elevation; multivariate analysis noted that lead aVR ST-segment elevation was the only variable from the initial ECG that was strongly associated with inhospital MACE-related adverse events.[17] Further, Kosuge noted that ST-segment elevation >0.5 mm in lead aVR on the admission ECG and elevated troponin T levels were determined to be independent predictors of adverse MACE-related events at 90 days, with an odds ratio of 13.8 and 7.9, respectively.[18] Thus, the early and reliable identification of LMCA as the culprit vessel is important, not only to guide therapy but also to assess the short-term risk of adverse cardiovascular outcomes.

Thus, ST-segment elevation in lead aVR in the patient with clinically suspected ACS suggests a strong possibility of LMCA obstruction. This finding is important in that such obstruction is associated with a markedly higher mortality rate and may be more amenable to mechanical reperfusion strategies rather than fibrinolysis. Furthermore, such electrocardiographic finding is associated with a significant risk of short-term adverse events.

Importantly, lead aVR ST-segment elevation, of any magnitude, is not considered a STEMI equivalent.[2]

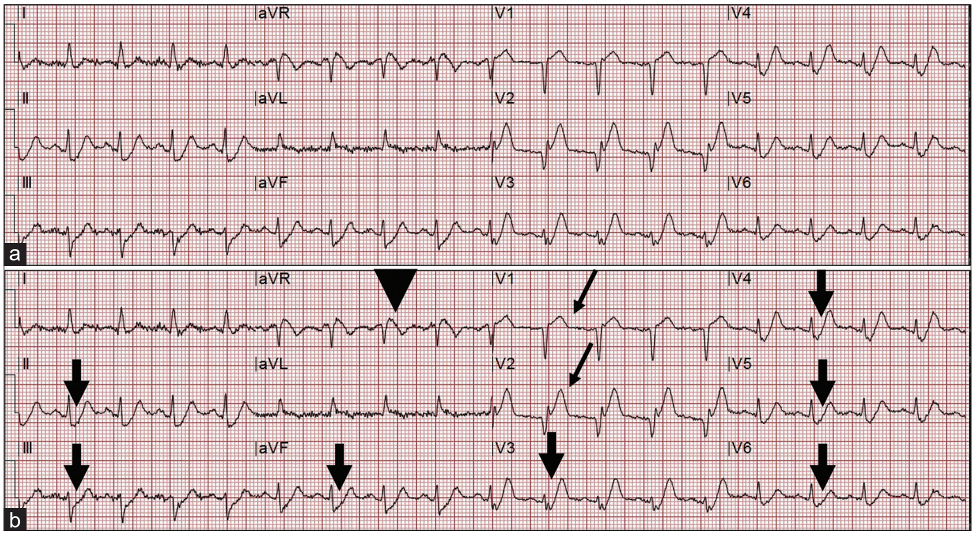

Figure 6: Acute partial occlusion of the left main coronary artery. (a) 12-lead electrocardiogram (ECG) with anterior wall STEMI with ST-segment elevation in leads V1 and V2. ST-segment elevation in lead aVR is suggestive of the left main coronary artery as the culprit vessel. In addition, widespread (i.e., >2 anatomic segments) with ST-segment depression. (b) Annotated ECG from "A" with anterior wall STEMI with ST-segment elevation in leads V1 and V2 (small arrows). ST-segment elevation in lead aVR (large arrowhead) is suggestive of the left main coronary artery as the culprit vessel. In addition, widespread (i.e., >2 anatomic segments) with ST-segment depression (large arrows)

Rather, this finding is felt to be a predictor of LMCA as the culprit vessel in the ACS presentation, a potential guide to early management of the patient, and a prognostic indicator. The ACS type is the primary indicator of the initial management strategy, with the following general treatment approaches suggested in the setting of co-existent lead aVR ST-segment elevation:

- STEMI– reperfusion therapy using mechanical means, if available in timely fashion [Figure 6];
- NSTEMI– anticoagulant and antithrombotic therapies coupled with early coronary angiography, if available; and
- Preinfarction angina (i.e., unstable angina)-anticoagulant and antithrombotic therapies coupled with early coronary angiography, if available
- Figure 6 depicts a patient with anterior wall STEMI resulting from LMCA near-complete occlusion. ST-segment elevation is seen in leads V1 and V2, indicating the anterior STEMI. Simultaneous ST-segment elevation in leads aVR and aVL indicates a high probability of the LMCA being the culprit artery. Finally, widespread ST-segment depression is noted in the inferior, anterior, and lateral regions and is another electrocardiographic indicator of LMCA being the culprit artery in an ACS presentation; in this case a STEMI; note that widespread in this context is defined as occurring in 2 or more anatomic regions.

Wellen's Syndrome

Wellen's syndrome was first described in patients hospitalized for ACS without established myocardial infarction who experienced adverse short-term outcomes, including anterior wall STEMI and/or sudden cardiac death. It is a high-risk coronary presentation with a constellation of electrocardiographic and clinical findings; Wellen's syndrome is associated with a proximal LAD coronary artery occlusion.[19,20] Electrocardiographically, these patients will demonstrate abnormal T-waves in the anterior leads. Clinically, these patients will present with chest discomfort, either active pain or recently resolved and now sensation-free. Despite being pain-free, the ECG abnormalities persist until ultimate management of the coronary occlusion occurs. Additional features of the syndrome include no biochemical (i. e., troponin elevation) nor electrocardiographic (i.e., ST-segment elevation or Q-waves) evidence of established myocardial infarction.

The electrocardiographic abnormalities[21] of Wellen's syndrome include characteristic T-wave findings [Figure 7] with occasional alterations of the ST-segment, usually encountered in leads V2 and V3; occasionally, the entire precordial lead array will demonstrate T-wave abnormality. The ST-segment itself is usually not elevated nor depressed; if abnormal, it is minimally elevated, usually < 1 mm with a high take-off of the ST-segment from the terminal portion of the QRS complex. If the ST-segment is elevated, it is either convex in contour or obliquely straight in appearance.[21] T-wave findings, which are the characteristic feature of this syndrome, will assume one of two morphologies, including the deeply inverted T-wave [75% of cases; Figure 7a] and the biphasic T-wave [25%; Figure 7b]. In the more common morphology, the T-wave is deeply inverted and symmetric in contour, meaning that the downstroke is a mirror image of the upstroke. The

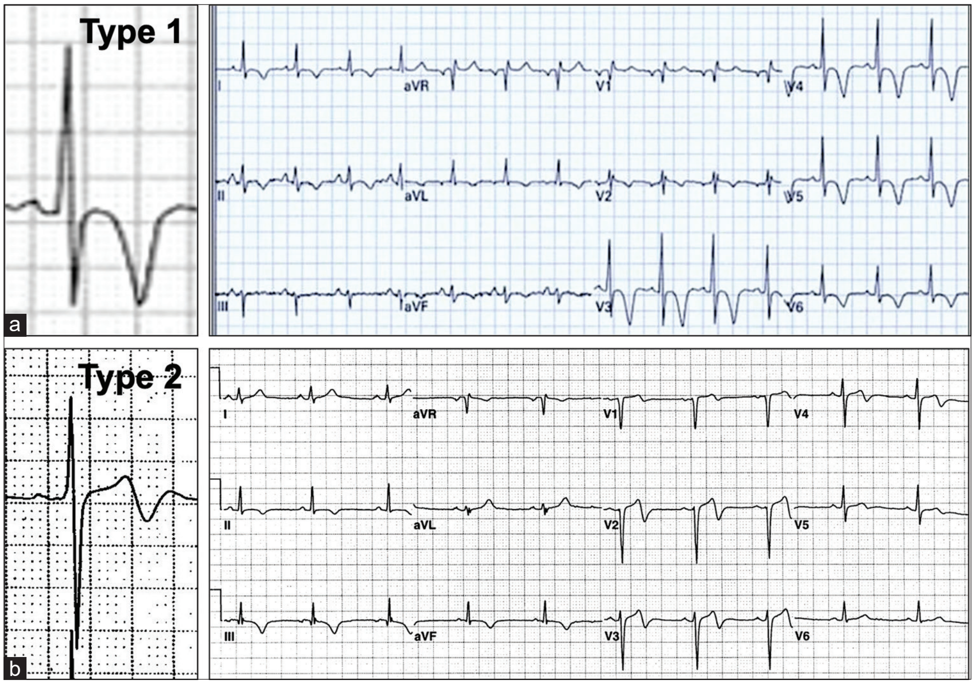

Figure 7: Wellens' syndrome. (a) Deeply inverted T-waves of Wellens' syndrome, encountered in 75% of cases. 12-lead electrocardiogram (ECG) with similar T-wave abnormalities in leads V3 to V5. (b) Biphasic T-waves of Wellens' Syndrome, seen in 25% of cases. 12-lead ECG with these T-wave abnormalities in leads V1 to V4

less common variant presents with biphasic T-waves; these T-waves have both positive and negative polarity components in a single T-wave.[19-21]

A summary of the syndrome's diagnostic features includes the following list:[19-21]

- symmetric and deeply inverted T-waves [Figure 7a] in leads V2 and V3, occasionally involving leads V1, V4, V5, and V6;

or

- biphasic T-wave [Figure 7b] in leads V2 and V3, again occasionally involving leads V1, V4, V5, and V6;

plus

- isoelectric or minimally elevated ST-segment (<1 mm);
- no precordial Q-waves;
- history of angina, current or painfree at presentation;
- ECG pattern present in pain-free state;
- normal or slightly elevated cardiac serum markers; and
- LAD artery occlusion.

Wellen's syndrome is not considered a STEMI equivalent; rather, it is a high-risk coronary presentation with an adverse short-term prognosis, related to the proximal LAD artery occlusion. Wellen's syndrome does not represent an indication for emergent reperfusion therapy, such as is true with STEMI and the STEMI equivalent presentations discussed in this article. Rather, these patients should be admitted to the hospital for coronary imaging, assuming such resources are available. Importantly, stress imaging should not be performed in these high-risk patients.[21]

Author contribution statement

All authors (Brady, Muck, and Moak) participated in the planning, writing, editing, and review of this manuscript. One author (Brady) developed the figures which were reviewed and edited as appropriate by the other authors (Moak and Muck).

Conflicts of interest

None Declared.

Ethical approval

Non applicable

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