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Electrocardiographic patterns of proximal left anterior descending artery occlusion in ST-elevation myocardial infarction may be modified by three-vessel coronary artery disease

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Abstract

Background—The electrocardiographic (ECG) pattern of ST-segment deviation in myocardial infarction is integral to the proper assessment of the location, extent, and functional significance of the infarct but may be modified by the underlying coronary artery anatomy.

Methods—We describe the electrocardiographic findings in two cases of proximal left anterior descending (LAD) artery occlusion in ST-elevation myocardial infarction (STEMI) associated with three-vessel coronary artery disease (CAD).

Results—Both patients had atypical ECG patterns of ST-segment elevation in leads V2, I, and aVL and ST-segment depression with positive T-waves suggestive of extensive subendocardial ischemia in leads II, III, aVF, and V3–V6; acute proximal LAD occlusion and concomitant three-vessel CAD was observed angiographically.

Conclusion—Electrocardiographic changes in proximal LAD STEMI may be modified by the presence of significant atherosclerotic disease elsewhere in the coronary vasculature. Recognition of this ECG pattern may aid the clinician in the rapid identification of high-risk STEMI.

Introduction

The electrocardiogram (ECG) is considered the most important initial clinical test for the diagnosis of ST-segment elevation myocardial infarction (STEMI). STEMI is defined electrocardiographically as an acute ST-segment elevation at the J-point in two contiguous leads (with varying cut-points for sex and lead type) in the setting of a clinical syndrome suggestive of myocardial ischemia (1). The changes in the ST-segment reflect currents of injury elicited by potential gradients between ischemic and non-ischemic myocardium, and

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the number of ECG leads demonstrating ST-segment changes correlates with the regional extent of the ischemia. The size and location of the affected region, in turn, depend on the coronary artery involved and the site of occlusion within the artery (2). Thus, the ECG pattern of ST-segment deviation is integral to the proper assessment of the location, extent, and functional significance of the infarct.

Multiple ECG patterns of ST-elevation have been reported and varying algorithms have been used to determine the site of occlusion within the culprit artery (3). However, the underlying coronary artery anatomy and presence of significant atherosclerotic disease elsewhere in the coronary vasculature may modify these electrocardiographic patterns. Here we describe variations in the ECG patterns of proximal left anterior descending (LAD) artery occlusion associated with three-vessel coronary artery disease (CAD) based on observations from two patients. Characteristic ECG changes observed include ST-segment elevation in leads V2, I, and aVL and ST-segment depression with positive T-waves suggestive of extensive subendocardial ischemia in leads II, III, aVF, and V3–V6.

Case history

Patient 1 is a 66 year old man with CAD who presented to the emergency department 45 minutes after the acute onset of severe, substernal chest pain that radiated to the back, was associated with light-headedness, dyspnea, and diaphoresis, and was not relieved with sublingual nitroglycerin. Examination revealed normal heart sounds with normal rate and regular rhythm, no murmurs or gallops, and no jugular venous distention. The initial ECG showed 2mm ST-elevation in V2 and aVL, 1mm ST-elevation in I, and ST-depressions in II, III, aVF, and V3–V6 (Figure 1). He was transported to the cardiac catheterization laboratory for emergent coronary angiography.

Patient 2 is a 68 year old woman with a history of multiple cardiovascular risk factors who experienced a prolonged episode of chest discomfort with onset at rest, radiating to the back, associated with dyspnea and diaphoresis. She was brought to a local hospital, arriving within 3 hours after symptom onset, where examination revealed normal heart sounds with normal rate and regular rhythm, no murmurs or gallops, and no jugular venous distention. The initial ECG showed mild ST-elevation in V1, 2mm ST-elevation in V2, 1mm ST-elevation in I and aVL, and ST-depressions in II, III, aVF, and V3–V6 (Figure 2). The patient was transported to the cardiac catheterization laboratory for emergent coronary angiography. Pertinent past medical history and clinical characteristics of both patients are presented in Table 1.

Catheterization findings

Patient 1 was found to have an acute thrombotic occlusion of the proximal LAD artery with TIMI 0 flow. He underwent successful percutaneous coronary intervention (PCI) with thrombus evacuation and placement of two drug-eluting stents to the proximal and mid-LAD with restoration of TIMI 3 flow (Figure 3). Coronary angiography also revealed 20% stenosis of the left main artery, 70% stenosis of the proximal left circumflex artery, and 70% stenosis of the proximal right coronary artery (RCA) with a small, patent conal branch, and no evidence of collateral circulation. A post-procedure echocardiogram revealed anterior and apical akinesis with an ejection fraction of 52%, which had changed significantly from his baseline LV ejection fraction of 69% with no regional wall motion abnormalities seen on echocardiogram eight months prior to presentation.

Patient 2 was found to have an acute sub-total occlusion of the proximal LAD artery with TIMI 1 flow. She underwent successful PCI with placement of two drug-eluting stents to the proximal and mid-LAD with restoration of TIMI 3 flow. Coronary angiography also revealed one diagonal branch arising from an apically terminating LAD with moderate ostial

and proximal disease, 70% stenosis of the proximal left circumflex artery and 90% ulcerated stenosis of the mid-RCA, without evidence of collateral circulation. Left ventriculography showed an estimated ejection fraction of 45% with moderate hypokinesis of the anterolateral wall and apex. No complications occurred during either catheterization.

Discussion

Here we describe how the electrocardiographic patterns of proximal LAD occlusion in STEMI may be modified by significant three-vessel CAD. Characteristic ECG changes included ST-segment elevation in leads V2, I, and aVL and ST-segment depression with positive T-waves suggestive of extensive subendocardial ischemia in leads II, III, aVF, and V3–V6. Our study confirms prior observations of atypical ST-segment elevation patterns in proximal LAD STEMI and contributes new and important insights into the modification of these patterns by concomitant three-vessel CAD, including alterations of the terminal T-wave vector in the inferior and lateral pre-cordial leads and recognition that this constellation of findings may represent a high-risk subset of STEMI patients with a significant atherosclerotic disease burden.

Proximal LAD-related myocardial infarction is associated with higher mortality compared with distal LAD or non-LAD-related infarcts (4) so prompt electrocardiographic recognition is critical to timely intervention. After occlusion of the LAD, ST-segment elevation 1 mm is most frequently observed in lead V2, with a sensitivity of 91% to 99% and specificity up to 100% (5–6). Other predictors of proximal LAD occlusion include ST-elevation in aVL with ST-depression in the inferior leads (7) and ST-elevation in lead aVR in association with concomitant ST-elevation in the pre-cordial leads (8). Proximal LAD occlusion leading to STEMI usually presents in two contiguous leads and frequently involves V3 or V4 in addition to V2, but the pattern may differ depending on the location of the lesion. If the occlusion occurs at the level of the first diagonal, a characteristic ECG pattern of ST-elevation with positive T-wave in nonconsecutive leads of aVL and V2 can be seen, associated with two different types of ST-depression: ST-depression with negative T-wave in III and aVF signifying true reciprocal changes and ST-depression with positive T-wave in V4–5 signifying subendocardial ischemia (9). If the lesion is more proximal, ST-elevation may be seen in V1 depending on the anatomic blood supply to the septum (10–11). We observed a unique combination of these ECG changes reflecting proximal LAD occlusion (ST-elevation in V2 and aVL) with involvement of the downstream 1st diagonal artery (ST-elevation in I and aVL and ST-depression with positive T-wave in V3–V6). However, in contrast to the report by Sclarovsky et al (9), our patients had ST-depressions in the inferior leads that were associated with a positive T-wave, not a negative T-wave. Given that both patients had significant disease in the RCA, this finding may be due to simultaneous subendocardial ischemia of the inferior wall due to RCA stenosis.

For patients in whom the infarct-related LAD artery extends beyond the apex to supply the inferior left ventricular wall, ST-segment morphology in the inferior and lateral ECG leads may be influenced. In a study by Sasaki et al (12), ST-elevation in leads I and aVL and reciprocal ST-depression in the inferior leads were associated with a short, apically-terminating LAD artery with proximal occlusion. In contrast, proximal LAD occlusion in patients with an LAD supplying the LV inferior wall were less likely to have inferior ST-segment deviation, with the reciprocal ST-segment depressions countered by ST-segment elevation attributable to epicardial injury of the inferior LV wall. Similarly, Huang et al (13) reported that an “anteroseptal” STEMI pattern of ST-elevation in leads V1–V3 may occur in patients with a proximal LAD occlusion with either a short LAD or at least one large side branch supplying the apex resulting in concomitant ST-depression in leads V5 and V6 (due to an oppositely directed injury vector), or in patients with a LAD extending beyond the

apex with balanced anterobasal, septal, and apical ischemia attenuating ST-elevation in leads V4–V6. Both of our patients had proximal total or sub-total occlusion of apically-terminating LAD arteries, consistent with the ST-depression observed in both the inferior and lateral pre-cordial leads of the ECG. However, given that both patients had apical wall motion abnormalities on imaging, an alternative explanation for these findings may be that significant stenoses in the circumflex and/or right coronary arteries contributed to apical subendocardial ischemia resulting in lateral pre-cordial ST-depressions with positive T-waves.

Three-vessel coronary artery disease portends a poor prognosis and management often involves surgical revascularization. In STEMI, it is difficult to predict on the basis of ECG which patients will have three-vessel disease at angiography. Data from the FRISC-II trial and others have demonstrated that widespread ST-segment depression on ECG is associated with a significant increase in the prevalence of three-vessel disease in non-ST-elevation acute coronary syndromes (14–15). Our two case patients had extensive ST-depression on ECG, consistent with this finding. However, we are unable to fully distinguish ST-depression as a result of extensive ischemia in three-vessel disease from reciprocal changes that present early after the onset of myocardial infarction in a subset of high-risk patients (16).

Limitations

Several limitations of our report must be acknowledged. First, the ECGs and the distribution of coronary artery disease in the two case patients are not identical. There appears to be mild ST-segment elevation in lead V1 in Patient 2's ECG that is consistent with proximal LAD occlusion; this finding is absent in Patient 1 and may be due to protection of the right paraseptal area from ischemia by the observed conal branch of the RCA supplying the right side of the ventricular septum (11). Additionally, in Patient 2, the LAD lesion is sub-totally occluded; therefore, an alternative explanation may be that her ECG pattern represents "mid-anterior" ischemia due to first diagonal branch occlusion and less ischemia due to the sub-total proximal LAD occlusion in the setting of a short LAD, or possibly ischemic pre-conditioning due to concomitant three-vessel disease. Indeed, Birnbaum et al. (17) found that ST-elevation in leads aVL and V2 accompanied by either isoelectric or depressed ST-segments in leads V3–V5 strongly favored occlusion of the first diagonal branch over the proximal LAD. However, ST-elevation in lead V1 is more consistent with LAD and not diagonal occlusion, which conflicts with this alternative hypothesis.

Conclusion

We describe how the electrocardiographic patterns of proximal LAD occlusion in STEMI may be modified by three-vessel CAD. Recognition of this pattern by ECG may aid the clinician in the rapid identification of high-risk STEMI and urgent referral for appropriate revascularization.

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Figure 1. Patient 1 ECG
ECG of Patient 1 on presentation to the Emergency Department
ECG=Electrocardiogram

Figure 2. Patient 2 ECG
ECG of Patient 2 on presentation to the Emergency Department
ECG=Electrocardiogram

Figure 3. Patient 1 Pre- and Post-Intervention Coronary Angiograms

Patient 1 Coronary Angiograms; Panel A: Pre-Intervention, RAO Cranial view, complete occlusion of the proximal LAD artery (arrow); Panel B: Post-Intervention, RAO Cranial view, PCI with restoration of flow (arrow)

RAO= Right Anterior Oblique; LAD=Left Anterior Descending; PCI=Percutaneous Coronary Intervention

Table 1

Clinical characteristics of the two case patients

Variable	Patient 1	Patient 2
Age (years)	66	68
Sex	Male	Female
CAD Risk Factors		
Hypertension		
Diabetes		
Hyperlipidemia		
Family History of CAD		
Smoker		
Known CAD		
Prior PCI		
Presentation	6 hours	
Killip Class (I–IV)	I	I
Blood Pressure (mmHg)	121/77	157/95
Heart Rate (bpm)	73	75
Peak cardiac markers		
CK-MB (ng/mL)	188.9	n/a
Troponin I (ng/mL)	31.4	13
Ejection Fraction (%)	52	45
Infarct-related artery	Proximal LAD	Proximal LAD
Intervention	DES × 2	DES × 2
Three vessel disease		

= present

Abbreviations: CAD, coronary artery disease; PCI, percutaneous coronary intervention; CK-MB, creatine kinase-myocardial band; n/a, not available; LAD, left anterior descending artery; DES, drug-eluting stent