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Electrocardiographic Recognition of Unprotected Left Main ST-Segment Elevation Myocardial Infarction

Looking Beyond aVR

Diego H. González-Bravo, MD, José Escabí-Mendoza, MD

ABSTRACT

ST-segment elevation in aVR has traditionally been used for electrocardiographic identification of left main coronary artery (LM) myocardial infarction. We present two ST-segment elevation myocardial infarction (STEMI) cases with acute total occlusion of the LM without aVR ST-segment elevation. This report reviews the different electrocardiographic discriminators suggestive of unprotected LM STEMI. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:754–9) Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

INTRODUCTION

Unprotected left main coronary artery (LM) ST-segment elevation myocardial infarction (STEMI) is certainly the most lethal type of acute myocardial infarction (60% to 90% mortality) often resulting in cardiogenic shock or sudden cardiac death (1,2). Our aim is to raise awareness and review the predominant electrocardiographic discriminators suggestive of LM STEMI by presenting two cases that defied the classically known marker for this disease: aVR ST-segment elevation (STE).

LEARNING OBJECTIVES

- To recognize and differentiate electrocardiographically a LM from a proximal-LAD occlusion in patients presenting with anterior or anterolateral STEMI.
- To demonstrate that acute total occlusion of an unprotected LM with a TIMI flow grade of 0 may present without aVR ST-segment elevation and therefore electrocardiographic analysis should look beyond this lead for diagnosis.

CASE 1

A 69-year-old man with type-2 diabetes mellitus and dyslipidemia and who is a former smoker presented to our emergency department with oppressive chest pain, dyspnea, and diaphoresis of 2-h evolution. Physical examination revealed tachypnea (22 breaths/min), hypotension (86/62 mm Hg), hypoxemia (89%), a regular heart rate (76 beats/min), no jugular venous distension, and no heart murmurs. He also presented with bibasilar rales, cold extremities, and weak arterial pulses; all of which are consistent with heart failure and early hemodynamic compromise.

His arrival electrocardiogram revealed left axis deviation (LADEV) and left anterior fascicular block (LAFB), plus STEs in leads V_2 to V_6, I and aVL, with

From the Cardiovascular Division, Department of Medicine, Veterans Affairs Caribbean Healthcare System, San Juan, Puerto Rico. The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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reciprocal inferior ST-segment depressions (STDs), which are consistent with anterolateral STEMI (**Figure 1**).

Immediate coronary angiography revealed an acute totally (100%) occluded (ATO) LM with TIMI (Thrombolysis In Myocardial Infarction) flow grade 0 without collaterals (**Figures 2A to 2C**, Videos 1 and 2). Intra-aortic balloon pump (IABP) was placed for hemodynamic support followed by successful percutaneous coronary intervention (PCI) to the LM with a drug-eluting stent achieving restoration of TIMI flow grade 3, and resolution of angina and STEs (**Figures 2D to 2F**, Video 3). He was discharged on day 5 post-STEMI with moderate systolic dysfunction of 35% to 40% and on guideline-directed medical therapy. Three months post-revascularization, his systolic function improved and became 50% to 55%.

CASE 2

A 67-year-old man with liver transplant (in 2012) and type-2 diabetes mellitus who arrived at the emergency department in Trendelenburg position after 4.5 h of severe oppressive chest pain. Paramedics found him hypotensive (60/40 mm Hg) and hypoxic (88%) at his home. His physical exam was worrisome for cardiogenic shock with the presence of jugular venous distension, bilateral rales, clammy extremities, and soft arterial pulses.

Initial electrocardiogram showed a new right bundle branch block (RBBB), LADEV, LAFB, plus STEs in V_2 to V_4, I, and aVL, with reciprocal STDs in V_5, V_6, and the inferior leads, compelling for anterolateral STEMI (**Figure 1**).

Emergency heart catheterization (**Figures 2G to 2L**, Videos 4 and 5) disclosed ATO with TIMI flow grade 0 of the LM with absent distal collateral filling, 80% stenosis of the right coronary artery, and severe systolic dysfunction (20%). After IABP placement, PCI with drug-eluting stent using simultaneous kissing stents was performed to reopen the LM, the proximal left anterior descending coronary artery (LAD), and the proximal circumflex coronary artery (**Figures 2I and 2J**, Video 6). Unfortunately,

ABBREVIATIONS AND ACRONYMS

ATO = acute total occlusion

IABP = intra-aortic balloon pump

LAD = left anterior descending coronary artery

LADEV = left axis deviation

LAFB = left anterior fascicular block

LM = left main coronary artery

NPV = negative predictive value

PCI = percutaneous coronary intervention

PPV = positive predictive value

RBBB = right bundle branch block

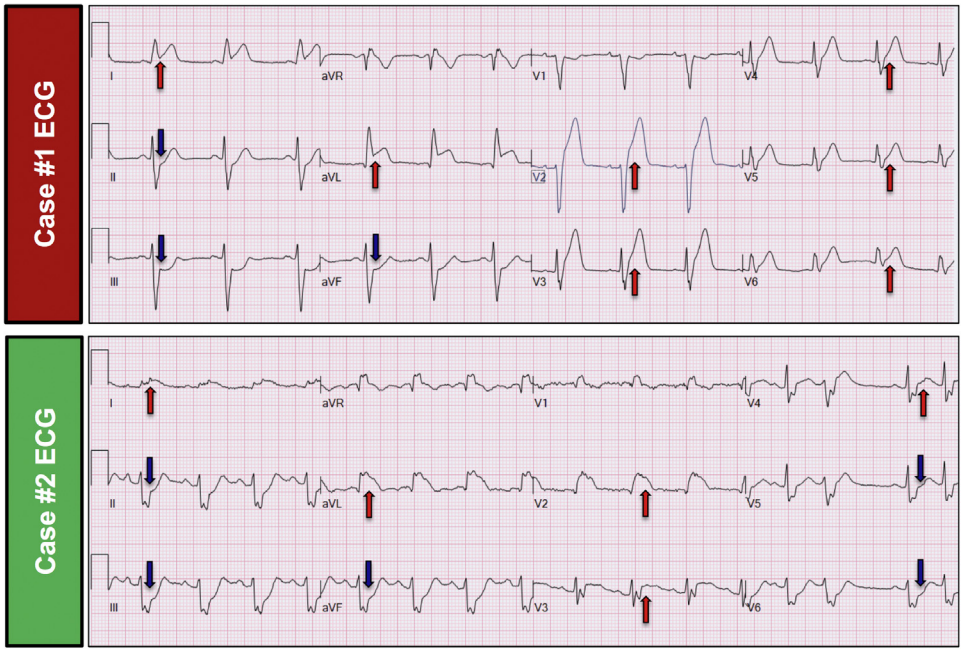
STD = ST-segment depression

STE = ST-segment elevation

STEMI = ST-segment elevation myocardial infarction

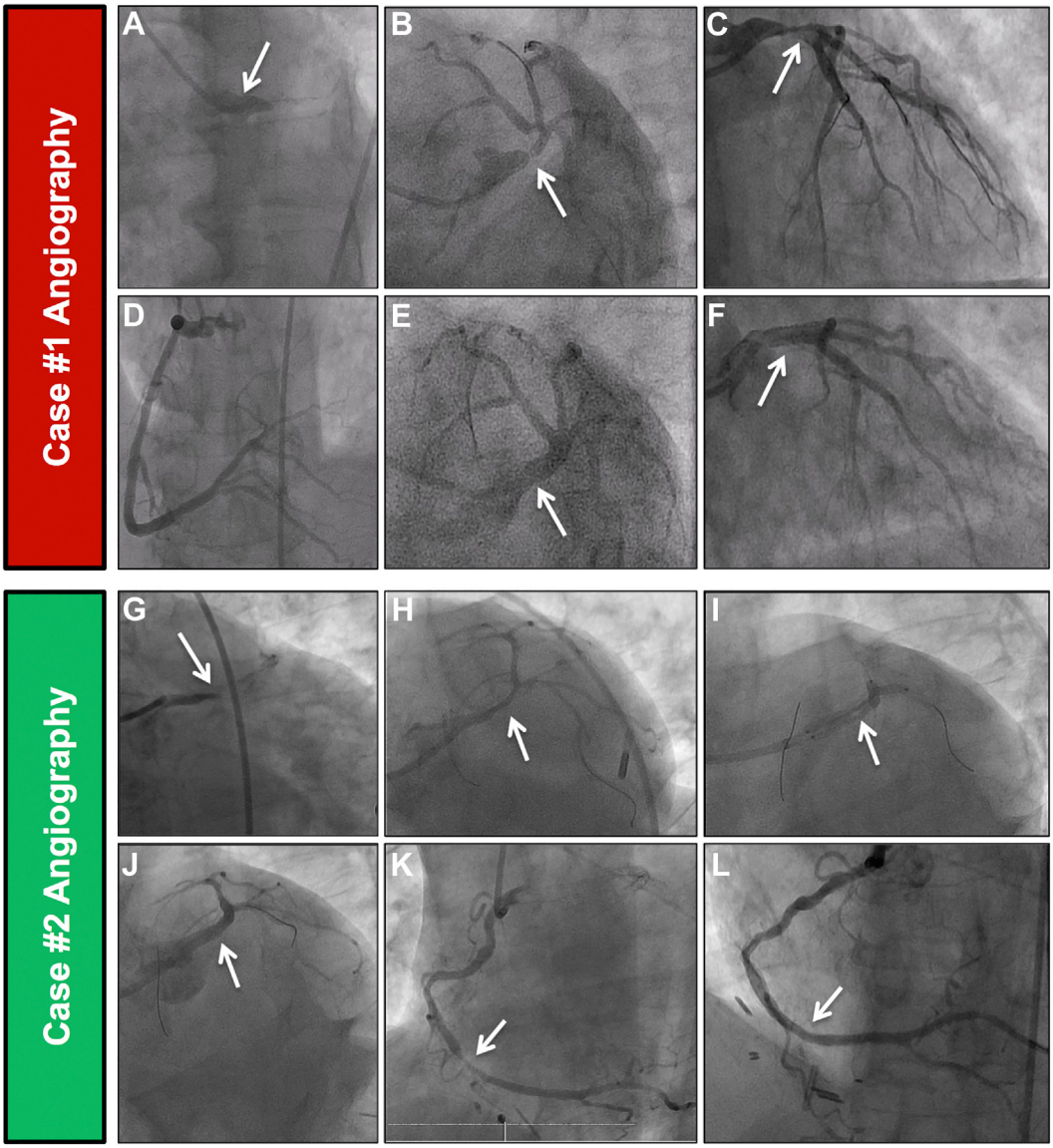
TIMI = Thrombolysis In Myocardial Infarction

FIGURE 1 Electrocardiograms



There are diffuse ST-segment elevations **(red arrows)** in the high-lateral (aVL, I) and anterior leads (case 1: V_2 to V_6; case 2: V_2 to V_4) plus reciprocal ST-segment depressions **(blue arrows)** in the inferior leads (II, III, aVF) and apico-lateral leads V_5 and V_6 (case 2), consistent with anterolateral ST-segment elevation myocardial infarction. ECG = electrocardiogram.

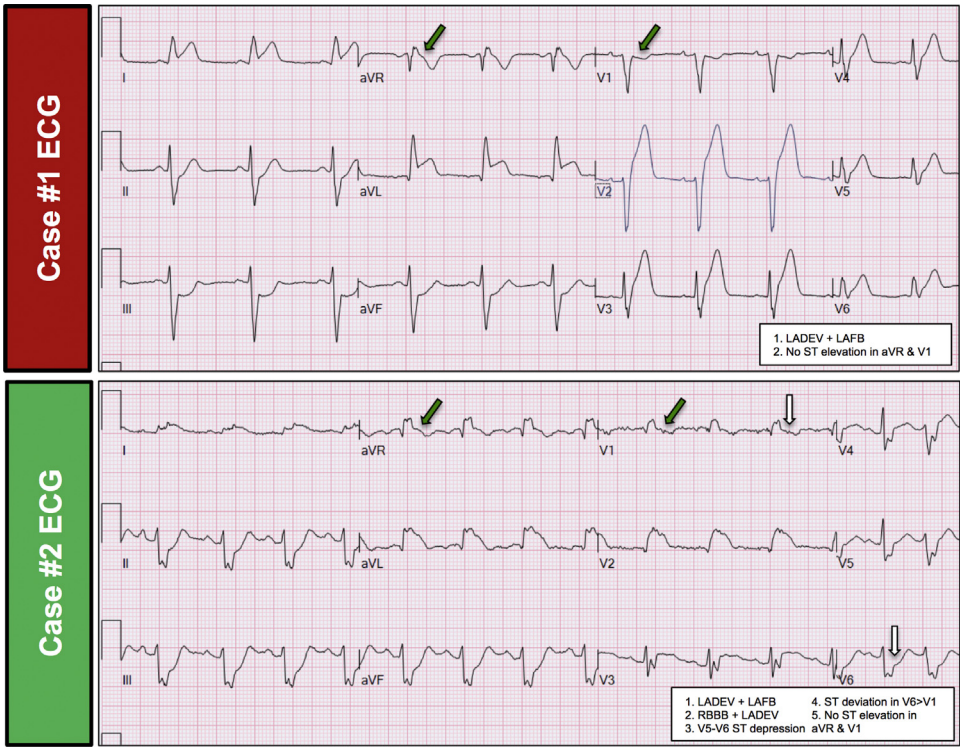
FIGURE 2 **Coronary Angiographies**



Case 1: **(A)** unprotected left main (LM) **(arrow)** acute total occlusion (Video 1); **(B,C)** severe obstructive atherothrombosis **(arrow)** was visualized (Video 2) after guidewire advancement; **(D)** normal dominant right coronary artery; **(E,F)** LM post-successful intra-aortic balloon pump–supported percutaneous coronary intervention (PCI) **(arrow)** (Video 3). Case 2: **(G)** LM acute total occlusion **(arrow)** (Video 4); **(H)** after guidewire advancement, severe atherothrombotic disease was observed (Video 5) involving the distal-LM **(arrow)**, proximal left anterior descending coronary artery, and the proximal circumflex coronary artery; **(I)** intra-aortic balloon pump–supported PCI was performed using simultaneous kissing stents technique **(arrow). (J)** Post-PCI of LM **(arrow)** into the proximal left anterior descending coronary artery and proximal circumflex coronary artery (Video 6); **(K, L)** mid-right coronary artery 80% stenosis **(arrows)** pre- and post-PCI, respectively.

no-reflow phenomenon occurred at the distal-LAD that was partially responsive to mechanical and pharmacologic interventions. His condition deteriorated into multiorgan failure requiring mechanical ventilation, vasopressors, and continuous IABP support. Cardiorespiratory arrest ensued 24 h later without return of spontaneous circulation despite resuscitation efforts.

FIGURE 3 **Electrocardiographic Findings Favoring a Left Main Coronary Artery Occlusion from a Proximal Left Anterior Descending Coronary Artery**



Electrocardiographic findings favoring a left main coronary artery occlusion from a proximal left anterior descending coronary artery: 1) coexistence of left axis deviation (LADEV) and left anterior fascicular block (LAFB); 2) concomitant right bundle branch block (RBBB) with LADEV (case 2); 3) V_5 to V_6 ST-segment depressions (case 2); 4) ST-segment deviation in $V_6 > V_1$ ($V_6/V_1 \geq 1$) **(white arrows);** 5) absence of aVR and V_1 ST-segment elevations **(green arrows).**

DISCUSSION

LM infarctions have two distinct electrocardiographic presentations. The first one is when a subtotal (≤99%) occlusion (TIMI flow grade >1) results in a non-STE acute coronary syndrome. It has been classically described as diffuse STDs (≥8 leads) involving the inferior and precordial leads plus a reciprocal STE in aVR that denotes global left ventricular subendocardial ischemia (1).

The second presentation, far more concerning and lethal, is a STEMI due to ATO with TIMI flow grade 0 of an unprotected LM, imposing an overwhelming burden of global left ventricular transmural ischemia (1,2). As in both cases reviewed (**Figure 1**), these patients will show STEs in the anterior (V_2 to V_5) and high-lateral leads (aVL, I) with reciprocal inferior STDs, plus they may or may not have aVR elevation as well as LADEV and RBBB (3). Conduction abnormalities can often arise as ischemia of the basal septum occurs. In fact, coexistence of LADEV (>−30°) and LAFB, as in cases 1 and 2 (**Figure 3**), has shown to predict LM occlusion with 95% specificity, and 88% positive (PPV) and negative predictive value (NPV) (4).

STE in aVR (≥1 mm) has been commonly used as a marker for LM infarction, yet it may still be absent in 20% to 38% of cases, as it was for both of our patients (3,5,6). Additionally, it may be present in almost 25% of LAD infarctions if occlusion is prior to the first septal branch (3). Hence, aVR STE if used alone may lead to overestimation of the LM infarction, or on the other extreme, miss this critical diagnosis. Consequently, meticulous evaluation of the electrocardiogram for the presence of other LM discriminatory findings (**Table 1**) seems a more reasonable interpretation strategy.

Two ST-segment deviation patterns (**Table 1**) other than aVR STE have shown to be more predictive of LM than proximal-LAD occlusion (5). The most predictive finding was the existence of larger STDs in lead II when compared with the magnitude of the STE

TABLE 1 Discriminatory ECG Findings Favoring LM Occlusion in Patients With Anterior or Anterolateral STEMI

First Author (Ref. #)	Electrocardiographic Findings Favoring LM Occlusion	Diagnostic Accuracy Findings
Hirano et al. (3)	New RBBB + LADEV	LM 37% vs. LAD 14%
Prieto-Solís et al. (4)	LADEV + LAFB	75% sensitivity and 95% specificity 88% of PPV and NPV
Hirano et al. (3)	STE in aVR	LM 62% to 88% vs. proximal LAD 8% to 43%
Fujii et al. (5)		PPV 36% to 42%
Yamaji et al. (7)		NPV 96% to 96.4%
Mahajan et al. (6)	STE in aVR/V_1 ≥1	63% sensitivity, 89% specificity, and 85% accuracy
Fujii et al. (5)	Magnitude of lead II STD > V_2 STE	LM 39% to 47% vs. proximal LAD 0% to 0.6% PPV 90% to 100% and NPV 94% to 95%
Fujii et al. (5)	STDs in V_5 and/or V_6	LM 59% to 62% vs. proximal LAD 4% to 7% PPV 46% to 62% and NPV 96% to 96.2%
Mahajan et al. (6)	V_6/V_1 ≥1	74% sensitivity, 89% specificity, and 82% accuracy
Fiol et al. (2)	No STE in aVR and V_1	100% (7 of 7) of patients with LM ATO had no STE in aVR and V_1, plus 60% of them had a new RBBB and LAFB

ATO = acute total occlusion; ECG = electrocardiography; LAD = left anterior descending coronary artery; LADEV = left axis deviation; LAFB = left anterior fascicular block; LM = left main coronary artery; NPV = negative predictive value; PPV = positive predictive value; RBBB = right bundle branch block; STD = ST-segment depression; STE = ST-segment elevation; STEMI = ST-segment elevation myocardial infarction.

in V_2 (PPV = 90% to 100%; NPV = 94% to 95%). The second best predictor was the presence of STDs in V_5 and/or V_6 (PPV = 46% to 62%; NPV = 96% to 96.2%) as present in case 2 (Figure 3).

Furthermore, as depicted by case 2 (Figure 3), a larger absolute magnitude of STD in V_6 over ST-segment deviation in V_1 (V_6/V_1 ≥1) was found to be more predictive of LM than LAD occlusion with 74% sensitivity, 89% specificity, and 82% accuracy (6). This finding performed comparably with the previously well-established criteria proposed by Yamaji et al. (7) of a higher STE in aVR than in V_1 (aVR/V_1 ≥1). The opposite (aVR/V_1 <1) is expected with proximal-LAD occlusions because the anterior ST-segment vector in V_1 is unopposed by the absence of posterior changes.

Unfortunately, most studies evaluating LM infarction electrocardiographic patterns made no distinction based on whether TIMI flow grade 0 was present (vs. TIMI flow grade ≥1) to compare and characterize this far more worrisome presentation (1,2).

In an attempt to address this dilemma, a case series of 7 STEMI patients with unprotected LM ATO and TIMI flow grade 0 revealed that 100% had no STE in leads aVR and V_1, just like in both of our patients (Figure 3), plus 60% of them had a new RBBB and LAFB (2). The explanation is that simultaneous transmural ischemia in the posterior (circumflex) and anterior (LAD) territories produced opposing ST-segment vectors that cancel each other, resulting in almost isoelectric ST-segments in these leads. Consequently, electrocardiographic analysis should look beyond lead aVR for different LM predictors (Table 1) to avoid missing this critical diagnosis.

CONCLUSIONS

Electrocardiographic recognition of an unprotected LM STEMI and its differentiation from a proximal-LAD occlusion can be quite challenging. Such analysis needs to be refined by looking beyond the traditional ST-segment changes in lead aVR. In case of aVR elevation, it requires comparison to V_1 (aVR/V_1 ≥1) for improved diagnostic accuracy. However, be aware that LM ATO with TIMI flow grade 0 may present without STEs in leads aVR and V_1, but still be recognizable after meticulous evaluation of the electrocardiogram for other supportive findings such as the presence of specific STDs, new conduction blocks, QRS axis deviation or after performing ST-segment displacement comparisons (Table 1).

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ADDRESS FOR CORRESPONDENCE: Dr. José Escabí-Mendoza, Director of the Accredited Chest Pain Center and the Cardiac Intensive Care Unit, Veterans Affairs Caribbean Healthcare System, Puerto Rico and U.S. Virgin Islands, 10 Casia Street, San Juan 00921, Puerto Rico. E-mail: Jose.Escabi@va.gov.

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KEY WORDS acute myocardial infarction, electrocardiogram, STEMI, unprotected left main coronary artery

APPENDIX For supplemental videos, please see the online version of this paper.