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Inferior Wall ST-Segment Elevation Myocardial Infarction (STEMI): Identifying The Culprit Artery from The Electrocardiogram

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ABSTRACT

The electrocardiogram (ECG) is an important tool in ST-segment elevation myocardial infarction (STEMI) for identifying the affected wall. In inferior wall STEMI, both the right coronary artery (RCA) and the left circumflex artery (LCxA) may be responsible for the event, and identification of the culprit artery becomes possible through the application of electrocardiographic criteria. We report two cases that illustrate the challenges and overlaps of certain ECG patterns and their angiographic correlations. In case 1, the first ECG showed ST-segment elevation in D2, D3 and aVF, with ST-segment elevation in D1 and ST-segment depression in aVR. In the second ECG, ST-segment elevation was evident in inferior wall, with higher elevation in D3 and with ST-segment depression in D1, aVL and V2. Coronary angiography showed an occluded LCxA with a negative image suggestive of thrombus, and chronic total occlusion of the RCA. In case 2, the ECG showed ST elevation in D3 and aVF, and ST depression in aVL, V2, V3 and V4. On a new ECG, ST-segment elevation was observed in D1, D2 and aVF, and ST-segment depression was observed in V2 and aVR. In coronary angiography, ostial chronic occlusion of the RCA was visualized, in addition to subocclusive stenosis in the second obtuse marginal branch and in the LCxA. The electrocardiographic findings of both cases highlight the importance of well-established criteria that contribute to identifying the occluded artery in inferior STEMI.

Keywords

ST Elevation Myocardial Infarction, Inferior Wall Myocardial Infarction, Electrocardiography, Coronary Reperfusion Therapy, Percutaneous Coronary Intervention, Coronary Vessels.

Introduction

Acute myocardial infarction with ST-segment elevation (STEMI) indicates total acute coronary occlusion and requires immediate reperfusion therapy. Primary percutaneous coronary intervention (PCI) is the preferred method for coronary reperfusion therapy in these cases, and the earlier it is performed, the better the patient's prognosis [1]. The electrocardiogram (ECG), a simple and low-cost test, is an important tool in identifying the affected wall in STEMI

and the occluded artery responsible for the event [2]. Furthermore, recognizing the affected artery from the ECG is important not only to determine which artery should be immediately treated, but also to estimate the risks of a more serious condition [3,4]. In inferior-wall STEMI, both the right coronary artery (RCA) and the left circumflex artery (LCxA) may be the culprit vessel [1], and this could lead to relevant clinical implications. Therefore, identifying the culprit artery before coronary angiography is of utmost importance, which becomes possible through the application of certain electrocardiographic criteria. We report two cases that illustrate the challenges and overlaps of certain patterns and their angiographic correlations.

Case Report Case 1

A 53-year-old male patient, hypertensive and a smoker for over 30 years, had been experiencing moderate retrosternal pain for approximately one month, without irradiation, lasting less than 20 minutes, related to exertion and improving with rest. In the early hours of December 3rd, 2023, he began experiencing severe retrosternal pain in a tight state, associated with dyspnea and paresthesia of the upper limbs, without improvement with rest. He sought the Emergency Care Unit, where the chest pain protocol was initiated, and an ECG was requested (Figure 1A), which showed ST-segment elevation in leads D2, D3 and AVF. Acetylsalicylic acid (ASA) and clopidogrel were administered in loading doses and a call was made to a referral hospital, aiming to perform primary PCI. After transfer, the patient was admitted to the referral service with controlled pain, hemodynamically stable, in Killip classification 1. A new ECG was performed on admission (Figure 1B) and showed a similar pattern, although with a higher ST-segment elevation in D3 than in D2, and with a deeper ST-segment depression in leads V2 and V4 (lead V3 was not registered).

The patient underwent urgent coronary angiography, using the right radial artery as the access route. Total occlusion was observed in the proximal third of the LCxA, suggestive of acute occlusion with negative imaging suggestive of intracoronary thrombus (Figure 2A). The LCxA was then approached, with implantation of two sirolimus-eluting drug-eluting stents (Supraflex Cruz®, SMT, Surat, India), successfully and uneventfully, with final TIMI III flow (Figure 2B). Time from onset of symptoms to ballon was six hours. Total occlusion of the RCA with a chronic appearance was also observed, with the presence of intracoronary collateral circulation. An attempt was made to approach the RCA with complementary PCI, but the attempt to cross the coronary occlusion with a 0.014" guidewire was unsuccessful (Figure 3B).

The patient showed good clinical progress, with no recurrence of chest pain. The transthoracic echocardiogram performed the following day showed mild left ventricular systolic dysfunction due to inferior basal and inferoseptal akinesia and lateral hypokinesia, with an ejection fraction of 48% by the Simpson method. After three days, the patient was discharged and referred for outpatient follow-up.

Case 2

A 64-year-old male patient with no previous comorbidities was referred from the infectious diseases outpatient clinic, where he was being investigated for progressive dyspnea over the last seven days, associated with an episode of fainting, without loss of consciousness. At the Emergency Room, he reported mild chest discomfort with atypical characteristics for approximately eight hours. An ECG was performed, which showed ST-segment elevation in leads D3 and aVF and ST-segment depression in leads aVL, V2, V3, and V4 (Figure 4A). After 30 minutes, a new ECG was performed with leads V7, V8, and V9 (Figure 4B), which showed ST-segment elevation in leads D1, D2, and aVF and STsegment depression in leads V2 and aVR. A serum ultrasensitive troponin level was also requested, and the result was 4,897 pg/ ml (reference value < 26.2 pg/ml). The Interventional Cardiology Department was contacted and loading doses of ASA and clopidogrel were administered.

The patient underwent coronary angiography using the radial access route. Total ostial occlusion of the RCA was observed, with the presence of intracoronary circulation and an angiographic aspect suggestive of a chronic lesion (Figure 5A). Subocclusive lesions were also observed in the second obtuse marginal branch (OM2) with reduced flow and in the LCxA, in addition to a severe obstructive lesion in the left anterior descending artery (LAD), in its proximal segment (Figure 5B). A PCI attempt for the RCA was made without success in passing the 0.014" guidewire through the ostial occlusion. Primary PCI for the OM2 branch and the LCxA

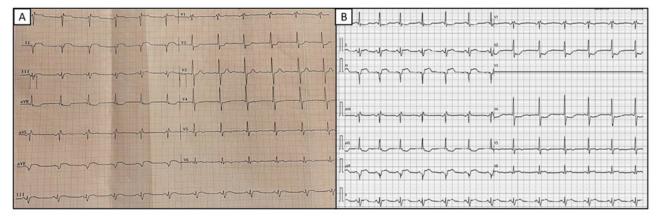


Figure 1: (A) First electrocardiogram performed showing acute myocardial infarction with ST-segment elevation in the inferior wall (leads D2, D3 and aVF), with ST-segment elevation in D1 and ST-segment depression in aVR and V2. It is noted that, in the inferior wall leads, the ST-segment elevation is wider in D2 than in D3. (B) Second electrocardiogram performed showing ST-segment elevation in the inferior wall (leads D2, D3 and aVF) and ST-segment depression in leads D1, aVL and V2. In this electrocardiogram, the ST-segment elevation is higher in D3 than in D2.

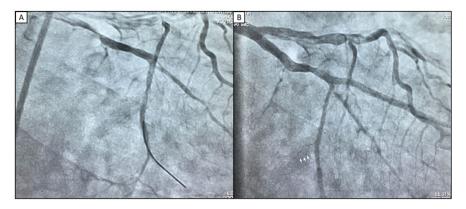


Figure 2: Left circumflex artery before and after primary percutaneous coronary intervention. (A) Left circumflex artery with occlusion in the proximal segment and negative image suggestive of thrombus. (B) Left circumflex artery after primary percutaneous coronary intervention with implantation of 2 drug-eluting stents.

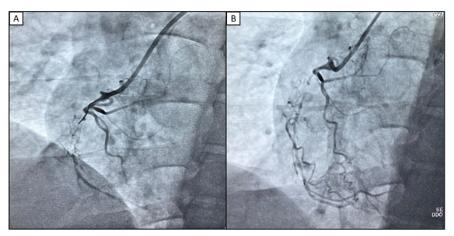


Figure 3: (A) Right coronary artery with total occlusion in the proximal segment with a chronic appearance and intracoronary collateral circulation; presence of a 0.014" guidewire during an attempt of percutaneous coronary intervention, which was unsuccessful in crossing the point of occlusion. (B) Final angiography of the unchanged right coronary artery, with opacification of the distal bed through intracoronary collateral circulation.

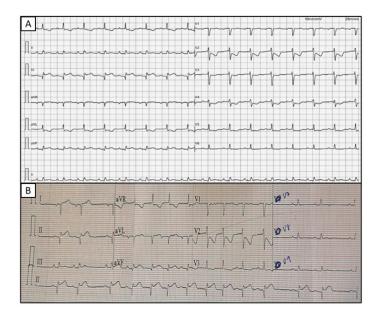


Figure 4: (A) First electrocardiogram showing ST-segment elevation in leads D3 and aVF and ST-segment depression in leads aVL, V2, V3 and V4. (B) Second electrocardiogram showing ST-segment elevation in leads D1, D2 and aVF and ST-segment depression in aVR and V2.

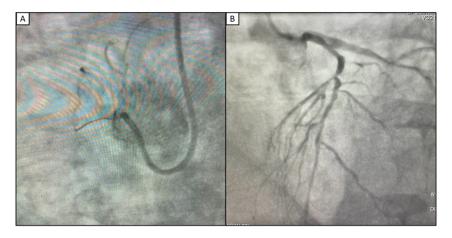


Figure 5: (A) Right coronary artery showing total occlusion at the ostium with a chronic appearance; presence of a 0.014" guidewire during an attempt of percutaneous coronary intervention, which was unsuccessful in crossing the point of occlusion. (B) Left coronary artery in left cranial projection, with multiple severe obstructive atherosclerotic lesions.

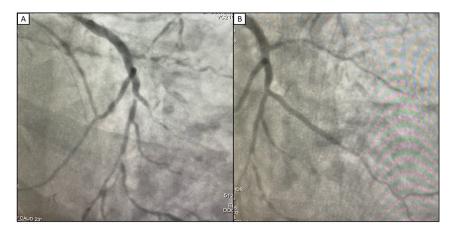


Figure 6: Left circumflex artery and second obtuse marginal branch with subocclusive lesions and restriction of antegrade flow, before (A) and after (B) primary percutaneous coronary intervention with implantation of two sirolimus-eluting stents.

was performed, with implantation of two drug-eluting stents (Supraflex Cruz®, SMT, Surat, India), achieving TIMI II flow (Figure 6). Flow disturbance during PCI was partially reverted after intracoronary administration of nytroglycerin and tirofiban. A complementary PCI was scheduled to address the residual coronary lesions within 48 to 72 hours. However, the patient died after presenting acute and progressive shock followed by cardiorespiratory arrest with pulseless electrical activity.

Discussion

The identification of the artery related to the myocardial infarction, although challenging, can be performed through the ECG and guide clinical management procedures. It is known that in 80% to 90% of cases of inferior wall STEMI, the RCA is the culprit artery [5], while in the other 10% to 20% the LCxA is occluded. In cases of occlusion of the RCA before the origin of the acute marginal branch, complications such as right ventricular (RV) failure can be expected [6], since this is the branch responsible for irrigating this chamber. Patients who present RV infarction in association with inferior wall STEMI have a worse prognosis and higher

mortality when compared to patients with isolated inferior STEMI [3]. Therefore, in addition to guiding clinical procedures, early identification of the affected artery based on the ECG is important to estimate the risk of a more serious condition.

ECG changes are dynamic, which can make it difficult to accurately identify the culprit artery [5]. This difficulty leads to prolonged time until coronary reperfusion, increasing the extent of myocardial injury. In addition, variations in the coronary anatomy of patients also pose a challenge in interpreting the ECG regarding the culprit artery in STEMI [7]. In both reported cases, it was noted that the characteristics of the serial ECGs changed, and none of them were conclusive in defining the affected artery using known algorithms. However, the application of electrocardiographic criteria can present high predictive values in identifying the affected artery, even in patients with multivessel disease and confounding factors [7].

Among the criteria that help determine the affected artery in inferior wall STEMI, the amplitude of ST-segment elevation and

the presence or absence of ST-segment elevation or depression in certain leads are used. The alterations that are consistent with involvement of the RCA are: 1: ST-segment elevation in D3 > ST-segment elevation in D2; 2: ST-segment depression in aVL; 3: ST-segment elevation in V1; 4: (ST-segment elevation in D2 + D3 + aVF) > (ST-segment depression in V1 + V2 + V3) [2,4,5]. The absence of such characteristics in the ECG is consistent with the culprit artery of the LCxA, and ST-segment depression in leads V1 and V2 is a very sensitive sign of its involvement [8]. The diagnostic algorithm in Figure 7 demonstrates the reasoning about the culprit artery in inferior wall STEMI based on the 12lead ECG.

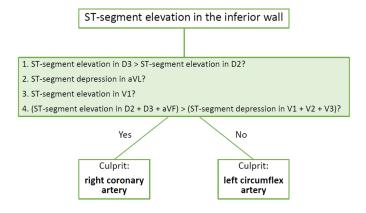


Figure 7: Simple diagnostic algorithm for reasoning about the culprit artery in ST-segment elevation myocardial infarction compromising the inferior wall, based on the 12-lead ECG. The characteristics described, if present, point to a greater possibility of involvement of the right coronary artery.

Other criteria that can be applied to diagnose the culprit LCxA artery are: ST-segment elevation in V6 \ge 0.1 mV or > ST-segment elevation in D3; and a ratio (ST-segment depression in V3) / (ST-segment elevation in D3) > 1.2 [9]. A recent study sought to trace electrocardiographic differences between LCxA and RCA involvement, with the advantage of studying patients with a wide range of clinical characteristics, such as a history of previous myocardial infarction, left ventricular hypertrophy, right bundle branch block, and multivessel coronary disease. Patients with RCA occlusion presented ST-segment elevation in leads D3 and aVF and ST-segment depression in leads D1 and aVL, while LCxA occlusion was more associated with ST-segment elevation in leads V5 and V6 and deeper ST-segment depression in leads V1 to V3 [10]. This new diagnostic algorithm, represented in the diagnostic flowchart in Figure 8, has a sensitivity of 77% and a specificity of 86% in identifying the artery related to the inferior infarction. Noteworthly, the first ECG in case 1 is more consistent with LCxA involvement, presenting ST-segment elevation in D3 that is smaller than ST-segment elevation in D2, absence of ST-segment depression in aVL and elevation in V1. In the second ECG, there was an inversion of this pattern, showing ST-segment elevation in D3 that is greater than ST-segment elevation in D2 and presence of ST depression in aVL, which is more consistent with RCA

involvement. Interestingly, the coronary angiography confirmed acute occlusion of the LCxA, while the RCA also presented total occlusion of its lumen, although with a chronic appearance.

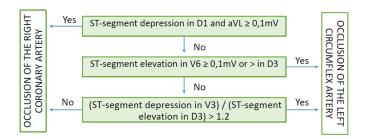


Figure 8: Diagnostic flowchart to differentiate the culprit artery in STsegment elevation myocardial infarction compromising the inferior wall, based on the 12-lead ECG. The criteria consist of comparisons of the amplitude of the ST-segment elevation or depression in different leads, enabling the identification of the right coronary artery or the left circumflex artery as the culprit vessel.

Adapted from: Vives-Borrás M, Maestro A, García-Hernando V, Jorgensen D, Ferrero-Gregori A, Moustafa AH, et al. Electrocardiographic Distinction of Left Circumflex and Right Coronary Artery Occlusion in Patients with Inferior Acute Myocardial Infarction. Am J Cardiol. 2019 Apr 1;123(7):1019-1025.

A variation in characteristics can also be seen in the ECGs of patient 2. The first ECG showed ST-segment elevation in D3 and aVF and ST-segment depression in aVL, which is more compatible with culprit RCA. The second ECG showed ST-segment elevation in D1, D2 and aVF and ST-segment depression in V2, which is more consistent with LCxA involvement. The cardiac catheterization showed a multivessel coronary artery disease, with an appearance of chronic occlusion of the RCA. These findings highlight the diagnostic challenge and the possibility of error, even with the proper application of diagnostic algorithms, in cases of multivessel coronary disease. Artificial intelligence algorithms, especially with machine learning and deep learning tools, may emerge to increase diagnostic accuracy in cases such as these, which require delicate ECG interpretation [11]. Such technologies are advancing rapidly and medical research is making progress in this direction, as well as in the field of imaging diagnosis. Although promising, more studies are needed to achieve greater diagnostic accuracy from electrocardiographic analysis and reduce the possibility of errors in the management of a highly prevalent condition as STEMI.

The analysis of these two cases, which are very similar in terms of electrocardiographic findings with sequential changes, shows the challenge in managing patients with inferior wall STEMI, in whom there may be a greater delay in coronary reperfusion due to erroneous interpretation of the ECG [5]. However, the application of diagnostic criteria can greatly contribute to good medical practice, enabling earlier identification of the culprit artery, optimizing clinical management and reducing the delay until coronary reperfusion. In this scenario of STEMI, every effort should be made to improve outcomes and reduce mortality.

References

- Gulati M, Levy P, Mukherjee D, et al. 2021 AHA/ACC/ASE/ CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. JACC. 2021; 78: e187-e285.
- Tragardh E, Claesson M, Wagner GS, et al. Detection of Acute Myocardial Infarction Using the 12-Lead ECG plus Inverted Leads versus the 16-Lead ECG (with Additional Posterior and Right-Sided Chest Electrodes). Clin Physiol Funct Imaging. 2007; 27: 368-374.
- 3. Mehta SR, Eikelboom JW, Natarajan MK, et al. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. J Am Coll Cardiol. 2001; 37: 37-43.
- 4. Berger PB, Ryan TJ. Inferior Myocardial Infarction. High-Risk Subgroups. Circulation. 1990; 81: 401-411.
- Zhou P, Wu Y, Wang M, et al. Identifying the culprit artery via 12-lead electrocardiogram in inferior wall ST-segment elevation myocardial infarction: A meta-analysis. Ann Noninvasive Electrocardiol. 2023; 28: e13016.

- 6. Marques A, Cruz I, Briosa A, et al. Isolated Right Ventricle Myocardial Infarction - Is the Right Ventricle Still the Forgotten Ventricle?. Arq Bras Cardiol. 2021; 116: 32-35.
- 7. Tierala I, Nikus KC, Sclarovsky S, et al. HAAMU Study Group. Predicting the Culprit Artery in Acute ST-Elevation Myocardial Infarction and Introducing a New Algorithm to Predict Infarct-Related Artery in Inferior ST-Elevation Myocardial Infarction: Correlation with Coronary Anatomy in the HAAMU Trial. J Electrocardiol. 2009; 42: 120-127.
- Samesima N, God EG, Kruse JCL, et al. Brazilian Society of Cardiology Guidelines on the Analysis and Issuance of Electrocardiographic Reports - 2022. Arq Bras Cardiol. 2022; 119: 638-680.
- Bairey CN, Shah PK, Lew AS, et al. Electrocardiographic Differentiation of Occlusion of the Left Circumflex versus the Right Coronary Artery as a Cause of Inferior Acute Myocardial Infarction. Am J Cardiol. 1987; 60: 456-459.
- Vives-Borras M, Maestro A, García-Hernando V, et al. Electrocardiographic Distinction of Left Circumflex and Right Coronary Artery Occlusion in Patients with Inferior Acute Myocardial Infarction. Am J Cardiol. 2019; 123: 1019-1025.
- Paixao GMM, Santos BC, Araujo RM, et al. Machine Learning in Medicine: Review and Applicability. Arq Bras Cardiol. 2022; 118: 95-102.

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