Enfarte Agudo de Miocárdio Inferior: Além da Elevação do Segmento ST

Acute Inferior Myocardial Infarction: Beyond the ST-Segment Elevation

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Resumo:

A síndrome coronária aguda (SCA) é considerada um *continuum*, que abrange tanto o SCA sem elevação do segmento ST, incluindo a angina instável e o enfarte agudo do miocárdio sem elevação do segmento ST (EAMSEST), e também o EAM com elevação do segmento ST (EAMCEST). O diagnóstico de EAM está associado à elevação da troponina e a sua classificação tem sido baseada fundamentalmente na presença ou ausência do supradesnivelamento do segmento ST no eletrocardiograma (ECG). No entanto, a elevação do segmento ST é um preditor inadequado de oclusão coronária aguda (OCA). Evidência crescente sugere que uma fração substancial de doentes com EAMSEST têm uma OCA. Pela ausência dos achados clássicos no ECG, estes doentes não são considerados para revascularização imediata. O caso clínico apresentado é referente a um padrão específico de ECG que potencialmente identifica uma OCA, mas que não cumpre os critérios eletrocardiográficos de EAMCEST, o padrão de Aslanger.

Palavras-chave: Electrocardiografia; Infarte Miocárdico de Parede Inferior.

Abstract:

Acute coronary syndromes (ACS) are considered a continuum, enclosing both non-ST-elevation (NSTE)-ACS, including unstable angina and non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). The diagnosis of myocardial infarction (MI) is associated with troponin release. Classification of MI has fundamentally been based on the presence or absence of ST-segment elevation (STE) on the electrocardiogram (ECG). STE is a poor surrogate for acute coronary occlusion (ACO) causing MI. Mounting evidence suggests that a substantial fraction of patients presenting with NSTEMI have an ACO of their culprit artery. Due to a lack of classic ECG findings, these NSTEMI patients, despite ACO of the culprit artery, may be missed, leading to either delay in or no revascularization. This case report highlights one specific pattern potentially identifying ACO that does not meet STEMI criteria, the Aslanger pattern. Early identification of this NSTEMI pattern potentially accelerates revascularization.

Keywords: Electrocardiography; Inferior Wall Myocardial Infarction.

Introduction

Acute coronary syndromes (ACS) involve a range of conditions, including patients presenting with recent changes in clinical signs or symptoms, with either the presence or absence of 12-lead electrocardiogram (ECG) changes or acute elevations in cardiac troponin concentrations.[1] When presenting with suspected ACS, patients are typically classified based on ECG at presentation. Patients presenting with a normal ECG or without persistent ST-segment elevation (STE) and a typical rise and fall in cardiac troponin levels, will be classified as non-ST-elevation myocardial infarction (NSTEMI); whereas, if troponin level is negative, a diagnosis of unstable angina is made. Conversely, ACS with persistent STE, in the appropriate clinical context, is suggestive of ongoing acute coronary artery occlusion, designating the priority for these patients for immediate reperfusion therapy, in the setting of ST-segment elevation myocardial infarction (STEMI).[1,2]

ACS is a major cause of mortality and morbidity, often being the first clinical manifestation of cardiovascular disease.[1,3] The refinement in the operational diagnosis of NSTEM, including the more frequent use of high-sensitivity troponin assays, has increased the fraction of NSTEMI amongst MI. In recent years, nearly two-thirds of acute MI are classified as NSTEMI.[4]

Current clinical practice guidelines recommend an emergent invasive strategy with a main focus on patients with STEMI.[1] Despite being considered the standard for many years, STE may not encompass the entire population of patients who may benefit from emergent revascularization. In fact, patients with MI with acute coronary occlusion (ACO) are the cohort that is believed to benefit from emergent reperfusion therapy.[2,5,6]

Investigators find STE a poor surrogate for ACO causing myocardial infarction, advocating for a new paradigm: ACO-MI (designated OMI)/non-OMI.6-9 This conceptual shift highlights the underlying pathology instead of focusing on insufficient surrogate test results, namely STE.[5,10] Furthermore,

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current guidelines demand the presence of STE in two contiguous leads, resulting in reduced sensitivity for STEMI in some configurations. This strict conception may even prevent the discovery of new ECG patterns indicating ACO and therefore limiting access to emergent acute reperfusion.[1,11]

Several studies demonstrate that a substantial fraction of NSTEMI has an ACO of their culprit artery on invasive coronary angiography (CA).[2,4,6,12] Literature shows STEMI criteria miss nearly one-third of ACO.[2,3,5,6,8,13]

It is important to recognize that the diagnosis of NSTEMI demands the necessity of CA, to be performed during hospitalisation, but often delayed relative to STEMI management, excepting cases of haemodynamic instability or clinical deterioration.[1] This delay, being more than 24 hours in most studies, poses a major concern.[4] Evidence suggests that the ACO population displays substantially higher major adverse cardiac event rates and all-cause mortality compared with patients without total occlusion; being the specific cohort that is believed to benefit from emergent reperfusion therapy.[2,3,6,10]

Based on several clinical observations, it has been hypothesized that a subgroup of inferior MI may show such a unique pattern and might be incorrectly labelled as NSTEMI.[11] This case report refers to an ACO presenting with solitary STE observed in lead III in the ECG at admission.

Case Report

We describe the case of a 91-year-old female, resident in a care unit facility, with a medical history of paroxysmal atrial fibrillation, diabetes mellitus, peripheral arterial disease and dyslipidaemia, who had been progressively more dependent in daily activities for the last few months, including self-care. She was admitted to the emergency department with a history of chest tightness and dyspnea that had started 2 hours before. In the initial evaluation, the patient was polypneic, with O2 saturation of 82% with oxygen nasal cannula at 2 L/min, basal rales audible on pulmonary auscultation, blood pressure of 165/60 mmHg, heart rate of 98 bpm and signs of poor peripheral perfusion, such as cold extremities and a capillary refill time around 4 seconds. The ECG revealed an Aslanger pattern (Fig. 1) and antiplatelet therapy was administered.

A point-of-care cardiac ultrasound was performed revealing hypokinesis of lateral and inferior cardiac walls and the apical segments of the remaining walls, with moderate to severely reduced left ventricle ejection fraction. Testing revealed elevated troponin-I of 777.2 ng/L (normal range < 16 ng/L) and haemoglobin 7.9 g/dL. A percutaneous coronary intervention (PCI) was delayed due to intolerance of the dorsal decubitus position. After clinical stabilization, a CA was performed and showed multi-vessel disease, including a 99% calcified ostial lesion in the circumflex artery and a subocclusive lesion of the medium segment of the right coronary artery, with collateral circulation (Fig. 2). Although the occlusion of the right coronary artery was considered the culprit, all attempts to pass through the lesions failed, confirming the chronic character of the lesion. Considering the complexity of the coronary disease and the advanced age and frailty of the patient, ultimately, a conservative strategy was favoured. Optimization of medical therapy was conducted, including management of anaemia with red blood cell transfusion.

Discussion

Current guidelines require STE to be present in two contiguous leads referring to a specific localization group (i.e., anterior, lateral, inferior) for the localization and diagnosis of STEMI.[11] Mounting evidence reveals that many patients with ACO causing MI have STE that does not meet STEMI criteria. Other ECG indicators associated with ACO have also been found, including, terminal QRS distortion, Q waves,

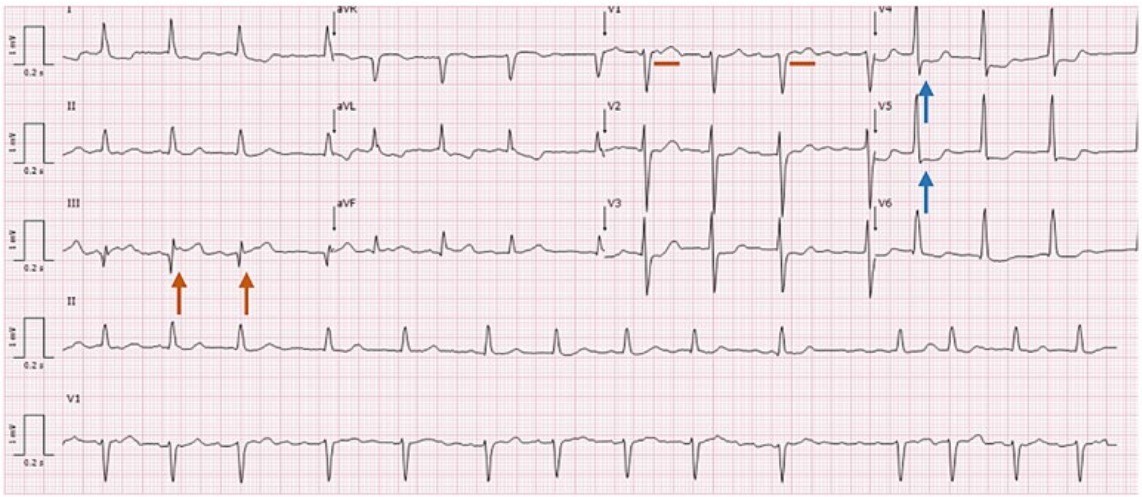

Figure 1: The 12-lead ECG at admission, besides atrial fibrillation, shows specific ECG findings of Aslanger pattern: STE in DIII, but not in any other inferior lead (orange arrows), ST-segment depression in leads V4-V5 (blue arrows) with a terminally positive T-wave and ST in lead V1 higher than ST in V2 (orange line).

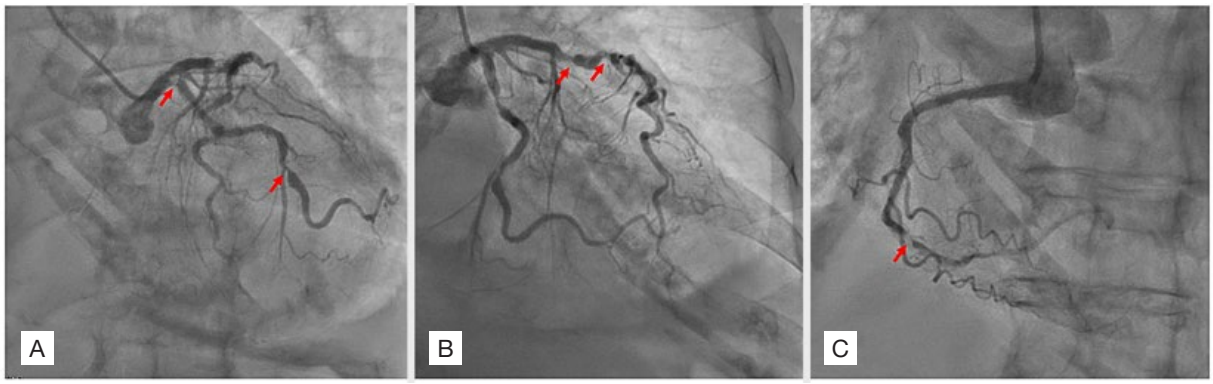

Figure 2: **(A)** Coronary angiogram showed an ostial calcified subtotal occlusion in the circumflex artery (upper arrow), as well as a stenosis in the mid-segment of 90% (arrow below). **(B)** The mid-segment of the anterior descending coronary showed a long lesion with a maximum severity of 90% (arrows). **(C)** The right coronary artery showed a subocclusion in the mid-segment with collaterals, suggesting a chronic occlusion (arrow).

hyperacute T-waves, Winter's sign, resting U-wave inversion, amongst others.[5] Data shows that ECG can reliably recognize ACO with the help of many findings other than STE.[1,5,9,10,14]

The term STEMI is commonly accepted as the surrogate for acute coronary occlusion MI, causing a direct implication in therapeutic strategies.[5,6] However, STEMI criteria have limited diagnostic accuracy for ACO, as 15% to 35% of emergent cardiac catheterization activations have no culprit lesion and are found to be false positives.[5,9]

Conversely, studies reveal that under the conventional STEMI/NSTEMI paradigm, up to 30% of NSTEMI have initially unnoticed ACO, discovered on delayed angiogram.[3,5,10] This subset of NSTEMI behaves like overt STEMI.[2] Clinical importance is obvious as patients with ACO presenting with NSTEMI had been shown to have similar infarct size measured by peak troponin, but greater delays to angiography compared with patients with OMI and STEMI presentation.[8,10,14] Studies show these patients have increased mortality and increased re-infarction rates compared to patients with patents vessels.[4,8] NSTEMI patients will experience a delayed revascularization procedure because of the lack of "appropriate" ECG findings, despite the presence of true coronary occlusions.[5,9]

Concerning NSTEMI, guidelines determine emergent angiography for patients with symptoms highly suspicious for ACO and with instability or persistent symptoms, regardless of ECG findings. By advocating multiple other diagnostic adjuncts in addition to the ECG, these guidelines corroborate the fundamental assumption that ACO is the underlying pathology that warrants emergent reperfusion, rather than the ECG millimetre criteria which may or may not be present.[5] Hence, there ought to be awareness of the current criteria limitations and strict ST--segment changes, and new electrocardiographic findings are considered necessary as an instrument in the diagnosis.[13,14]

Aslanger *et al* recently reported an electrocardiographic finding in acute inferior MI that does not meet STEMI criteria. The Aslanger pattern focuses on subtle 12-lead ECG changes, dispending right-sided leads, and identifying patients with inferior MI, frequently with multi-vessel disease.[11] Among the NSTEMI patients, this group has a higher in-hospital mortality rate and is more likely to have hemodynamic deterioration, requiring induction of mechanical circulatory support.[13] Despite not presenting as STEMI, the recognition of this pattern must lead to rapid revascularization, as it presents as both a diagnostic tool to complement STEMI criteria and as a predictor of poor prognosis.[11,13]

The Aslanger pattern is defined by three criteria: (1) ST depression in any of leads V4 to V6 but not in V2, (2) any STE in DIII but not in other inferior leads, (3) ST in lead V1 higher than ST in V2. Despite being classified as NSTEMI, there is an acute atherothrombotic event frequently resulting in inferior MI with at least one accompanying stable but critical stenosis in one of the non-culprit arteries. These patients, tend to have not only multi-vessel disease but also multiple comorbidities and higher baseline risk, resulting in increased short and long-term mortality.[11]

The ECG has impaired sensitivity to detect acute ischemia or necrosis affecting posterior or lateral myocardial walls, i.e. when the right coronary or left circumflex arteries are involved. Indeed, studies suggest that the inferolateral territory is more frequently involved among patients with NSTEMI with ACO. Specifically, data indicates that approximately 13% of inferior MI may present with the Aslanger pattern.[10,11]

The patient described, initially presented an ECG pattern consistent with the characteristics described by Aslanger *et al*[11] Subsequent CA confirmed multi-vessel disease. Technically, percutaneous revascularization of the culprit lesion was not feasible, despite multiple attempts. Attending to the complexity of the coronary disease, including calcified and complex lesions, the risk of intervention was considered high for periprocedural complications and future restenosis of the stent, demanding potent antiplatelet therapy. The basal characteristics of the patient, advanced age and comorbidities, including severe anaemia, favoured a conservative strategy. The conservative approach in this group of patients may be

considered, as some studies show that invasive management did not modify long-term outcomes in comorbid elderly patients, or its benefit is progressively reduced with an increased comorbidity burden.[15]

In conclusion, as the incidence of NSTEMI increases worldwide, the number of NSTEMI presenting with ACO is also increasing. Studies show that there is an unmet need regarding the identification of ACO and OMI in this group. The Aslanger ECG pattern can be used as a diagnostic criterion for inferior MI indicating the presence of severe stenosis in multiple vessels requiring emergency intervention. Our case illustrates the importance of rapid identification of this NSTEMI pattern, potentially demanding rapid revascularization in this group. ■

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Todos os autores aprovaram a versão final a ser publicada

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DR, PS, JG - Preparation of the manuscript, writing and bibliographical review.
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REFERENCES

1. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J. 2023, 12;44:3720-826. doi: 10.1093/eurheartj/ehad191
2. Sankardas MA, Ramakumar V, Farooqui FA. Of Occlusions, Inclusions, and Exclusions: Time to Reclassify Infarctions? Circulation. 2021, 144:333-5. doi: 10.1161/CIRCULATIONAHA.121.055827
3. Khan AR, Golwala H, Tripathi A. Impact of total occlusion of culprit artery in acute non-ST elevation myocardial infarction: a systematic review and meta-analysis. Eur Heart J. 2017;38:3082-89. doi: 10.1093/eurheartj/ehx418.
4. Tziakas D, Chalikias G, Al-Lamee R, Kaski RC. Total coronary occlusion in non ST elevation myocardial infarction: Time to change our practice? Int J Cardiol. 2021; 329:1–8. doi: 10.1016/j.ijcard.2020.12.082
5. Meyers HP, Bracey A, Lee D, Lichtenheld A, Li WJ, Singer DD, et al. Accuracy of OMI ECG findings versus STEMI criteria for diagnosis of acute coronary occlusion myocardial infarction. Int J Cardiol Heart Vasc. 2021;33:100767. doi: 10.1016/j.ijcha.2021.100767
6. Aslanger EK, Meyers PH 2, Smith SW: STEMI. A transitional fossil in MI classification? J Electrocardiol. 2021;65:163-9. doi: 10.1016/j.jelectrocard.2021.02.001.
7. Aslanger EK, Meyers HP, Bracey A, Smith SW. The STEMI/NonSTEMI Dichotomy needs to be replaced by Occlusion MI vs. Non-Occlusion MI. Int J Cardiol. 2021;330:15. doi: 10.1016/j.ijcard.2021.02.015
8. Meyers HP, Bracey A, Lee D, Lichtenheld A, Li WJ, Singer DD, et al. Comparison of the ST-Elevation Myocardial Infarction (STEMI) vs. NSTEMI and Occlusion MI (OMI) vs. NOMI Paradigms of Acute MI. J Emerg Med. 2021;60:273-84. doi: 10.1016/j.jemermed.2020.10.026.
9. Aslanger EK, Yıldırımtürk O, Simsek B, Bozbeyoglu E, Simsek MA, Yücel Karabay C, et al. DIagnostic accuracy oF electrocardiogram for acute coronary OCClUsion resuLTing in myocardial infarction (DIFOCCULT Study). Int J Cardiol Heart Vasc. 2020;30:100603. doi: 10.1016/j.ijcha.2020.100603.
10. Aslanger EK, Meyers HP, Smith SW. Time for a new paradigm shift in myocardial infarction. Anatol J Cardiol. 2021;25:156-62. doi: 10.5152/AnatolJCardiol.2021.89304.
11. Aslanger E, Yıldırımtürk O, Simsek B, Sungur A, Türer Cabbar A, Bozbeyoglu E, et al. A new electrocardiographic pattern indicating inferior myocardial infarction. J Electrocardiol. 2020;61:41-6. doi: 0.1016/j.jelectrocard.2020.04.008.
12. Koyama Y, Hansen PS, Hanratty CG, Nelson GIC, Rasmussen HH. Prevalence of coronary occlusion and outcome of an immediate invasive strategy in suspected acute myocardial infarction with and without ST--segment elevation. Am J Cardiol. 2002;90:579-84. doi: 10.1016/s0002-9149(02)02559-6.
13. Miyauchi E, Kuwazuru K, Arikawa R, Tokutake D, Chaen H, Oketani N, Ohishi M. Clinical Features of the Aslanger Pattern to Compensate for the Limitation of ST-Elevation Myocardial Infarction (STEMI) Criteria. Cureus. 2023;15:e33227. doi:m10.7759/cureus.33227.
14. Al-Zaiti SS, Martin-Gill C, Zègre-Hemsey JK, Bouzid Z, Faramand Z, Alrawashdeh MO, et al. Machine learning for ECG diagnosis and risk stratification of occlusion myocardial infarction. Nature Med. 2023;29:1804–13. doi: 10.1038/s41591-023-02396-3.
15. Sanchis J, Núñez E, Barrabés JÁ. Randomized comparison between the invasive and conservative strategies in comorbid elderly patients with non--ST elevation myocardial infarction. Eur J Intern Med. 2016;35:89-94. doi: 10.1016/j.ejim.2016.07.003.