Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Cardiology

Subtle Inferior Occlusion Myocardial Infarction with Aslanger's Pattern: A Case Report

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DOI: https://doi.org/10.36347/sjmcr.2026.v14i03.055 | **Received:** 03.02.2026 | **Accepted:** 17.03.2026 | **Published:** 28.03.2026

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Abstract | **Case Report**

Background: Delayed myocardial infarction presentations can show subtle or non-diagnostic ECG changes despite acute coronary occlusion, particularly in patients with diabetes. ***Case:*** A 65-year-old woman with type 2 diabetes presented 36 hours after persistent constrictive chest pain radiating to the left arm and jaw. She was hemodynamically stable. ECG showed sinus rhythm with first-degree AV block and minimal ST elevation in lead III with reciprocal depression in I and aVL, not meeting formal STEMI criteria. High-sensitivity troponin rose markedly; bedside echocardiography demonstrated inferior wall hypokinesis with preserved LVEF (53%). ***Management and outcome:*** Given high clinical suspicion, she received aspirin and prasugrel and was taken for urgent angiography, which revealed a proximal thrombotic RCA occlusion and a critical proximal LAD stenosis. PCI of the RCA with two drug-eluting stents restored TIMI 3 flow without complications. She recovered in the CICU and was discharged after 48 hours on guideline-directed therapy. ***Conclusion:*** This case highlights the need to integrate clinical presentation, biomarkers, bedside imaging, and early invasive assessment in delayed or atypical MI—especially in diabetics—to achieve timely revascularization and favorable outcomes.

Keywords: Subtle STEMI, Inferior Myocardial Infarction, Type 2 Diabetes, Delayed Presentation, Echocardiography, Percutaneous Coronary Intervention.

INTRODUCTION

Certain STEMI presentations exhibit only minimal ST-segment abnormalities, making diagnosis challenging and potentially leading to delayed reperfusion. These "subtle STEMI" patterns can closely mimic NSTEMI, especially when ST-segment deviations fall below conventional diagnostic thresholds, thereby requiring a high index of suspicion and careful correlation with symptoms.

Here we report a case of a patient who presented with inferior myocardial infarction but non-diagnostic ST changes, highlighting that ECG is frequently insufficient to diagnose acute myocardial ischemia and must be interpreted according to the clinical context.

CASE PRESENTATION

A 65-year-old woman with type 2 diabetes presented to the emergency department 36 hours after the onset of persistent, constrictive chest pain radiating to the left upper limb and jaw. Upon admission, she was fully conscious, hemodynamically stable, and showed no evidence of acute heart failure on cardiovascular examination.

Her initial electrocardiogram (ECG) (figure 1) showed sinus rhythm, first-degree atrioventricular block, and a discrete ST-segment elevation in lead DIII with reciprocal ST depression in leads DI and aVL, without fulfilling conventional STEMI criteria. No prior ECG was available for comparison. High-sensitivity troponin was markedly elevated with a significant dynamic rise. Bedside echocardiography demonstrated inferior wall hypokinesis with preserved left ventricular systolic function (LVEF 53%) and mild mitral regurgitation.

Given the high clinical suspicion for acute myocardial infarction, the patient received a 300 mg loading dose of aspirin and a 60 mg loading dose of prasugrel and was taken emergently to the cardiac catheterization laboratory. Coronary angiography (figure 2, A) revealed a proximal thrombotic occlusion of the right coronary artery and a 90% stenosis of the proximal

left anterior descending artery. Successful percutaneous coronary intervention (PCI) of the right coronary artery (figure 2, B) was performed with implantation of two drug-eluting stents, restoring TIMI 3 flow without procedural complications. The patient was transferred to the cardiac intensive care unit for monitoring and optimization of medical therapy, and was discharged home after 48 hours on guideline-directed medical therapy.

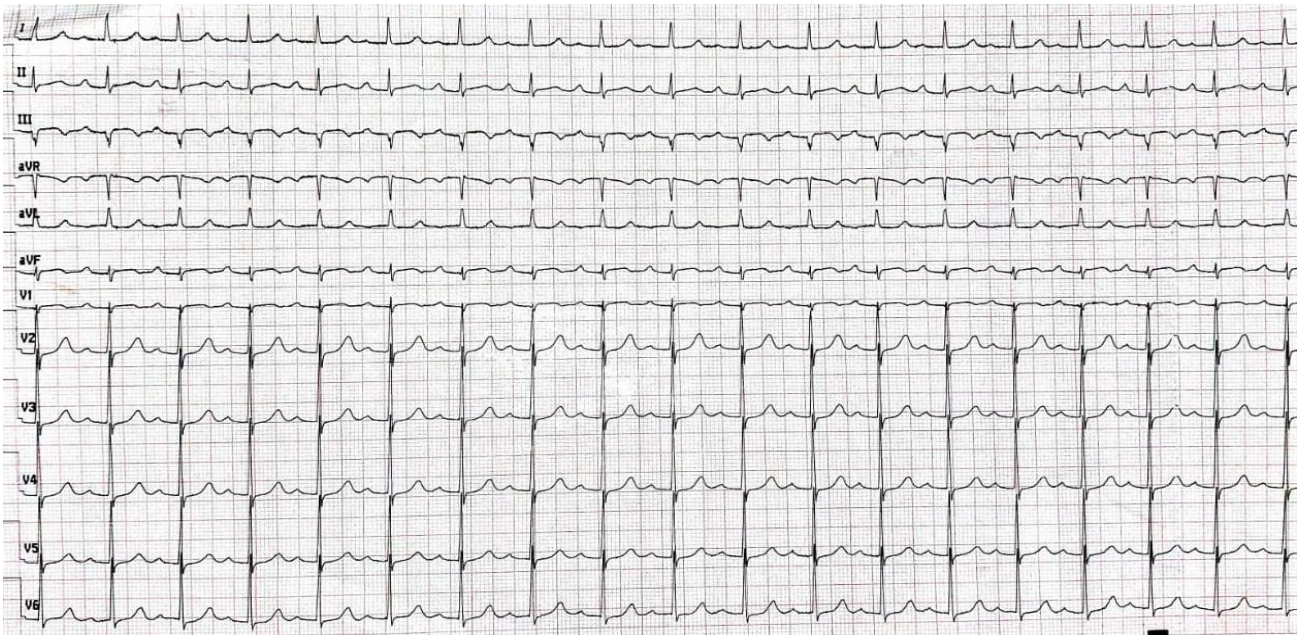

Figure 1 :Electrocardiogram shows subtle ST-segment elevation in lead DIII (Red arrow) with reciprocal subtle ST-segment depression in DI and aVL (Blue arrow), with a first-degree atrioventricular block

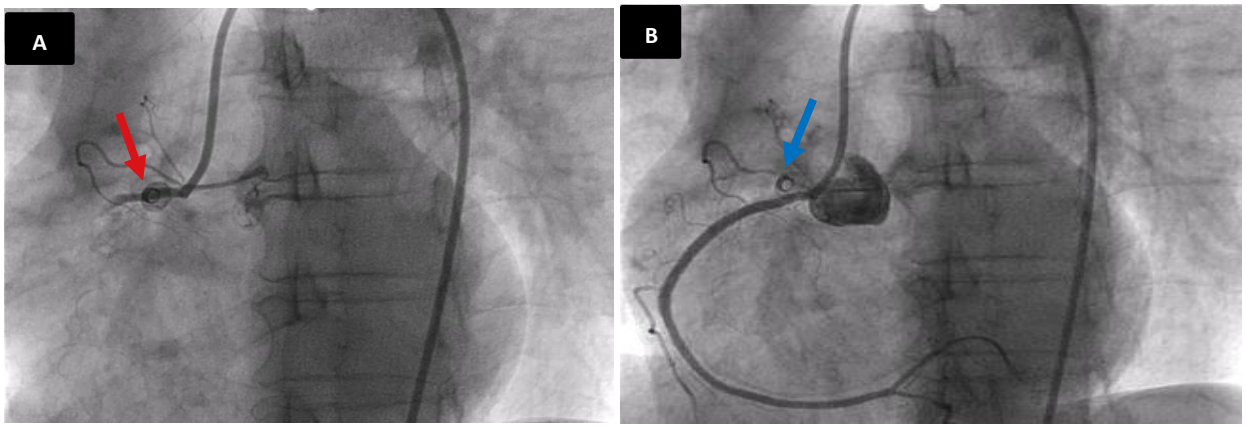

Figure 2: (A) Coronary angiography (RAO view): showing total occlusion of the proximal right coronary artery. (B) two drug eluting stent was placed with good angiographic results

DISCUSSION

The conventional paradigm for managing acute coronary artery occlusion has long relied on the presence of ST-segment elevation meeting standard STEMI criteria as the primary trigger for emergent coronary angiography. However, a growing body of evidence has demonstrated that a substantial proportion of patients with acute total or near-total coronary occlusion — now conceptualized under the term occlusion myocardial infarction (OMI) — do not fulfill these conventional electrocardiographic thresholds, with approximately 40% of OMI patients failing to meet STEMI criteria, resulting in delayed reperfusion and worse myocardial outcomes [1, 2]. The need to identify these patients promptly has driven the proposed reclassification from the STEMI/NSTEMI paradigm to the OMI/NOMI framework [3]. Our case illustrates this diagnostic challenge and underscores the importance of integrating clinical, biological, and imaging data alongside careful ECG interpretation.

The electrocardiographic findings in our patient — an isolated ST-segment elevation in lead DIII with reciprocal depression in leads DI and aVL, in the absence of ST elevation in other contiguous inferior leads — are consistent with the pattern first described by Aslanger *et al.,* in 2020 [4]. Defined by three criteria — (1) ST-

segment elevation in DIII but not in any other inferior lead, (2) ST-segment depression in leads V4–V6 with a positive or terminally positive T-wave, and (3) ST-segment elevation in V1 greater than in V2 — this pattern, now eponymously known as Aslanger's pattern, identifies a subset of inferior OMI in the context of multivessel coronary artery disease that does not meet standard STEMI criteria [4, 5]. It has been found in 6.3% of patients classified as NSTEMI, and is associated with larger infarct size, higher troponin levels, higher frequency of angiographic culprit lesions, and greater short- and long-term mortality [4, 6]. Its recognition is therefore clinically critical, as it reflects a true acute coronary occlusion requiring emergent reperfusion despite the absence of classic ST elevation across contiguous leads.

Beyond Aslanger's pattern, the broader OMI framework encompasses a spectrum of equivalent ECG signs that may indicate acute coronary occlusion without fulfilling conventional STEMI criteria. These include hyperacute T waves — recently shown to be specific for OMI even in the absence of diagnostic ST-segment elevation [7] — as well as proportional ST/QRS changes, de Winter T waves, reciprocal ST depression, and posterior OMI patterns. A comprehensive narrative review of OMI-associated ECG patterns published in Annals of Emergency Medicine in 2025 provides a detailed step-by-step approach to their recognition [6]. Recent work has further sought to formalize the diagnostic approach through dedicated scoring systems and rigorous sensitivity analyses of existing STEMI criteria, consistently highlighting their limitations [8]. Clinician familiarity with these patterns is essential for any provider involved in the triage of acute chest pain.

In this context, multimodal assessment proved decisive in our case. The marked elevation of high-sensitivity cardiac troponin (hs-cTn) with a significant dynamic rise provided strong biochemical evidence of ongoing myocardial injury. The 2023 ESC Guidelines for the management of acute coronary syndromes recommend hs-cTn as the preferred biomarker and endorse rapid 0h/1h or 0h/2h algorithms for early rule-in and rule-out of myocardial infarction [9]. Bedside echocardiography demonstrating inferior hypokinesis offered critical anatomical corroboration. The 2023 ESC guidelines further recommend emergency transthoracic echocardiography when diagnosis is unclear, while cautioning against delaying transfer for catheterization when acute coronary occlusion is suspected [9]. Together, these findings justified emergent coronary angiography despite the absence of overt STEMI criteria, ultimately confirming a proximal right coronary artery occlusion. This multimodal approach is consistent with the emerging evidence supporting a low threshold for invasive strategy when clinical and paraclinical findings are discordant with a reassuring ECG [1, 3].

Coronary angiography also identified a significant 90% stenosis of the proximal left anterior descending artery as a non-culprit lesion. Revascularization of this lesion was deferred in accordance with a staged PCI strategy. This approach is supported by the COMPLETE trial, which demonstrated that staged complete revascularization in patients with STEMI and multivessel coronary artery disease reduces the composite of cardiovascular death or new myocardial infarction at three years compared with culprit-lesion-only PCI [10]. The timing of non-culprit PCI — whether during the index hospitalization or after discharge — did not significantly affect clinical outcomes in this trial [11]. More recent data from the MULTISTARS AMI trial confirmed that immediate multivessel PCI is non-inferior to staged PCI in hemodynamically stable patients [12], while the BIOVASC trial further supported complete revascularization as a strategy in the broader ACS population [13]. Given the preserved hemodynamic stability of our patient and the clinical complexity of the acute setting, a staged approach was deemed appropriate.

CONCLUSION

This case highlights the diagnostic limitations of conventional STEMI criteria in the setting of acute coronary occlusion and reinforces the clinical relevance of the OMI paradigm. Subtle ECG patterns such as Aslanger's pattern, when interpreted in conjunction with high-sensitivity troponin kinetics and bedside echocardiography, can guide timely reperfusion decisions even in the absence of overt ST elevation. The management of non-culprit lesions, guided by current evidence from landmark trials, should be integrated into the post-PCI care plan. Clinician awareness of OMI-equivalent ECG signs remains essential to avoid treatment delays and improve myocardial salvage in this challenging subset of patients.

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