

A new electrocardiographic pattern suggestive of occlusion myocardial infarction: a report of two cases.

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Abstract

Background: Classical teaching assumes that the location of ST-segment elevation (STE) on the electrocardiogram (ECG) reflects the site of myocardial infarction (MI), and current guidelines require STE in two contiguous leads to diagnose ST-elevation MI (STEMI). This framework can miss high-risk occlusive infarctions presenting with atypical, non-contiguous STE.

Methods: We retrospectively describe two patients presenting with acute coronary syndrome whose initial 12-lead ECG displayed the Aslanger pattern: isolated STE in lead III, ST-segment depression in leads V4-V6, and STE in V1 greater than in V2.

Results: Both patients were diagnosed with a STEMI-equivalent Aslanger pattern and underwent urgent coronary angiography. The first, with known multivessel disease, had critical left anterior descending (LAD) artery stenosis; the second presented in cardiogenic shock with an occlusive thrombus in the right coronary artery. Both were successfully treated with percutaneous coronary intervention.

Conclusion: The Aslanger pattern is a high-risk STEMI-equivalent that can be missed under conventional STE criteria. Our cases, including an atypical LAD culprit lesion, support incorporating this pattern into routine ECG interpretation and emergency training to enable timely reperfusion.

Keywords: Aslanger pattern; STEMI-equivalent; acute coronary occlusion; electrocardiogram.

INTRODUCTION

For more than a century, the electrocardiogram (ECG) has been the most accessible clinical tool for diagnosing and localizing acute myocardial infarction (MI). Classical teaching assumes the infarct lies beneath the leads showing ST-segment elevation (STE), and international guidelines accordingly require STE in two contiguous leads of the same localization group (anterior, lateral, or inferior) to diagnose and localize STEMI (1). However, this framework is more intuitive than evidence-based, and recent studies show that the leads demonstrating STE do not reliably indicate the location of the infarcted myocardium (2,3). Instinctively assigning lead groups to specific infarct locations may misclassify MI location (4) and often requires

unsupported explanations, such as a single infarct-related artery supplying two coronary territories (5), to account for atypical STE patterns.

Moreover, this requirement reduces STEMI sensitivity in certain configurations (6,7) and may obscure novel ECG patterns that nonetheless signal acute coronary occlusion requiring emergent reperfusion.

We report two cases presenting with acute coronary syndrome whose initial 12-lead electrocardiogram (ECG) met the diagnostic triad described by Aslanger et al. (8): isolated ST-segment elevation (STE) in lead III, ST-segment depression in leads V4-V6, and STE in V1 greater than in V2. Clinical presentation, ECG tracings, laboratory results, and coronary

angiography findings were extracted from the patients' medical records. Informed consent was obtained for the anonymized publication of clinical data and images.

CASE PRESENTATION

Case 1: A 71-year-old man with a history of hypertension, heart failure, and a prior NSTEMI two months earlier due to critical triple-vessel disease (including distal left main stenosis) for which he had declined percutaneous coronary intervention (PCI), presented to the emergency department with rest angina and acute dyspnoea. On admission, the patient was assessed following the ABCDE approach. A (Airway): patent, able to speak in short sentences. B (Breathing): acute respiratory distress with bilateral pulmonary crackles; respiratory rate 20 breaths/min) and SpO₂ 96% on room air. C (Circulation): markedly hypertensive (220/110 mmHg) and tachycardic (110 bpm), with clinical signs of acute pulmonary oedema. D (Disability): Glasgow Coma Scale 15/15; capillary glucose 1 g/L, no focal neurological deficit. E (Exposure): temperature 37 °C; no other relevant findings on complete exposure.

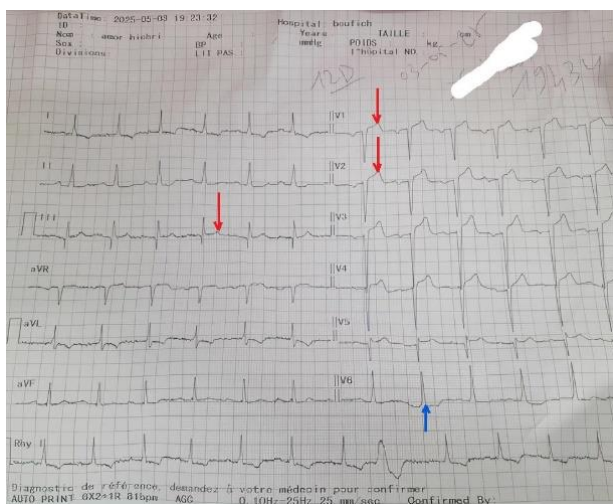


Figure 1: The first case's prehospital ECG.

↓ ST-segment elevation in lead III, V1, and V2 (V1 > V2).
↑ ST-segment depression at the J point in lead V6.

The initial ECG demonstrated an isolated ST-segment elevation in lead III, ST-segment

elevation in V1 greater than that in V2, and ST-segment depression in leads V4-V6, which is a triad characteristic of the Aslanger pattern. The high-sensitivity troponin I level was elevated to 2000 ng/L. Chest radiography confirmed pulmonary oedema and cardiomegaly (Figure 1). The patient was diagnosed with ST-elevation myocardial infarction (STEMI) equivalent due to the Aslanger pattern. The patient was stabilized with non-invasive ventilation, intravenous diuretics, and nitrates. Subsequent coronary angiography confirmed severe multivessel disease, including critical distal left main and proximal left anterior descending (LAD) artery stenoses. The patient successfully underwent PCI with drug-eluting stent implantation into the LAD (Figure 2).

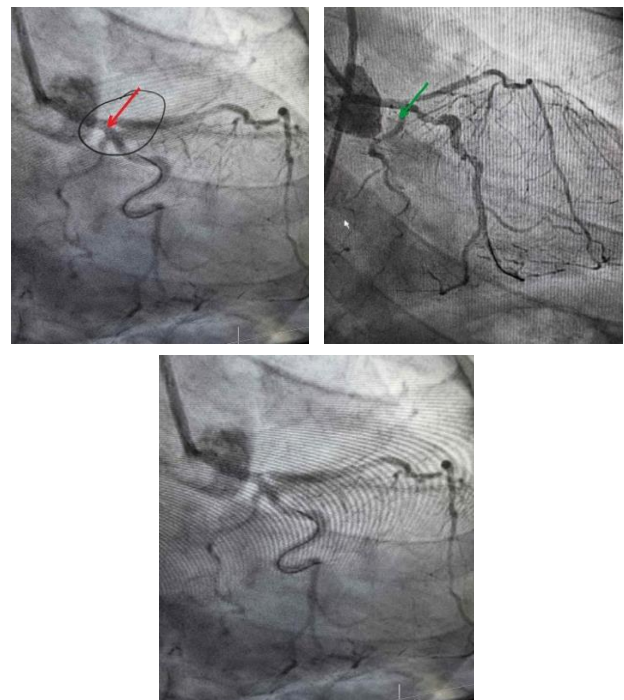


Figure 2: Coronary Angiography of the Left Anterior Descending Artery

↙ site of the acute LAD occlusion (pre-PCI). ↗ restored antegrade flow after PCI, showing resolution of the occlusion.

Antithrombotic loading doses: aspirin 300 mg, clopidogrel 600 mg, and anticoagulation with unfractionated heparin (70–100 IU/kg IV bolus) were administered. Reperfusion timing: door-to-ECG 10 min and door-to-reperfusion (door-to-

balloon) 40 min. Clinical course and outcome: short-term evolution was favourable with haemodynamic and respiratory stabilization; the patient was discharged on day 5 in stable condition.

Case 2: A 62-year-old woman with a history of heart failure and chronic respiratory disease was admitted to the primary emergency department with severe respiratory distress. On admission, she was assessed following the ABCDE approach. A (Airway): patent. B (Breathing): severe respiratory distress with tachypnoea (30 breaths/min), critical hypoxaemia (SpO₂ 67% on room air) and bilateral pulmonary crackles. C (Circulation): hypotension (BP 90/50 mmHg) and tachycardia (HR 110 bpm) with peripheral oedema, consistent with cardiogenic shock. D (Disability): Glasgow Coma Scale 14/15; capillary glucose 0.9 g/L. E (Exposure): temperature 37 °C; no other relevant findings. The initial ECG showed ST-segment elevation in leads III and V1 with complete right bundle branch block (Figure 3).

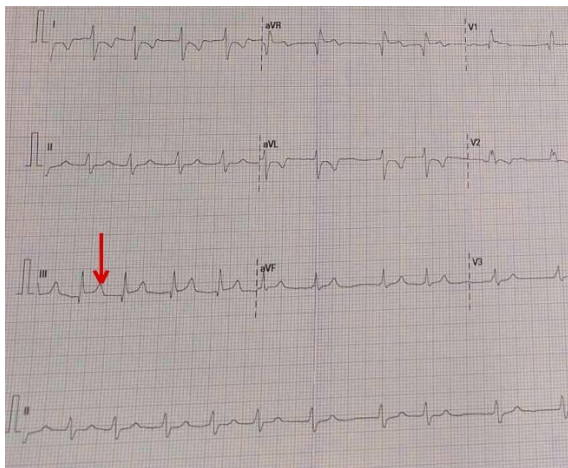


Figure 3: The second case's prehospital ECG.

The patient was managed for cardiogenic shock secondary to suspected acute coronary occlusion. She was placed in a semi-recumbent position and started on Continuous Positive Airway Pressure (CPAP) for respiratory support. The patient was stabilized and transferred to a tertiary hospital for further critical care and urgent coronary

angiography. Haemodynamic management: Given the cardiogenic shock, continuous intravenous noradrenaline (4 mg/h) combined with dobutamine (5 µg/kg/min) was initiated and maintained for 72 h, with dose adjustments according to haemodynamic response and end-organ perfusion; Antithrombotic loading doses: aspirin 300 mg, clopidogrel 600 mg and anticoagulation with unfractionated heparin (70–100 IU/kg IV bolus). Reperfusion timing: door-to-ECG 8 min and door-to-reperfusion 60 min. Clinical course and outcome: after PCI with stent implantation in the right coronary artery, the short-term evolution was favourable, and the patient was discharged on day 6.

This case underscores the significance of the Aslanger pattern as a critical STEMI-equivalent that can manifest with profound haemodynamic instability. Its recognition is essential for triggering urgent reperfusion strategies. Subsequent coronary angiography confirmed the diagnosis, revealing an occlusive thrombus in the right coronary artery, which was successfully treated with stent implantation. This case highlights that this pattern, despite its subtlety, signifies a high-risk occlusive myocardial infarction that can be misdiagnosed because of the absence of widespread ST-segment elevation.

DISCUSSION

The findings from our case series and the original study by Aslanger et al. collectively challenge the long-standing paradigm of electrocardiographic interpretation of acute myocardial infarction. For decades, the diagnosis of STEMI has been predicated on an intuitive yet incompletely evidence-based framework that requires ST-segment elevation (STE) in two contiguous leads, with the infarct location assumed to reside beneath those leads (3,8). However, recent evidence has confirmed that the location of STE does not reliably indicate the location of the infarcted myocardium, and rigid adherence to contiguous lead criteria can result in the

misclassification of MI location and a failure to recognize high-risk occlusive patterns (3,4). The Aslanger pattern serves as a critical example of this limitation. This demonstrates that STE in a single inferior lead (DIII) can possess profound clinical value, signifying an acute atherothrombotic event that frequently results in an occlusive MI (8). This atypical pattern, characterized by non-contiguous STE, arises when the frontal ST vector is oriented towards the edges of the standard 12-lead coverage. The underlying pathophysiology is theorized to be a summation of vectors: the ST vector of the inferior MI, directed inferiorly and rightwards, combined with the ST vector of concomitant subendocardial ischaemia from critical multivessel disease, which is characteristically directed towards lead aVR. The resultant average ST vector is directed rightward, projecting into the positive poles of leads III and aVR, while projecting into the negative poles of leads I and II, and appearing nearly isoelectric in aVF (8). This elegant vectorial explanation accounts for the full ECG picture: STE in III and aVR, ST depression in I and II, and ST depression in the lateral chest leads (V4-V6) as the vector points away from them.

This brings us to a pivotal question raised by the subsequent commentary: Should STE in aVR be formally incorporated into the pattern's definition? (9) While the original diagnostic triad (STE in DIII, STD in V4-V6, STE in V1 > V2) is highly specific, the pathophysiological model and example tracings consistently show accompanying STE in aVR. Therefore, while the triad remains the diagnostic cornerstone, the presence of STE in aVR should be recognized as a common and supportive feature that reinforces the vectorial diagnosis and may aid in the pattern recognition.

Clinically, the implications of missing this pattern are significant. The data of Aslanger et al. indicate that 13.3% of inferior MIs may present with this pattern and be incorrectly labelled as

NSTEMI, thereby depriving them of emergent revascularization therapy (8). These patients are not low-risk; they have a high frequency of multivessel disease, often involving the circumflex artery, and exhibit increased short- and long-term mortality risk comparable to patients with classical inferior STEMI (8). Therefore, an immediate invasive strategy is crucial to decrease morbidity and mortality in this patient group (8,9). Therefore, the Aslanger pattern is a high-risk STEMI-equivalent that underscores the limitations of the traditional ECG interpretation. Its recognition is paramount for triggering urgent reperfusion therapy. By educating clinicians on this pattern and its vectorial basis, rapid diagnosis in emergency settings can be improved, ultimately reducing major adverse cardiovascular events through appropriate and timely treatment. In contrast to the literature, which most frequently implicates the right coronary or circumflex arteries, our first case uniquely identified the left anterior descending (LAD) artery as the culprit vessel. This finding challenges the conventional anatomical association of this pattern and opens the door for a new discussion regarding its potential variability.

Our two cases directly illustrate these principles. In Case 1, recognition of the Aslanger pattern prompted urgent angiography that revealed critical distal left main and proximal LAD disease, enabling life-saving PCI in a patient who had previously declined revascularization, and showing that the culprit vessel is not invariably the RCA or circumflex. In Case 2, the same pattern identified an occlusive RCA thrombus in a patient presenting in cardiogenic shock, underscoring that the pattern can herald haemodynamic collapse and must trigger immediate reperfusion. Together, the cases support our central message that the Aslanger pattern should be managed as a STEMI-equivalent regardless of the culprit artery or the initial haemodynamic status.

The LAD culprit lesion observed in Case 1 warrants a mechanistic explanation. The Aslanger pattern reflects the summation of an inferior injury vector with a vector of concomitant subendocardial ischaemia from multivessel disease. In our patient, several non-exclusive mechanisms may account for an LAD culprit producing this pattern: (i) a long, wrap-around (type III) LAD supplying the inferior wall, which can generate inferior ST changes when occluded distally (5); (ii) proximal LAD stenosis superimposed on critical distal left main and triple-vessel disease, so that diffuse subendocardial ischaemia orients the average ST vector rightward toward leads III and aVR (8); and (iii) mid-anterior or diagonal involvement, known to produce ST-elevation in non-contiguous leads (6,7).

CONCLUSION

The Aslanger pattern is a significant advance in the electrocardiographic diagnosis of acute MI, demonstrating that high-risk occlusive coronary disease can present without the classic requirement of contiguous ST-segment elevation. Its hallmark triad—STE in lead III, STD in V4-V6, and STE greater in V1 than V2—identifies acute inferior MI, almost always in the setting of multivessel disease and concomitant critical stenosis. Our case series confirms the high clinical stakes of this pattern, with infarct sizes and mortality rates rivalling classical inferior STEMI, while the LAD culprit lesion in our first case suggests its underlying anatomy may be more variable than initially described. Failure to recognize this pattern risks misclassification as NSTEMI and delayed revascularization.

We therefore advocate integrating the Aslanger pattern into emergency medicine and cardiology training, and encourage future guidelines to formally adopt it as a STEMI-equivalent to ensure high-risk patients receive timely reperfusion.

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