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CASE REPORT

Aslanger Pattern: An unusual electrocardiogram in Acute pulmonary Embolism

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Introduction: Pulmonary embolism (PE) is a critical condition that can present with diverse and often non-specific electrocardiographic findings. The Aslanger pattern, identified in 2020, is an uncommon electrocardiography (ECG) finding typically associated with acute non-ST-segment elevation myocardial infarction (NSTEMI). Although this pattern is usually linked to myocardial infarction with significant coronary stenosis, we report a rare case where the Aslanger pattern was observed in a patient with acute pulmonary embolism.

Case Presentation: A 52-year-old male with Parkinson's disease presented with sudden chest pain, shortness of breath, sweating, and fatigue. Initial ECG showed Aslanger pattern, suggesting acute coronary syndrome. Despite elevated troponin I (1624 ng/mL) and normal coronary angiography, further investigation revealed right ventricular dilation and massive pulmonary embolism confirmed by CT angiography. The patient was treated with thrombolytics and anticoagulation, stabilizing over five days and discharged on rivaroxaban.

Conclusion: The Aslanger pattern usually linked to myocardial infarction can also occur in acute pulmonary embolism. This case underscores the need for careful differential diagnosis and timely treatment to prevent complications associated with delayed care.

Keywords: pulmonary embolism, Aslanger pattern, electrocardiogram

Background

Pulmonary embolism (PE) is a major cause of morbidity and mortality worldwide.[1] In PE, electrocardiographic results are frequently variable and nonspecific. After acute myocardial infarction and stroke, acute pulmonary embolism (APE) ranks as the third leading cause of cardiovascular mortality.[2] In pulmonary embolism, electrocardiogram may show a variety of morphologic and/or rhythm abnormalities or it may be normal.[3]

Electrocardiography (ECG) is an essential diagnostic tool for assessing patients experiencing chest pain or dyspnea. The presence of ST-elevation, a critical and potentially life-threatening change on the ECG, often prompts physicians to consider primary percutaneous coronary intervention or thrombolysis. However, several conditions beyond ST-elevation myocardial infarction (STEMI) can also cause ST-elevation on an ECG, including Prinzmetal angina, Takotsubo cardiomyopathy, Brugada syndrome, left ventricular aneurysm, hypothermia, hyperkalemia, and acute pericarditis.[4]

After analyzing 1000 electrograms (ECG) of acute non-ST-segment elevation myocardial infarction (NSTEMI), Aslanger et al presented Aslanger's pattern[1,2]] in April 2020. The following elements are present in this ECG pattern:[1] isolated ST-segment elevation in lead III and no ST-segment elevation in the other inferior leads[2] Lead V4–V6 had depression in the ST segment while having a positive T wave and terminal vector[5] Lead V1 had higher ST segment elevation than Lead V2. Aslanger's pattern typically indicates a higher risk of acute inferior myocardial

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infarction (AMI) in patients with concurrent significant stenoses in other coronary arteries, resulting in a larger infarct size and increased mortality.[4,6] Here, we present an unusual case of pulmonary embolism presented with the Aslanger pattern ECG.

Case Presentation

A 52-year-old male with a history of Parkinson's disease, treated with Levodopa and Carbidopa, presented to the emergency department with sudden onset chest pain, shortness of breath, sweating, and fatigue. He had no history of hypertension, diabetes, smoking, alcohol, or coronary artery disease. There have been no reports of drug or food allergies or previous surgery.

The patient experienced severe distress upon arrival. His heart rate was 150 beats per minute with atrial fibrillation and a rapid ventricular response. He was tachypneic with a respiratory rate of 25 breaths per minute, oxygen saturation of 80%, and blood pressure of 62/50 mmHg, consistent with shock. Despite these alarming vital signs, physical examination of heart, lungs, and abdomen did not reveal any obvious abnormalities.

Owing to hemodynamic instability, immediate cardioversion was performed at 100 joules, followed by 150 joules, which successfully stabilized his heart rhythm. The patient was intubated for airway protection, vasopressors and intravenous fluids were administered.

Subsequently, the electrocardiogram (ECG) revealed isolated ST elevation in lead III, ST depression in leads V4-V6 with terminal T-wave inversion, and ST elevation in lead V1 greater than in V2. This pattern was consistent with the; Aslanger pattern and initially raised concerns of acute coronary syndrome (Figure 1). Coronary angiography was decided first because the ECG suggested acute coronary syndrome (ACS), which is life-threatening and requires immediate intervention.

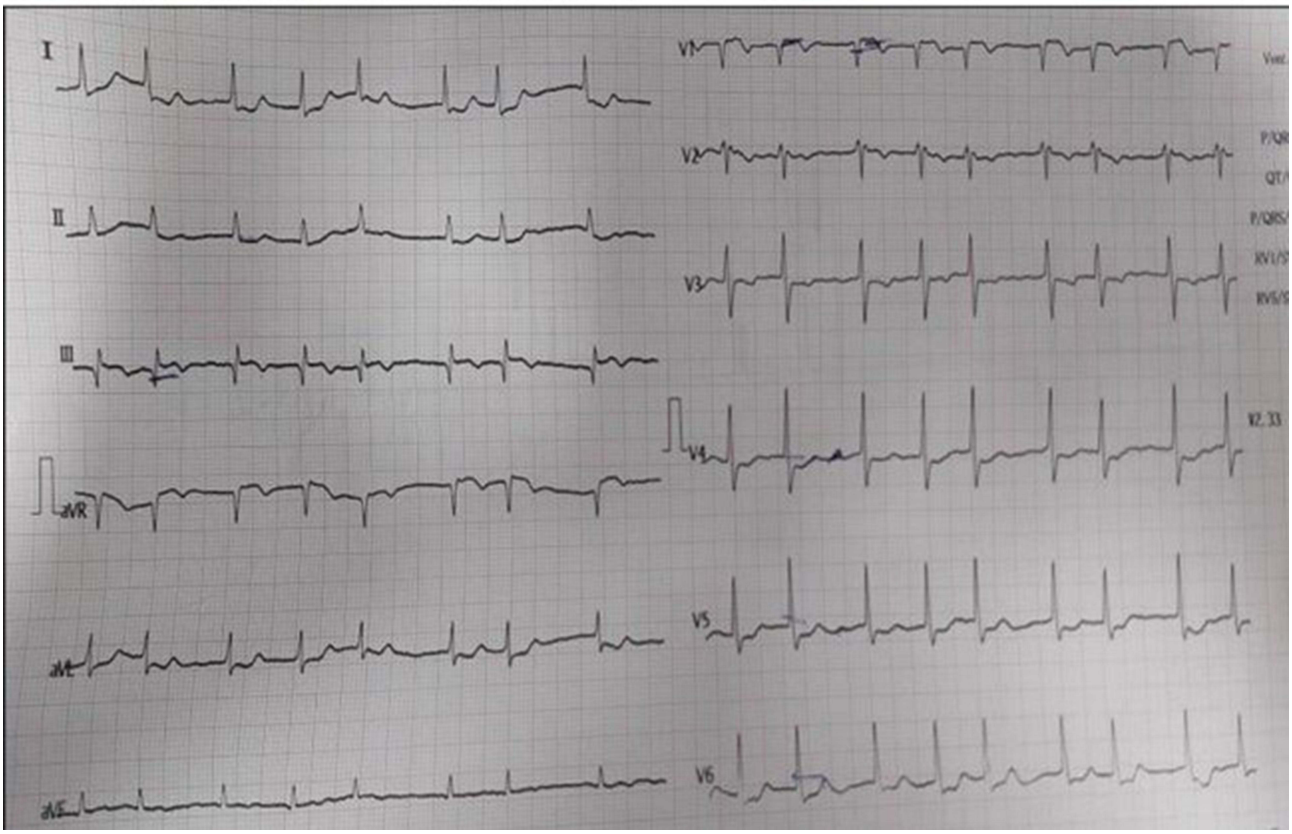

Figure 1 The ECG shows isolated ST elevation in lead III, ST depressions in leads V4-V6, lead I and AVL. This pattern is consistent with Aslanger pattern.

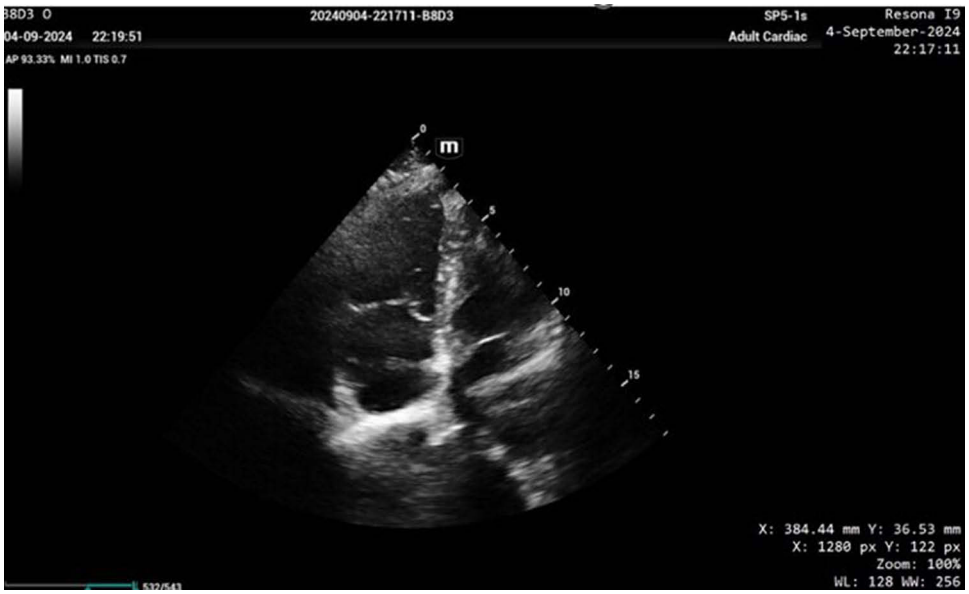

Figure 2 Apical four chamber view showing dilated right chambers and McConnell`s sign.

The patient was promptly taken to the catheterization laboratory for coronary angiography. However, the coronary arteries were normal, ruling out acute coronary syndrome (Figure 2). Despite this, laboratory tests revealed a significantly elevated troponin I level of 1624 ng/mL (normal range: 0.02–0.06 ng/mL), complicating the initial diagnosis.

Further diagnostic workup included a transthoracic echocardiogram, which demonstrated a left ventricular ejection fraction (LVEF) of 63%, no segmental wall motion abnormalities but massive right atrial and ventricular dilation with reduced right ventricular function (TAPSE: 11 mm, RV S': 8 cm/s) (Figure 3). These findings suggested right heart strain, leading to the decision to perform pulmonary CT. This revealed a thrombus in the pulmonary arteries, confirming the diagnosis of a pulmonary embolism (Figure 4A and B).

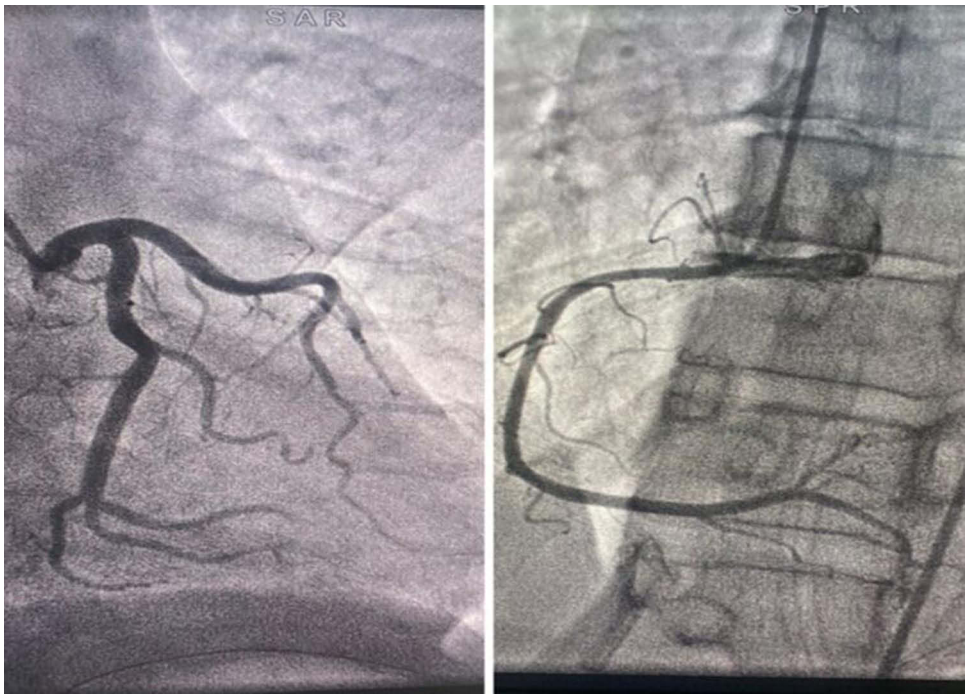

Figure 3 Showing normal coronary arteries Left (LMCA, LAD and LCX) and the right coronary artery (Orange arrow).
Abbreviations: LMCA, Left main coronary artery (black arrow); LAD, left anterior descending artery (blue arrow); LCX, left circumflex artery (red arrow).

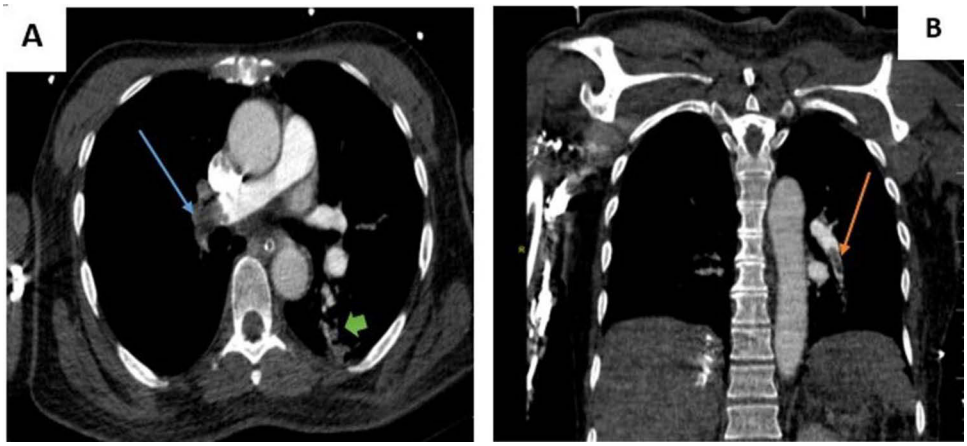

Figure 4 Axial & Coronal Angio CT scan: (**A**) Filling defect in the right main pulmonary artery (Blue arrow) (**B**) Filling defect in the left lower segmental arteries of pulmonary artery (Orange arrow) with associated peripheral wedge-shaped consolidation areas representing infract (Green head arrow).

The patient was transferred to the coronary care unit, where thrombolytic therapy was administered owing to unstable hemodynamics. He also received supportive care including vasopressors and anticoagulation with subcutaneous heparin. After five days, his condition stabilized, and he was transferred to the inpatient ward. After an additional three days of observation, the patient was discharged on rivaroxaban for ongoing anticoagulation.

At two-week follow-up, the patient was asymptomatic, with no recurrence of chest pain or shortness of breath. Blood tests revealed total cholesterol of 3.16 mmol/L (Normal: <5.2 mmol/L), LDL-C of 1.41 mmol/L (Optimal: <2.6 mmol/L) and NT-proBNP of 45.0 pg/mL (Normal: <125 pg/mL for age <75). A follow-up echocardiogram showed improved cardiac function with a left ventricular ejection fraction of 71%, left ventricular end-diastolic diameter of 44 mm, and mild residual dilation of the right atrium. Right ventricular function normalized. Repeat ECG showed resolution of ST elevation in leads III and V1, with an upright T-wave and return to isoelectric ST segments.

Discussion

In pulmonary embolism (PE), although advanced imaging modalities are widely used for definitive diagnosis of PE, ECG remains a valuable tool to raise clinical suspicion. Common ECG findings in PE include sinus tachycardia, transient right bundle branch block (RBBB), nonspecific ST-T wave changes, characteristic S1Q3T3 pattern, T wave inversions in precordial leads, and rightward shift of the QRS axis. Although these findings are nonspecific, they support the suspicion of PE. Distinguishing between myocardial infarction and pulmonary embolism (PE) can be challenging due to their similar initial symptoms. PE may also show ST-segment elevations, highlighting the need to consider other conditions that can resemble STEMI.[7] Electrocardiographic alterations that resemble a myocardial infarction can be seen after a significant pulmonary embolism, presenting a diagnostic challenge for clinicians working in emergency medicine and intensive care.[8]

However, the appearance of the Aslanger pattern in PE is particularly noteworthy, as it has not been previously reported association with PE. As an ischemic ECG pattern, the Aslanger pattern typically prompts clinicians to investigate myocardial ischemia rather than PE. This diagnostic challenge highlights the importance of recognizing that ischemic ECG changes may also occur in PE, despite their usual association with cardiac ischemia. Pulmonary CTA is the most widely utilized technique for diagnosing pulmonary embolism. While it is not a definitive test for PE, ultrasound can reveal echocardiographic changes like right ventricular dilation or systolic dysfunction, which are crucial for diagnosis, differential diagnosis, and risk assessment.[9]

Published reports have shown that arteries associated with ischemia can differ from case to case. Zhang et al described an elderly patient who exhibited Aslanger pattern and significant stenosis in the distal left main (LM) and left anterior descending (LAD) arteries. Furthermore, near-total occlusion of the proximal right coronary artery (RCA), with thrombosis visible on coronary angiography.[10]

Our patient presented with acute chest pain and an ECG showing Aslanger pattern, which initially suggested a new myocardial infarction. However, coronary angiography revealed normal coronary arteries. Further investigation confirmed the patient's primary diagnosis of acute pulmonary embolism. Similar to this case, Kasmani et al reported a case where a new left bundle branch block was observed as an unusual ECG finding in pulmonary embolism.[11] In PE, electrocardiographic results are frequently variable and nonspecific. An electrocardiogram (ECG) may show a variety of morphologic and/or rhythm abnormalities or it may be normal.[3] The Aslanger pattern, while useful for identifying acute obstructive myocardial infarction and guiding timely revascularization, should also prompt consideration of pulmonary embolism if myocardial infarction is ruled out.

This novel ECG pattern, the Aslanger, indicates potential acute myocardial infarction, particularly with ST segment elevation in lead III and associated changes in other leads. It is critical for guiding urgent cardiac intervention in patients with acute chest pain or hemodynamic instability. Given the potential overlap in ECG findings between these conditions, it is crucial to thoroughly evaluate pulmonary embolism when primary suspicion of myocardial infarction is excluded. This approach ensures that both conditions are appropriately addressed and managed.

Conclusion

We present a case of acute pulmonary embolism in which the ECG displayed Aslanger pattern characterized by isolated ST-segment elevation in lead III, ST-segment depression in leads V4-V6 with a positive T wave/terminal vector, and greater ST-segment elevation in lead V1 than lead V2. The diagnosis was confirmed by pulmonary CT angiography. In clinical practice, particularly in emergency settings, it is crucial to urgently evaluate patients with an ECG of Aslanger pattern. Prompt treatment is essential, and emergency coronary angiography should be considered along with evaluation for pulmonary embolism to prevent adverse outcomes resulting from delayed treatment.

Ethics Approval

Based on the regulations of the review board of Mogadishu Somali Türkiye Training and Research Hospital, institutional review board approval is not required for case reports.

Consent for Publication

Written informed consent was obtained from the patient's daughter for publication of this case report and accompanying images.

Author Contributions

All authors made a significant contribution to the work reported, whether in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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