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CASE REPORT

Small Bowel Perforated Viscus Mimicking Inferior Wall Myocardial Infarction: A Case Report

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Abstract

Acute coronary syndrome (ACS) is a serious medical condition caused by cardiac muscle ischemia, which can lead to myocardial injury or necrosis. EKG is a critical diagnostic tool for patients with suspected ACS, as it can determine the vascular territory and guide therapy. However, there are various non-cardiac causes of EKG changes that mimic ACS and can lead to misdiagnosis and inappropriate management. We present the case of a 57-year-old lady with an extensive past medical history of coronary artery disease (CAD) who presented to our institution unresponsive and pulseless secondary to a perforated viscus and was found to have ST-segment elevations in inferior leads on the EKG.

Keywords: Perforated viscus, Pneumoperitoneum, Acute coronary syndrome

1. Introduction

Acute coronary syndrome is a medical condition caused by cardiac muscle ischemia that leads to myocardial injury or necrosis. It is considered a medical emergency that requires prompt diagnosis and management. EKG is a critical tool for diagnosing patients with suspected ACS, as it can determine the vascular territory and guide therapy. However, EKG changes can also be caused by various non-cardiac conditions that mimic ACS, including gastrointestinal, cerebrovascular, and pulmonary pathologies, among others. These conditions can lead to misdiagnosis, delayed management, and potential harm to patients. Therefore, it is crucial for clinicians to be aware of non-cardiac causes of EKG changes and to differentiate them from ACS to ensure timely and accurate diagnosis and appropriate management.[1]

2. Case presentation

The patient is a 57-year-old lady with a past medical history of (coronary artery disease) CAD status post multiple stents placement including left circumflex artery, left anterior descending artery (LAD), and right coronary artery (RCA), dyslipidemia, End-Stage Renal Disease (ESRD) on hemodialysis who was brought by her family to our Emergency Department (ED) unresponsive and pulseless. Before arrival at our ED, the patient was hospitalized in another institution for one week with complaints of abdominal pain, however, she decided to sign and leave against medical advice on Friday. Over the weekend, her symptoms worsened, and she had persistent abdominal pain and shortness of breath which prompted her to seek medical attention the following Monday. According to the patient's family, she became unresponsive and collapsed while they were driving her in a particular vehicle before arrival at the hospital. As per the patient's relatives, she was unresponsive for at least 5 min before she arrived at our ED.

In our ED, the patient was found unresponsive and pulseless, and ACLS protocol was initiated, the patient was intubated. The initial rhythm was (pulseless electrical activity) PEA and after 17 min, a return of spontaneous circulation was achieved. She was persistently hypotensive with a blood pressure of 75/55 mmHg and tachycardic with a heart rate of

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110. A temperature of 96 °F. Pertinent labs included WBC 6.1 [4.8–10.8 K/UL], lactic acid was 12.7 [0.5–2.2 MMOL/L], BNP 624 [<100 pg/ml], AST 317 [15–41 U/L], ALT 135 [14–54 U/L], creatinine was 3.76 [0.4–1 MG/DL], HCO3 14 [22–32 MMOL/L], and AGAP 21 [3–9 MMOL/L]. ABG revealed a pH of 7.2 [7.35–7.45], pCO2 of 37 [35–45 mmHg], and HCO3 15 [22–26 mmol/L]. Initial troponin was 0.22 [<0.5 ng/ml], and follow-up troponin was 0.30 [<0.5 ng/ml]. Initial EKG showed sinus tachycardia with a heart rate of 117 beats per minute, voltage criteria for left ventricular hypertrophy, and an acute inferior injury pattern with ST-segment elevation in lead II, III, and aVF (Fig. 1). CXR showed pneumoperitoneum (Fig. 2), and general surgery was consulted immediately. She was started on broad-spectrum antibiotics with vancomycin and piperacillin-tazobactam along with norepinephrine for blood pressure support. The patient was taken emergently to the operative room for exploratory laparotomy. According to the operative report, upon entering the abdominal cavity there was free air as well as foul-smelling succus entericus. A perforation in the mid to distal ileus was evidenced for which she underwent ileal resection, with an end-to-end anastomosis along with a gastrostomy tube placement.

The patient was transferred to the ICU for post-operative care intubated, sedated, and on continuous vasopressor support. Follow-up EKGs showed resolution of the ST-segment elevation in the inferior leads (Fig. 3). Due to the extensive cardiac history, cardiology was consulted and ordered a 2D-echo which showed an ejection fraction of 50–55%, and moderate left ventricular hypertrophy without any wall motion abnormalities. At that time, since the EKG changes resolved after the surgical intervention, recommendations for medical management including daily aspirin were given. The

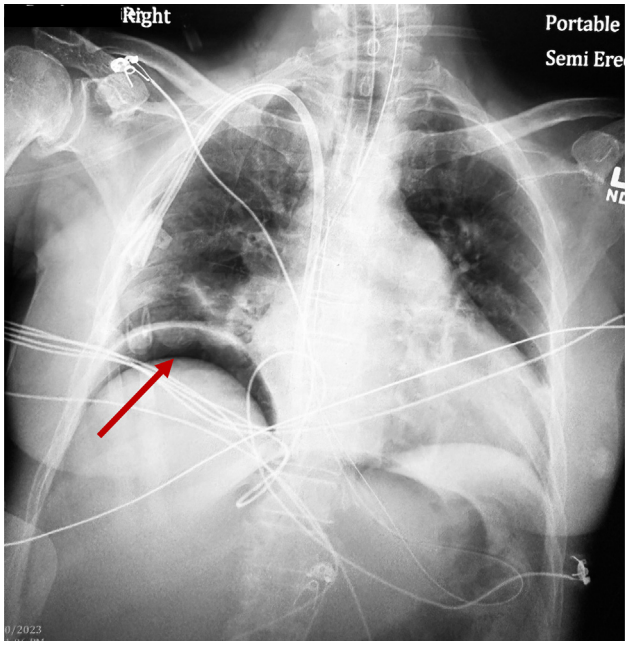

Fig. 2. CXR shows free air under the diaphragm.

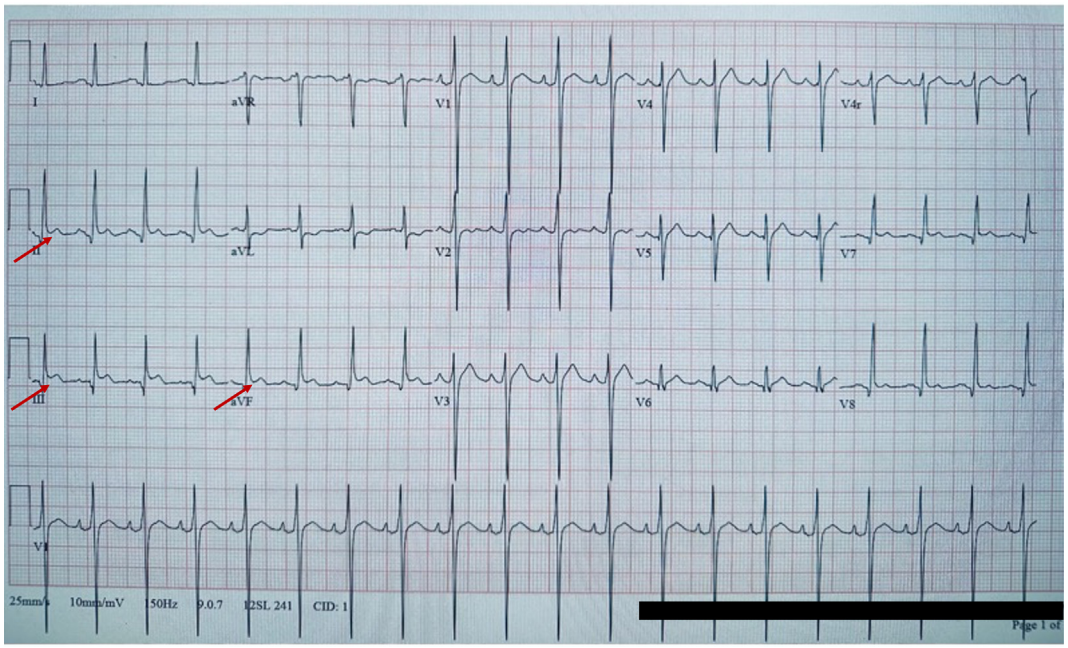

Fig. 1. EKG showing ST-segment elevation in lead II, III, and aVF.

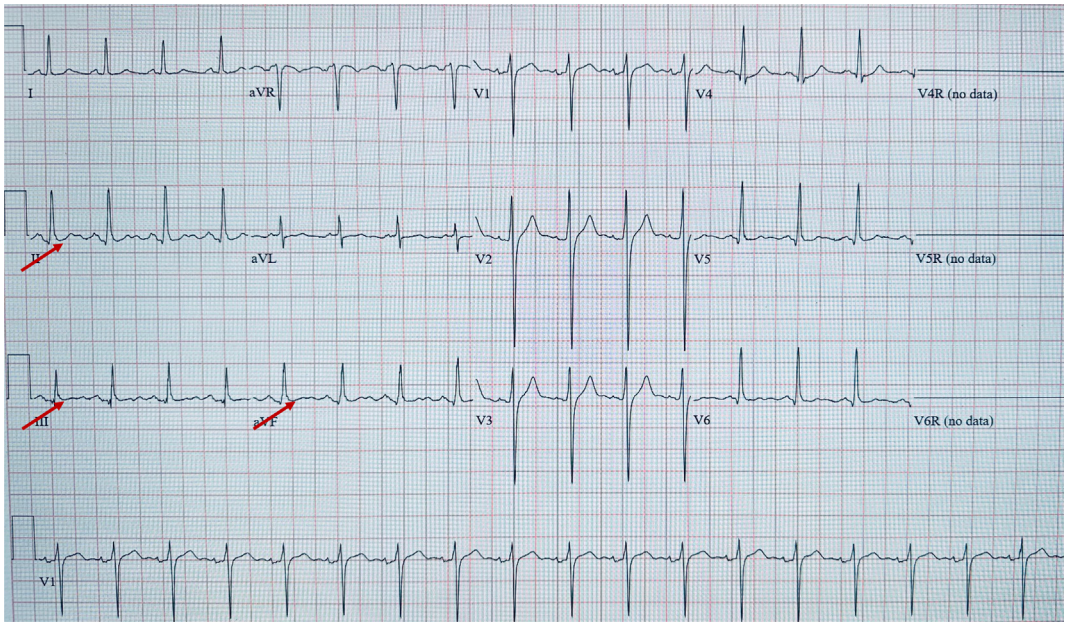

Fig. 3. Repeat EKG shows the resolution of the ST-segment elevation in the inferior leads.

infectious disease specialist was consulted and recommended discontinuing piperacillin-tazobactam to start the patient on meropenem and continue with vancomycin with hemodialysis sessions. Blood culture showed *Klebsiella pneumoniae*.

The patient remained hypotensive despite norepinephrine, needing up to three pressors including vasopressin and neosynephrine. Her abdomen became distended and tense, and she was persistently acidotic with a pH as low as 6.87 [7.35–7.45], pCO2 32 [35–45 mmHg], and HCO3 4.6, despite hemodialysis sessions and sodium bicarbonate administration. Also, her lactic acid initially improved after the surgical intervention to 7.8 [0.5–2.2 MMOL/L], however, it rapidly increased with reported values as high as 25.4 [0.5–2.2 MMOL/L]. Bladder pressure was measured which ruled out intraabdominal compartment syndrome. In the light of the whole clinical picture, a concern for bowel ischemia and/or compartment syndrome was raised, however, she was deemed to be a poor surgical candidate for any intervention as per the surgical team. Unfortunately, she went into pulseless electrical activity and despite the full ACLS protocol being implemented, she was pronounced on her third day of hospitalization.

3. Discussion

ACS results from ischemia of the cardiac muscles and is a medical emergency. Early diagnosis using patient history, EKG, and cardiac biomarkers is critical. Although various diagnostic tests are available, EKG remains the primary tool for initial diagnosis and determination of vascular territory in patients with suspected ACS. However, EKG changes can also be caused by other cardiac conditions (myocarditis, pericarditis, ventricular hypertrophy, cardiomyopathies, and neoplastic invasion of the myocardium) and noncardiac causes such as GI-related (acute cholecystitis, acute pancreatitis, gastric or esophageal perforation, hiatal hernia), cerebrovascular events (aneurysmal subarachnoid hemorrhage, acute stroke, traumatic brain injury), and pathologies involving the pulmonary system (acute community-acquired lobar pneumonia, acute pulmonary embolism, lung cancer, spontaneous pneumothorax), among others.[1]

Furthermore, changes in the position of the heart and diaphragm can also result in EKG changes, as seen in restrictive and obstructive lung diseases. This can lead to alterations in the P-axis orientation or changes in voltage due to a shift in the mean QRS axis. Increased vagal tone due to the visceral-cardiac reflex secondary to gastric distention can also cause EKG changes, such as symmetrical T-wave inversions in patients with biliary pathology. Also, stress-related cardiomyopathy or variant angina may be the cause of EKG changes.[1]

Intan RE et al. describe the case of a 70-year-old individual presenting with an acute abdomen and pneumoperitoneum consequent to gastric perforation, who had EKG changes resembling anteroseptal ST-segment elevation myocardial infarction.[2] Also, Vutthikraivit reported the case of a 78-year-old man who presented to the ED

complaining of severe epigastric pain and was found to have a perforated gastric ulcer along with ST-segment elevations in leads V2 and V3. Similar to our case, the EKG modifications were resolved after the individual underwent surgical intervention in both cases.[3] Furthermore, small bowel obstruction has been linked to EKG changes mimicking acute ST-segment elevation myocardial infarction, as reported by Patel et al., who illustrated the case of a 42-year-old female who exhibited recurrent small bowel obstruction and displayed ST-segment elevations in lead II, III, aVF, and V3–V6.[4] Differing from our case, these patients did not have a significant cardiac history such as ours.

Abdominal conditions can lead to ECG changes through various mechanisms, such as irritation or compression affecting the heart, alterations in heart position due to abdominal distention, heightened vagal tone resulting from the visceral-cardiac reflex, stress-induced cardiomyopathy, or variant angina.[1]

4. Conclusion

The presented case underscores the crucial importance of considering noncardiac etiologies when interpreting electrocardiogram (ECG) findings, especially in patients with a complex medical history. While acute coronary syndrome (ACS) remains a primary concern, various noncardiac conditions, such as abdominal pathologies, can mimic ACS-related ECG changes. In this instance, a 57-year-old patient with a significant cardiac history, including coronary artery disease and multiple stent placements, presented with unresponsiveness and pulselessness. Despite initial ECG indications of ACS, further investigation revealed an abdominal cause – a perforated ileus necessitating emergent surgical intervention.

The diverse range of noncardiac conditions leading to ECG alterations, including irritation, compression, positional changes due to abdominal distention, heightened vagal tone, stress-induced cardiomyopathy, or variant angina, emphasizes the need for comprehensive evaluation in complex clinical scenarios.

This case also highlights the challenges in managing patients with both cardiac and noncardiac issues. Despite achieving the return of spontaneous circulation and successful surgical intervention for the abdominal pathology, the patient faced persistent hemodynamic instability, and metabolic derangements, and ultimately succumbed to the complex interplay of factors.

Clinicians must remain vigilant in differentiating between cardiac and noncardiac causes of ECG changes, particularly in individuals with pre-existing comorbidities. Timely and accurate diagnosis is paramount for initiating appropriate management strategies. Acknowledging the potential mimics of ACS is crucial to preventing misdiagnosis, ensuring prompt intervention, and optimizing patient outcomes. This case underscores the intricate nature of medical decision-making, necessitating a comprehensive and multidisciplinary approach to addressing the complexities arising from concomitant cardiac and noncardiac conditions.

Conflict of interest

There is no conflict of interest.

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