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CASE REPORT | SMALL BOWEL

Beyond Cardiology: A Rare Encounter With Small Bowel Obstruction Disguised as Inferior Wall ST-Elevation Myocardial Infarction

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ABSTRACT

ST segment elevations (STEs) on an electrocardiogram (EKG) gravitate immediate attention to the heart. However, these EKG changes can sometimes be the result of noncardiac pathologies. Here, we present an interesting case of small bowel obstruction (SBO) masquerading as an inferior wall myocardial infarction. A 77-year-old woman with a history of aortic stenosis status postsurgical aortic valve replacement presented with chest pain. Workup revealed elevated high-sensitivity troponins and STE in the inferior leads. She subsequently underwent a left heart catheterization, which showed no critical plaques or stenosis. Persistent abdominal pain prompted further evaluation with a computed tomography scan of the abdomen, which demonstrated evidence of SBO. Conservative treatment with bowel decompression resulted in symptom improvement and complete resolution of the STEs on a follow-up EKG. This case underscores the importance of considering noncardiac etiologies, such as SBO, in the differential diagnosis of STE on EKG for accurate diagnosis and management.

KEYWORDS: small bowel obstruction; STEMI; myocardial infarction; ST elevation; bowel distention

INTRODUCTION

ST-elevation myocardial infarction (STEMI) carries significant mortality and morbidity. Therefore an urgent diagnosis with a 12-lead surface electrocardiogram (EKG) is important to facilitate timely intervention to restore myocardial perfusion and prevent permanent myocardial injury.[1] However, EKG has limitations, as about 2.3% of patients with suspected STEMI, based on initial EKG, are subsequently diagnosed with conditions that fall outside the ambit of acute coronary syndrome—these are termed STEMI mimics.[2,3]

These STEMI mimics can be categorized as cardiac (myocarditis, myopericarditis, early repolarization, ventricular aneurysm following an infarction, Brugada syndrome, stress cardiomyopathy, and coronary vasospasm), vascular (such as aortic dissection type A and pulmonary embolism), and noncardiac (including subarachnoid hemorrhage, pneumonia, chronic obstructive

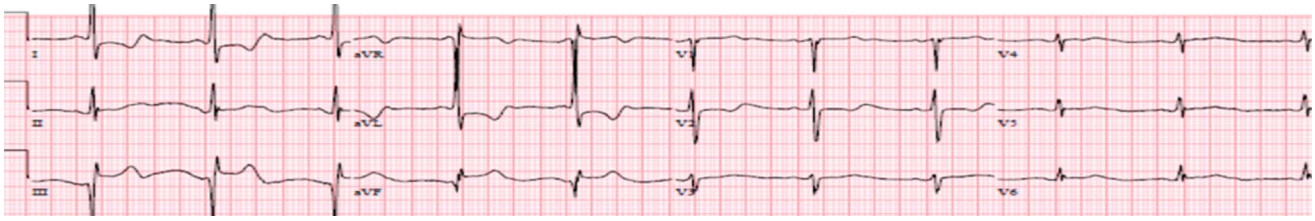

Figure 1. Electrocardiogram on admission showing ST elevation in III and augmented vector foot and ST depression in I and augmented vector left.

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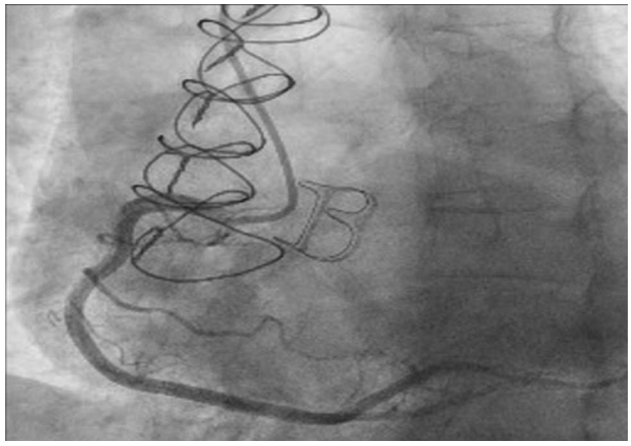

Figure 2. Left heart catheterization showing no significant coronary artery stenosis in the right coronary artery, posterior descending artery, and the posterolateral branch.

pulmonary disease, and mediastinal tumors). In rare cases, intra-abdominal pathologies such as pancreatitis, cholecystitis, and small bowel obstruction (SBO) can also mimic STEMI patterns on the EKG.[4–6]

We present a 77-year-old patient who presented with chest pain and demonstrated STE on EKG. This prompted coronary angiography, which showed no evidence of coronary artery stenosis. Subsequent abdominal imaging revealed SBO and conservative management with bowel decompression, led to immediate resolution of the EKG changes. This case highlights the importance of considering noncardiac causes, such as SBO, in the differential of STE on EKG to prevent unnecessary interventions and ensure appropriate treatment.

CASE REPORT

A 77-year-old White woman presented to the emergency department with chest pain, abdominal discomfort, nausea, and vomiting for 1-day duration. She had a medical history of calcific aortic stenosis status postsurgical bovine bioprosthetic aortic valve replacement, cholecystitis status post laparoscopic cholecystectomy, hyperlipidemia, hypothyroidism, and obstructive sleep apnea. The chest pain was nonexertional and was described as an intermittent, pressure-like sensation. This was associated with nausea and 4 episodes of nonbloody and bilious vomiting. The patient also reported dull, intermittent, and poorly localized abdominal discomfort.

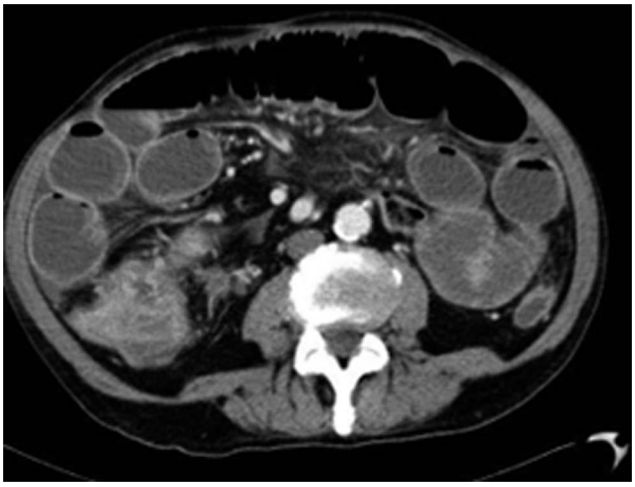

Figure 3. Computed tomography abdomen showing small bowel dilations concerning small bowel obstruction.

Vital signs on presentation were unremarkable, except for an elevated blood pressure of 175/84 mm Hg. Physical examination showed an ill-appearing, diaphoretic older woman. A systolic murmur, most prominent in the aortic area, was noted. The abdominal tenderness was generalized with no peritoneal signs, and bowel sounds were present.

An initial EKG showed ST elevations (STEs) in leads II, III, and augmented vector foot with associated Q waves and reciprocal ST depressions in I and augmented vector left (Figure 1), not noted on her previous EKGs. High-sensitivity troponins were 111 ng/L (normal value <54 ng/L). The patient was started on aspirin (325 mg), unfractionated therapeutic dose of heparin, ticagrelor, and a high-intensity statin. The cardiac catheterization laboratory was activated for an urgent coronary angiography. Coronary angiography with left heart catheterization (Figure 2) was performed, which revealed a 50% eccentric ostial right coronary artery stenosis with a thrombolysis in myocardial infarction 3 flow throughout. The absence of hemodynamically significant stenosis was confirmed by intravascular ultrasound, eliminating the need for coronary revascularization with either percutaneous coronary intervention or thrombolysis. A follow-up transthoracic echocardiogram revealed a normal LV systolic function with an estimated ejection fraction of 60%, and no regional wall motion abnormalities were noted. Stenosis of the bioprosthetic aortic valve was also noted. In the absence of hemodynamically significant coronary stenosis, minimal elevation in high-sensitivity troponins, and transthoracic echocardiogram showing normal wall motion abnormalities, cardiology concluded that the EKG changes were from a noncardiac origin and recommended discontinuation of therapeutic heparin and ticagrelor.

However, due to persistent abdominal and chest discomfort and nausea, a computed tomography scan of the abdomen and pelvis with and without intravenous contrast was obtained. This revealed dilation of the small bowel consistent with SBO (dilated small bowel loops >3 cm) (Figure 3). There was no transition point or enhancement of the bowel wall noted. A hiatal hernia was also present. Conservative management with nasogastric (NG) tube decompression and serial abdominal examinations were pursued. Following this, the patient reported significant improvement in her symptoms. A follow-up EKG revealed a complete resolution of STEs immediately after NG decompression (Figure 4). The NG tube was removed later, and the diet was advanced without concerns. An outpatient follow-up with gastroenterology was arranged for a potential colonoscopy

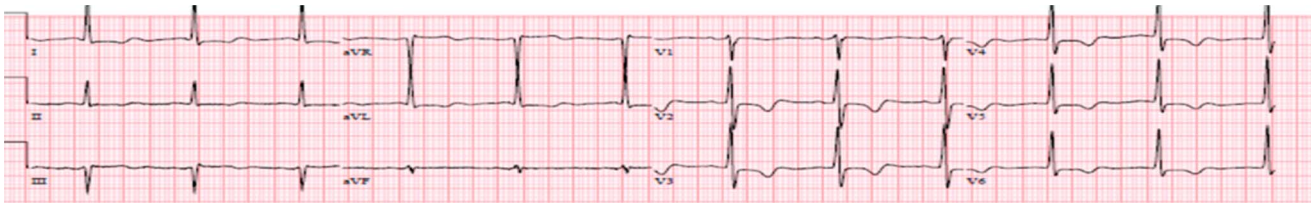

Figure 4. Electrocardiogram showing resolution of ST elevation in inferior leads after nasogastric decompression.

and/or esophagogastroduodenoscopy. Unfortunately, after understanding the risks and benefits, the patient refused to pursue any endoscopic intervention for further evaluation.

DISCUSSION

STE on an EKG that is not attributable to coronary artery disease is uncommon.[7] However, it can inadvertently lead to the activation of the catheterization laboratory and potentially delay the correct diagnosis and appropriate treatment.[8] Thus, knowledge of STEMI mimics becomes important to consider in the differential. A proportion of these mimics can be intra-abdominal pathologies, including diseases related to the pancreas, gallbladder, and esophagus; splenic ruptures; hiatal hernias; and SBO.[9,10] Specifically, the literature reports fewer than 20 cases of SBO without acute coronary syndrome presenting with STEs on an EKG.[7,9–14]

Although the precise mechanism remains elusive, several hypotheses have been put forth to explain these EKG changes in the setting of SBO. These include changes in the QRS axis of the heart due to gastric distension or irritation, cardiac displacement in relation to the surrounding structures, visceral-cardiac reflex, stress-induced cardiomyopathy, and pericarditis[10,11,14] (Figure 5). In this case, we hypothesize that increased intra-abdominal pressure and distention of the stomach from SBO could have resulted in diaphragmatic elevation, which may have irritated the diaphragmatic/inferior surface of the heart, leading to STE in the inferior leads.[10,15,16] In addition, the increased intra-abdominal pressure from SBO could have resulted in displacement of the heart due to elevation of the hemidiaphragm. This displacement might have altered the axis of the heart and caused electrophysiological changes, manifesting as STE on the EKG.[12,14] Though it is possible that the STE on the EKG and SBO noted on imaging could be unrelated, the subsequent alleviation of abdominal distension and resolution of STEs immediately following a NG tube insertion strongly supports the idea of an abdominal origin of the STE. Furthermore, the minimal elevation in high-sensitivity troponins could have resulted from a type-2 myocardial infarction (demand ischemia) in the setting of SBO. Giant hiatal hernias have been shown to cause STE, possibly through direct pressure on the global surface of the heart causing electrical alteration on EKG. Though our patient had a hiatal hernia, it was small and less likely to lead to EKG changes.

A meticulous medical history and physical examination are imperative to distinguish a true STEMI from STEMI mimics. For this patient, the lack of typical chest pain, the absence of significant risk factors of ischemic cardiac disease, a history of previous abdominal surgeries, and the presence of abdominal tenderness pointed toward an alternative cause of the STE. This was further corroborated by the angiography showing the absence of critical coronary artery stenosis. Therefore, echocardiography or computed tomography scans might have been more appropriate initial diagnostic tools to exclude STEMI and explore other potential causes. Thus, maintaining a high degree of clinical suspicion for STEMI mimics is crucial for atypical presentations. The advantages of timely intervention for true STEMI cases should be weighed against the potential risks of percutaneous coronary

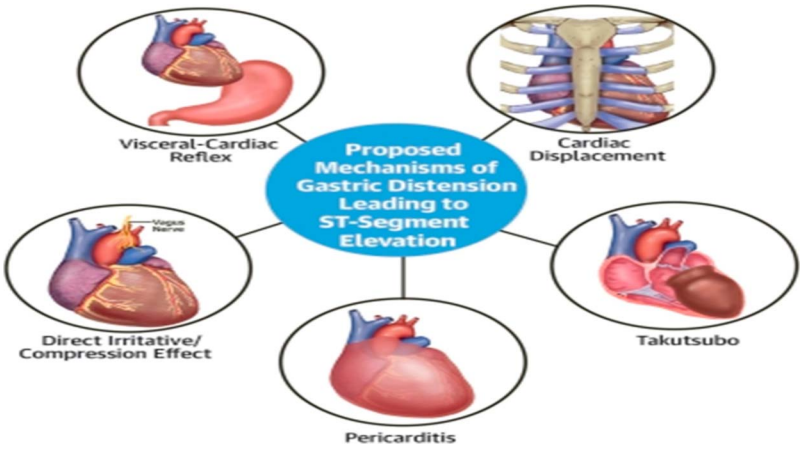

Figure 5. Diagram showing various proposed mechanisms of gastric distention leading to ST elevation.

intervention, antithrombin, and antiplatelet treatments, as well as the delays in addressing the actual underlying cause in cases of misdiagnosis.

This case underscores the diagnostic challenges posed by STEMI mimics such as SBO and raises awareness about the potential pitfalls of relying solely on initial EKG findings to evaluate chest pain in acute care settings. Timely intervention for STEMI is life-saving. However, clinicians must have a broad differential, especially in patients with atypical presentations and histories that suggest alternative causes. A comprehensive evaluation of historical, clinical, and investigational data is vital for providing clues to diagnosis.

DISCLOSURES

Author contributions: A. Duddu: contributed to the abstract, discussion, and picture captions. MR Shaik: contributed to the discussion. J Sairam: wrote the introduction and reference citations. R. Sharma and N. Jaafar: wrote and edited the case presentation. A. Duddu is the article guarantor.

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Informed consent was obtained for this case report.

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REFERENCES

1. Choudhury T, West NE, El-Omar M. ST elevation myocardial infarction. *Clin Med (Lond)*. 2016;16(3):277–82.
2. Gu YL, Svilaas T, van der Horst IC, Zijlstra F. Conditions mimicking acute ST-segment elevation myocardial infarction in patients referred for primary percutaneous coronary intervention. *Neth Heart J*. 2008;16(10):325–31.
3. Mahmoudi E, Hui JMH, Leung KSK, et al. Spiked helmet electrocardiographic sign: A systematic review of case reports. *Curr Probl Cardiol*. 2023;48(3):101535.
4. Agrawal A, Sayyida N, Penalver JL, Ziccardi MR. Acute pancreatitis mimicking ST-segment elevation myocardial infarction. *Case Rep Cardiol*. 2018;2018:9382904.
5. Patel N, Ariyarathenam A, Davies W, Harris A. Acute cholecystitis leading to ischemic ECG changes in a patient with no underlying cardiac disease. *JSLS*. 2011;15(1):105–8.
6. Jolobe OMP. Differential diagnosis of the association of gastrointestinal symptoms and ST segment elevation, in the absence of chest pain. *Am J Emerg Med*. 2021;49:137–41.
7. Khurana KV, Ranjan A. ST-segment elevation in conditions of non-cardiovascular origin mimicking an acute myocardial infarction: A narrative review. *Cureus*. 2022;14(10):e30868.
8. Larson DM, Menssen KM, Sharkey SW, et al. "False-positive" cardiac catheterization laboratory activation among patients with suspected ST-segment elevation myocardial infarction. *JAMA*. 2007;298(23):2754–60.
9. Patel K, Chang NL, Shulik O, DePasquale J, Shamoon F. Small bowel obstruction mimicking acute ST-elevation myocardial infarction. *Case Rep Surg*. 2015;2015:739147.
10. Singh M, Sood A, Rehman MU, Othman M, Afonso L. Elevated hemidiaphragms as a cause of ST-segment elevation: A case report and review of literature. *J Electrocardiol*. 2017;50(5):681–5.
11. Zhang J, Basrawala H, Patel S, et al. Gastrointestinal distention masquerading as ST-segment elevation myocardial infarction. *JACC Case Rep*. 2020;2(4):604–10.
12. Parikh M, Amor MM, Verma I, Osofsky J, Paladugu M. Small bowel obstruction masquerading as acute ST elevation myocardial infarction. *Case Rep Cardiol*. 2015;2015:685039.
13. Baldwin NK, Ives CW, Morgan WS, Bowman MH, Chatterjee A. Small bowel obstruction mimicking acute inferior ST-elevation myocardial infarction. *Am J Med*. 2021;134(5):599–602.
14. Herath HMMTB, thushara Matthias A, Keragala BSDP, Udeshika WAE, Kulatunga A. Gastric dilatation and intestinal obstruction mimicking acute coronary syndrome with dynamic electrocardiographic changes. *BMC Cardiovasc Disord*. 2016;16(1):245.
15. Mixon TA, Houck PD. Intestinal obstruction mimicking acute myocardial infarction. *Tex Heart Inst J*. 2003;30(2):155–7.
16. Asada S, Kawasaki T, Taniguchi T, Kamitani T, Kawasaki S, Sugihara H. A case of ST-segment elevation provoked by distended stomach conduit. *Int J Cardiol*. 2006;109(3):411–3.