

CASE REPORT

Inferior ST-Segment Elevation Pattern as a Result of a Small Bowel Obstruction: A Case Report

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Abstract: ST segment elevation patterns on Electrocardiogram (ECG) are a crucial finding in the diagnosis and treatment of acute coronary syndrome. An ST segment elevation pattern can be a sign of acute myocardial ischemia requiring immediate intervention. However, ST elevation patterns have been reported to occur due to obstructive intraabdominal pathology, a diagnosis often confirmed by cardiac catheterization. Here we report a 75-year-old female who presented to the emergency department with worsening chest and epigastric abdominal pain. ECG demonstrated ST-segment elevations in inferior leads (II, III, and aVF) with reciprocal changes in the lateral leads (I and aVL). Physical exam was suggestive of a bowel obstruction at the site of a large incarcerated ventral hernia, which was later confirmed by imaging. Due to the lack of typical chest pain symptoms and a strong suspicion of obstructive intraabdominal pathology, activation of the catheterization laboratory was deferred. Decompression of the bowel obstruction was achieved with a nasogastric tube, which resulted in immediate resolution of ST-segment elevations. During her admission, her ventral hernia was repaired, and left heart catheterization was deferred per cardiology recommendations. While an ST-segment elevation due to occlusive myocardial infarction is a diagnosis that cannot be missed and requires an emergent workup, it is important to be aware that it is possible for a small bowel obstruction (SBO) to present with ECG changes consistent with an ST-segment elevation myocardial infarction (STEMI). We also found that ST-segment elevations due to obstructive intraabdominal pathology are more reportedly seen in the literature in the inferior leads than any other contiguous leads, which is a novel pattern not discussed in past literature.

Keywords: Intestinal atresia; ST elevation myocardial infarction; Hernia, ventral; Myocardial infarction

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1. Introduction

Obtaining an Electrocardiogram (ECG) is a common order for patients presenting with abdominal pain, especially those who are older or have other risk factors for presenting as an atypical Myocardial Infarction (MI). In the setting of an acute occlusive MI, there are often elevations of the ST-segments in concordant leads, correlating with a region of the heart undergoing ischemic changes. An occlusive myocardial infarction is a diagnosis that cannot be missed and requires an emergent workup and usually a consultation with the interventional cardiologist. The 2018 Universal Definition of Myocardial Infarction (ESC/ACCF/AHA/WHF) classifies the five different types of myocardial infarction, with type 1 considered the classic spontaneous myocardial infarction due to atherosclerotic plaque or erosion (1). However, it is important to be aware that there are non-MI and non-cardiogenic causes of ST segment elevation. There have been numer-

ous case studies where intrabdominal obstructive pathology have been reported to cause a ST-segment elevation myocardial infarction (STEMI)-like pattern on ECG. In one example, Herath et al., 2016, described a case of acute gastric distension causing ST changes in septal and inferior leads, and it was postulated to have happened due to a possible change in heart position and cardiac axis from the obstructive pathology (2). In another case, Hibbs et al., 2016, describe a case of a patient presenting with ileus and an ECG showing ST changes in inferolateral leads that resolved after resolution of the ileus (3).

In this case report, we describe one such encounter in which a patient with a primary obstructive intraabdominal pathology was noted to have ST segment changes, which resolved with resolution of her intraabdominal disease. We also describe the prevalence of inferior ST-segment elevation patterns seen in cases of obstructive intraabdominal pathology in the literature, which is a novel pattern not discussed in past literature.

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2. Case presentation

A 75-year-old female with a medical history of morbid obesity, non-insulin dependent diabetes, hypertension, and hyperlipidemia, presented to our emergency department with worsening abdominal and chest pain. She reported watery diarrhea for 1 week, 3 days of abdominal pain, as well as 1 day of nausea and vomiting. On the day of presentation, her pain worsened and was localized to the epigastric and sub-sternal regions.

She presented as afebrile, tachycardic to 106 beats per minute, and normotensive. Physical exam was significant for a tender, firmly distended abdomen with a large irreducible ventral hernia. An ECG demonstrated ST-segment elevations in the inferior leads II and III as well as septal lead V2, with reciprocal changes in lateral leads I and aVL (Figure 1A). Due to the lack of typical chest pain symptoms and a strong suspicion of obstructive intraabdominal pathology, activation of the catheterization laboratory was deferred. Laboratory analysis revealed a white blood cell count of 27,000 cells/microliter [reference range 4,500 - 11,000 cells/microliter], Bicarbonate of 15 mEq/L [reference range 22-29 mEq/L], and blood urea nitrogen/creatinine (BUN/Cr) of 71/3.25 [reference range BUN 6-20 mg/dL; Cr 0.6 to 1.1 mg/dL]. An initial high sensitivity-troponin level was below the level of detection (<6 µg/L), and a repeat level was ordered to rule out developing cardiac ischemia. Computed tomography (CT) scan of the abdomen demonstrated a small bowel obstruction at the site of a large ventral hernia (Figure 2).

A nasogastric tube was inserted, with immediate 1 liter of feculent output. Her abdominal and chest pain improved with nasogastric decompression. Repeat ECG demonstrated resolution of ST-segment elevations (Figure 1B). Serial troponins remained below the limits of detection. Our patient was admitted to the intensive care unit for aggressive intravenous fluid hydration for acute kidney injury, prior to urgent ventral hernia repair. Echocardiogram during admission showed that the ejection fraction was unchanged from previous echocardiogram and no abnormal wall motion were seen. Left heart catheterization was deferred during this admission per cardiology recommendation, and the patient was discharged with instructions to follow up outpatient with her cardiologist.

3. Discussion

ST-segment elevations arising from intra-abdominal pathology such as gastric dilation and bowel obstruction have been reported in a few case reports (2, 3). The etiology of the STEMI pattern in many of these cases were diagnosed only after negative cardiac angiography (4, 5). The mechanism of ST-segment elevation is not-fully understood, but it is speculated to be related to increased intraabdominal pressure causing compression of the diaphragmatic surface of the heart (6, 7). In a case series done by Takato et al., the results

of a breathing test indicated that increased pressures causing cardiac compression can cause ST segment changes (8).

We present a rare case of a small bowel obstruction secondary to an incarcerated ventral hernia causing a STEMI pattern in inferior leads II and III and septal lead V2 and reciprocal changes in the lateral leads, consistent with an inferior STEMI with septal involvement. Upon literature review of the few case reports and series reporting STEMI patterns in cases of small bowel obstructions, most of the STEMI patterns had involvement of the inferior leads (II, III, and aVF) (3-7, 9, 10). This is consistent with the speculation that the ST-segment elevation is caused by compression of the diaphragmatic surface of the heart, comprising of the inferior wall. Only two case reports described instances of anteroseptal STEMI patterns seen with small bowel obstructions, with Herath et al. describing accompanying J point elevations in the inferior leads suggesting inferior wall involvement (2, 11). J point is a notched deflection after the QRS complex that is sometimes associated with ST-segment elevation and generally considered a non-specific early repolarization pattern on ECG (3). Further studies are necessary to further elucidate the prevalence of inferior ST-segment elevations and J points secondary to small bowel obstructions compared to other STEMI territories.

In our case, serial high-sensitivity troponin levels remained below the limits of detection. In previous case reports of STEMI patterns secondary to small bowel obstructions (SBOs), serial troponin results varied from being undetectable in some cases like ours and being elevated initially in others (2-5, 10). An undetectable serial troponin may aid in the diagnosis of ST-segment elevations caused by SBOs, but further studies need to be conducted to determine the diagnostic value of troponins in ST-segment elevations caused by obstructive intraabdominal pathology.

4. Conclusions

While an ST-segment elevation due to occlusive myocardial infarction is a diagnosis that cannot be missed and requires an emergent workup, it is important to be aware that it is possible for an SBO to present with ECG changes consistent with a STEMI. We also found that ST-segment elevations due to obstructive intraabdominal pathology are more reportedly in the inferior leads than any other contiguous leads, which is a novel pattern not discussed in past literature.

5. Declarations

5.1. Acknowledgments

None.

5.2. Author contributions

Conceptualization: Andrew Ryu, Andrew J. Jacobs, Christopher C. Lee

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5.4. Conflict of Interest

The authors declare no conflicts of interest to report.

5.5. Using Artificial Intelligence Chatbots

None.

5.6. Ethical considerations and patient consent

Authors certify that:

1. Is in agreement with the regulations of their institution(s) and generally accepted guidelines governing such work
2. Contains no violation of any existing copyright or other third party right
3. Is free of any obscene, indecent, libelous, or otherwise unlawful material

Patient consent obtained.

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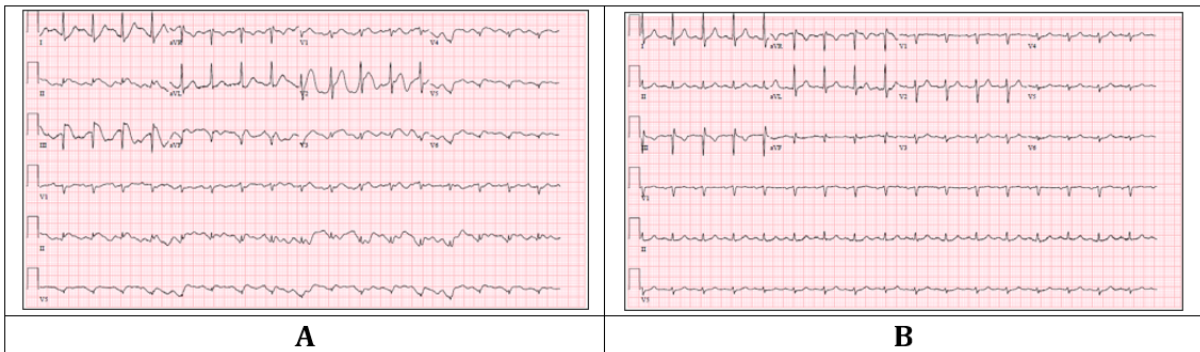


Figure 1: A: Initial 12-lead Electrocardiography demonstrating ST-segment elevations in anterior and inferior leads; B: Resolution of ST-segment elevations after nasogastric decompression.



Figure 2: Sagittal computed tomography image demonstrating large midline ventral hernia (red arrow) containing loops of small bowel, with dilated proximal small bowel.