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Small bowel obstruction imitating a fatal acute ST elevation myocardial infarction

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Introduction

ST-Segment Elevation Myocardial Infarction (STEMI) is an Acute Coronary Syndrome (ACS) that warrants immediate attention either by medical or surgical/interventional therapy. The clinical presentation can be elemental to diagnosing a STEMI however, the revelation on an EKG can prove ischemia causing tissue necrosis and/or myocardial injury. This may very well lead to an eventful cardiac dysfunction then consequentially proceed to arrhythmia and death. Hence, when a high index of suspicion of ACS is on a clinical presentation, it is classified as a medical emergency till proven otherwise. Although, there are variants to a STEMI; cardiac versus non cardiac, and in this case, we share a rarely reported case of small bowel obstruction imitating an ST-segment elevation myocardial infarction on an electrocardiogram.

Case presentation

A 60-year-old male with a past medical history significant for Ulcerative Colitis (UC) not on any treatment presented to the emergency department with a chief complaint of chest pain. The patient further described the pain located epigastric, lasting 2 hrs, squeezing, retrosternal, radiating to the left side of the chest, nausea, and intensity 9/10. The patient denied diaphoresis, dizziness, loss of consciousness, shortness of breath, dyspnea on exertion, orthopnea, vomiting, fever, cough, infectious symptoms, or other constitutional symptoms. The patient temp. 96F, HR 88 bpm, BP 144/91 mmHg, and RR 18. As part of the workup initiated a 12-lead ECG revealed ST elevations in V1, V2, and V3 with ST depression in inferior leads.

At this time, concern for anteroseptal wall infarction was suspected, and the catheterization lab activated. A Left Heart

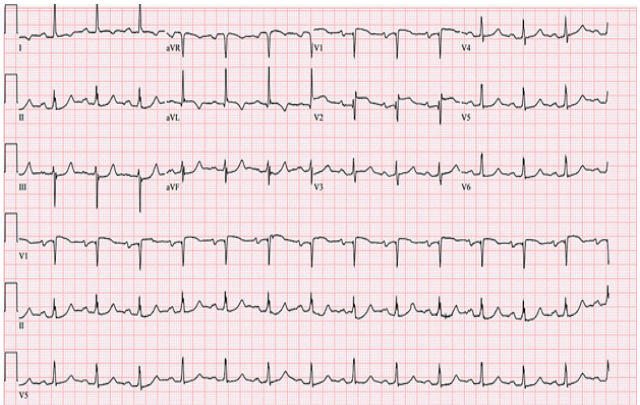

Figure 1: Initial 12 lead Electrocardiogram (EKG), Showing ST elevation in the anterior and septal wall leads. ST depression in the inferior leads.

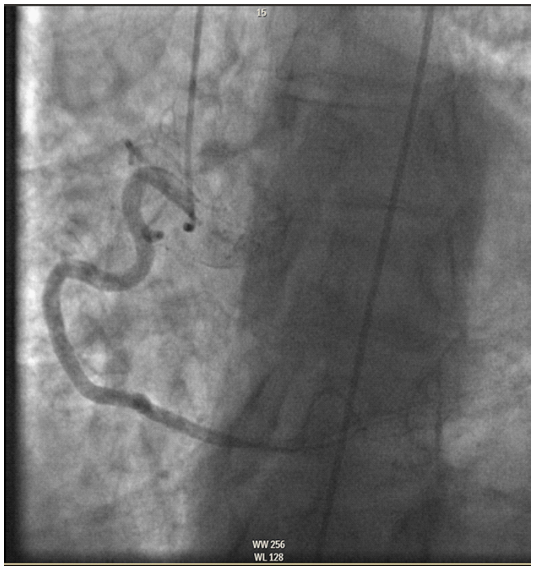

Figure 2: Diagnostic catheterization showing Right Coronary Artery (RCA), Image showing no evidence of narrowing and obstruction.

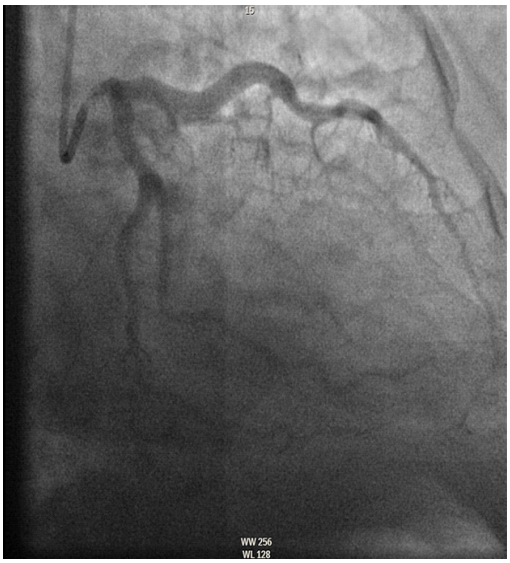

Figure 3: Diagnostic catheterization showing the left coronaries, Image with evidence of patent left coronaries.

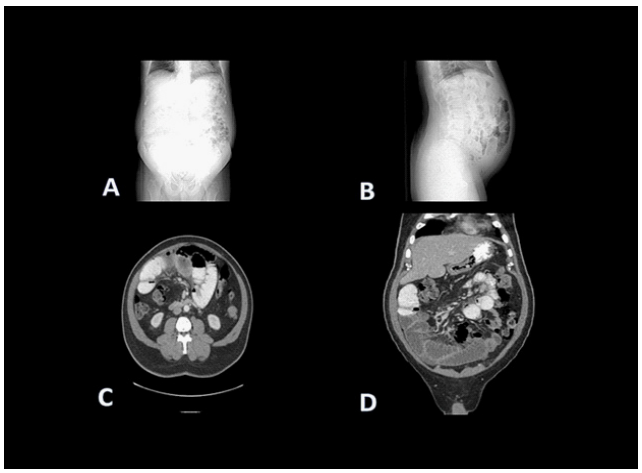

Figure 4: Computed Tomography (CT) Abdomen and pelvis **(A)** Scout Image showing distended abdomen. **(B)** Scout image showing dilated loops of bowel saggital view. **(C)** Evidence of dilated loops of bowel with contrast transition in mid-abdomen Axial view. **(D)** Coronal view of dilated loops of bowel and contrast reaching likely transition point in the mid- abdomen.

Catheterization (LHC) was performed and negative for thrombosis or stenosis of epicardial coronary arteries and showed non-obstructive coronary artery disease.

While on the table, the patient complained of abdominal pain then an episode of nonbilious, non-bloody emesis occurred. At this time, a change in the review of systems identified with abdominal distention. Immediately, post LHC, a Computed Tomography (CT) of the abdomen and pelvis was performed which was concerning for small bowel obstruction.

At this time, labs were reviewed and only remarkable for lactic acid 3.0 millimoles/liter (0.5-2.2). The patient remains afebrile, BP 150/90 mmHg, HR 110 bpm, RR 20. At this point, the patient was conservatively managed with the placement of a nasogastric tube to continuous suction and intravenous fluids. A repeated level of lactic acid at 1.6 millimoles/liter. A surgical evaluation was initiated and the patient underwent a diagnostic laparoscopy, and no evidence of any mass effect, adhesions, and/or hernias. In 72 hrs abdominal pain improved, with noted flatus, and a bowel movement. A repeat EKG was performed with no evidence of ST elevations or depression as seen in previous in anterior, septal, and inferior leads respectively.

A follow-up surgical evaluation led to the removal of the nasogastric tube and initiation of a clear liquid diet. Further discussion with the gastroenterology team recommended holding off any treatment for UC and plan for elective colonoscopy. An Echocardiogram showed a normal Left Ventricular Ejection Fraction (LVEF). At this time, the patient continued to improve and diet advanced as tolerated and discharged home with adequate follow-up with respective outpatient follow up services.

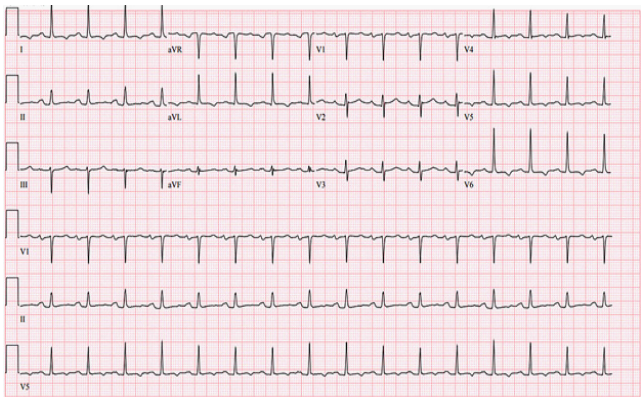

Figure 5: 12 lead Electrocardiogram post LHC and resolution of bowel obstruction. Normal sinus rhythm, and no evidence of ST elevation anterior and septal leads. No ST depression in inferior leads as seen in previous EKG.

A follow-up surgical evaluation led to the removal of the nasogastric tube and initiation of a clear liquid diet. Further discussion with the gastroenterology team recommended holding off any treatment for UC and plan for elective colonoscopy. An Echocardiogram showed a normal Left Ventricular Ejection Fraction (LVEF). At this time, the patient continued to improve and diet advanced as tolerated and discharged home with adequate follow-up with respective outpatient follow up services.

Discussion

STEMI is a life-threatening condition causing major causes of death in the United States of America (USA). STEMI comes under one of the three types of Acute coronary syndrome causing suddenly reduced blood flow to the heart. The other two

types are Non-ST elevation Myocardial Infarction (NSTEMI) and Unstable angina. STEMI is a time-sensitive emergency that must be diagnosed and treated promptly by coronary revascularization, frequently by percutaneous coronary intervention. A 12-lead Electrocardiogram (ECG) is the most readily available and reliable early diagnostic method for directing the treatment of patients with possible myocardial infarction [1]. According to the Third Universal Definition of Myocardial Infarction (MI) consensus, ST Elevation (STE) is defined as a new STE at the J-point in two consecutive leads. This however, is a corresponding cut points calculated at the J-point compared to the PQ junction: 0.1 milliVolts (mV) in all leads except leads V2-V3, which have a cut point of 0.2 mV in men ≥≥40 years; 0.25 mV in men ≤≤40 years, or 0.15 mV in women respectively [2].

There are variants to STEMI, and understanding the cardiac causes can however, Cardiac causes like myocarditis, acute aortic dissection, Acute pericarditis, Takotsubo cardiomyopathy, LBBB, and ventricular aneurysm are some of the causes other than ACS to cause STE and noncardiac causes such as pancreatitis, cholecystitis, bowel perforation, splenic rupture, hernias, and distension have also been reported to have STE [7].

Mechanism behind STE during abdominal causes is still not clear. There are very few case reports reported on possible pathophysiology, which includes an increase in abdominal pressure causing raise in diaphragm and change in cardiac axis, movement of the heart in anterior-posterior plane, irritative effect on the heart by the distended stomach, increasing vagal tone from the pain and causing variant angina [5].

The differential like right ventricular infarction, cor-pulmonale or myocardial contusion could be considered in this patient, but this patient did not have any hemodynamic changes, right atrial enlargement, elevated JVP, or crackles, making this diagnosis less likely [5]. Takotsubo syndrome is another differential for this patient, as ECG changes are usually seen in the precordial leads. However, in takotsubo, ECG changes stay for more than days unlike few hours as in our patient [6]; also will have characteristic apical ballooning on echocardiogram.

Here, the patient's distended abdomen on physical exam and a history of ulcerative colitis has a high likelihood of abdominal cause for ST elevations. A POC echocardiogram would also make a difference in decision making as absence of wall motion abnormalities would essentially rule out STEMI or make STEMI unlikely. Gastric distention should be considered as a non cardiac mimicker of STEMI [8].

Conclusion

STEMI is a time-sensitive, fatal condition and relies mostly on EKG to diagnose in acute settings. Along with EKG, it's crucial to give importance to a patient's history, physical examination, echocardiogram, cardiac enzymes, and overall clinical scenario rather than only EKG. When there is a high suspicion for intra-abdominal pathologies and STE is seen in the EKG, SBO should be considered as it can mimic STEMI, and we can avoid unnecessary work up and dangerous interventions like thrombolysis or angiography.

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