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Gastritis induced ST segment elevation on electrocardiogram

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Abstract

ST segment elevation on an electrocardiogram (EKG) is an alarming finding that warrants an urgent coronary angiogram. Early diagnosis and intervention are extremely important in the setting of acute coronary syndrome (ACS) to prevent irreversible myocardial damage and reduce the mortality rate. However, it is very important to know that not all ST elevations (STE) on EKG are due to myocardial infarction. Etiologies can be divided into cardiac and non-cardiac causes. Cardiac causes can include coronary aneurysm and acute pericarditis while non-cardiac causes can include acute cholecystitis and pulmonary embolism. In this paper, we are presenting a unique case of a patient with inferior STE on EKG that was found to be induced by gastritis. Knowing that this condition exists will help prevent patients from undergoing unnecessary interventions.

Keywords

ST elevation, Gastritis

Introduction

ST segment elevation on an electrocardiogram (EKG) is an alarming finding that warrants an urgent coronary angiogram. However, it is important to note that non-cardiac causes have triggered ST-elevation (STE) in certain cases. In this paper, we are presenting a unique case of a patient with inferior STE on EKG that was found to be induced by gastritis.

Case Presentation

A 63-year-old non-smoker male presented to the emergency department (ED) complaining of epigastric pain radiating to his right chest and nausea of one day duration. Medical history was significant for anxiety, hyperlipidemia, dyspepsia, and gastritis. In the ED, his vital signs were stable with a blood pressure of 124/83 mmHg, pulse rate of 74, respiratory rate of 20, and temperature of 97.6 F. On examination, his lung sounds were clear to auscultation, heart sounds were regular, abdomen was soft and not tender with normal bowel sounds, and peripheral edema was not present. Patient denied any shortness of breath, palpitations, dizziness, or diaphoresis. Initial laboratory workup, including electrolytes, cardiac enzymes, urine drug screen, amylase and lipase, were found to be within normal limits. An EKG was obtained and showed sinus rhythm with first degree atrioventricular block and no ST segment elevation (Image 1). Chest x-ray was normal. The patient was admitted for observation. He was given Mylanta as well as proton pump inhibitor (PPI); significant improvement occurred in the epigastric pain, decreasing in severity from 8 to 2. As part of the admission order set, a repeat EKG was done and showed sinus rhythm with STE in leads II, III, and AVF (Image 2). At that time, a second set of troponin came back normal. The patient was immediately re-assessed, and he denied any chest pain. Vital signs were stable with an unremarkable physical exam. The patient was loaded with Plavix and started on a heparin drip. He underwent coronary angiography, which showed no coronary artery disease (Images 4,5,6,7 and 8). Left ventriculogram showed normal ejection fraction with normal wall motion. Serial troponins were followed and found to be normal. Repeat EKG showed that

the STE significantly receded (Image 3). Patient was discharged in stable condition and was lost to follow up.

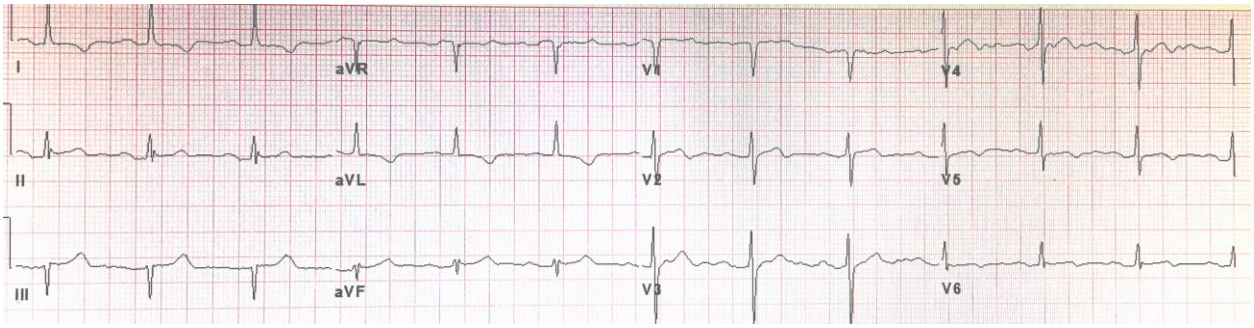

Image 1

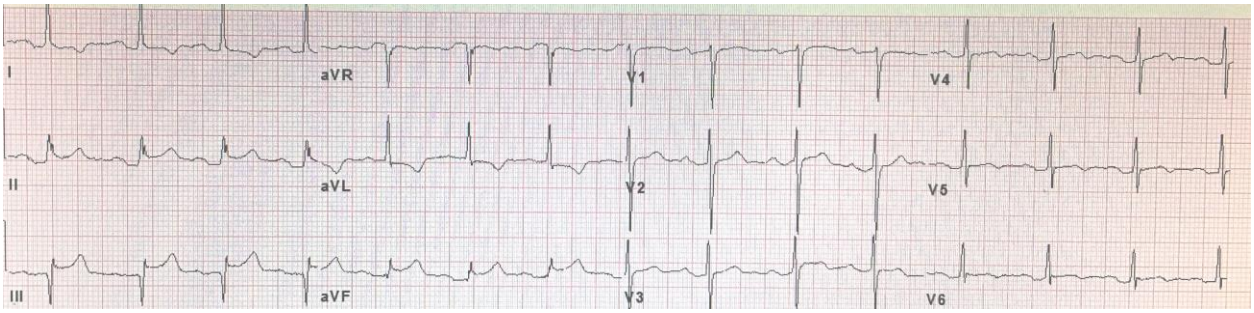

Image 2

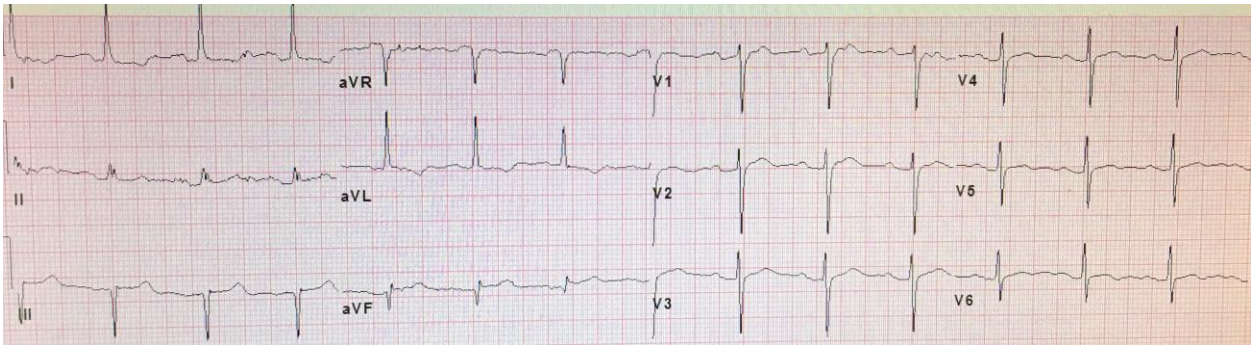

Image 3

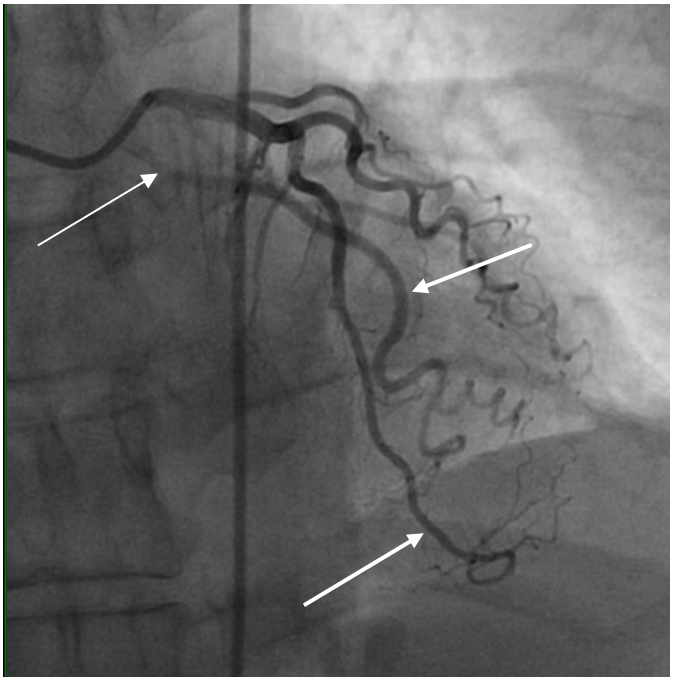

Image 4
Upper arrow: LCX; Middle arrow: OM; Lower arrow: LAD
View: AP

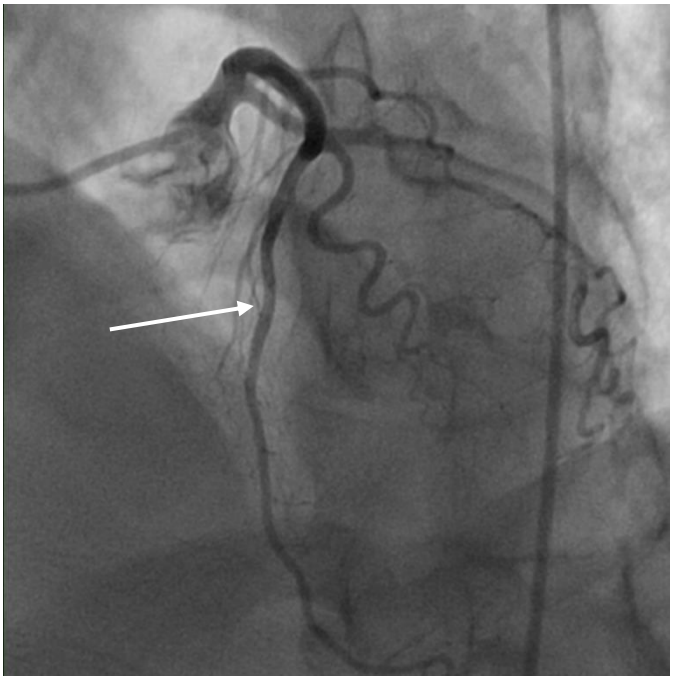

Image 5
Arrow: LAD
View: LAO Cranial

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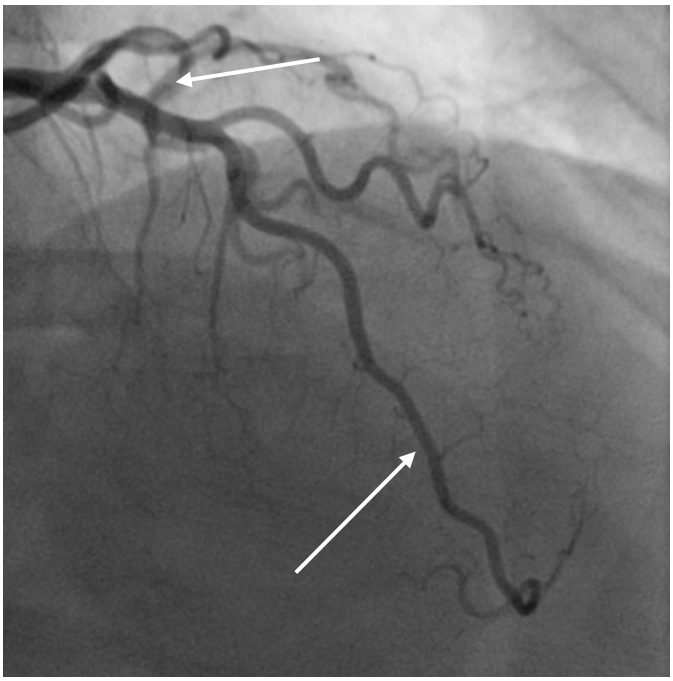

Image 6
Upper arrow: LCX; Lower arrow: LAD
View: RAO cranial

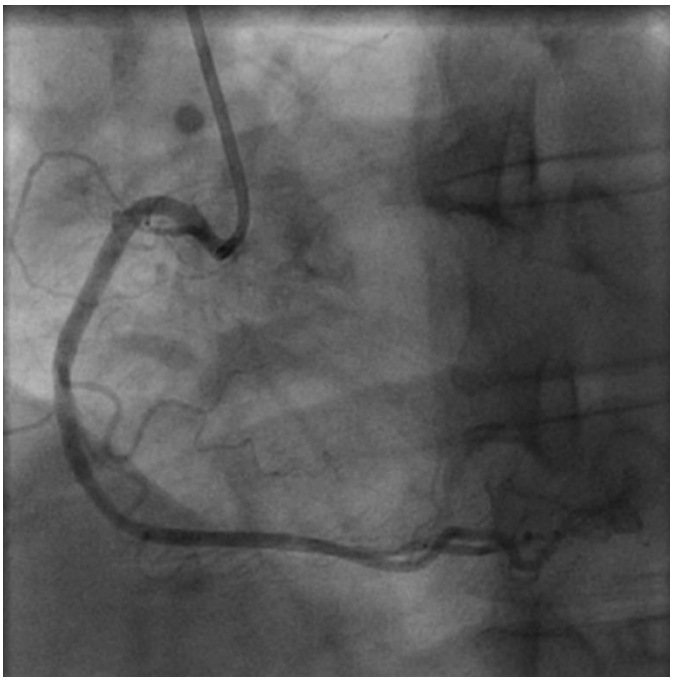

Image 7
RCA
View: LAO

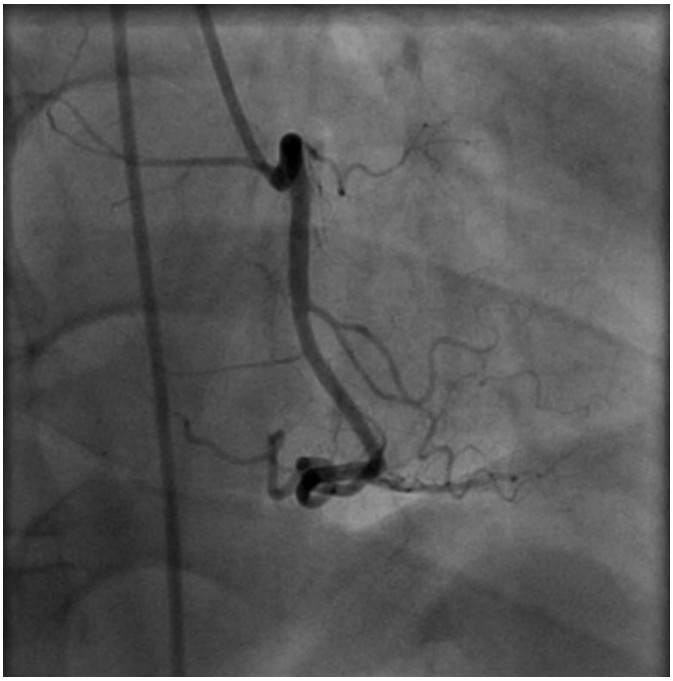

Image 8
RCA
View: RAO

Discussion

ST segment elevation myocardial infarction (STEMI) is a life threatening disease. In acute coronary syndrome (ACS), early diagnosis and intervention are extremely important in preventing irreversible myocardial damage and reducing the mortality rate. Currently, time to treatment is considered an indication for the quality of care.[1] However, it is important to note that in certain cases, non-cardiac causes have triggered STE. It is also important to recognize that 2.3% of STE are caused by etiologies other than myocardial infarction (MI).[2] These etiologies include cardiac causes such as coronary aneurysm, coronary artery spasm, acute pericarditis, cardiomyopathy, and Brugada syndrome.[2,3] Non-cardiac causes leading to STE have also been reported including subarachnoid hemorrhage, pulmonary conditions such as pneumonia, pulmonary emboli, chronic obstructive pulmonary disease, and gastrointestinal conditions such as acute pancreatitis, acute cholecystitis, and small bowel obstruction.[2-5] When compared to patients with STEMI, these patients are less likely to be smokers, report symptoms of angina, or have family history positive for cardiovascular disease.[1] The exact mechanisms as to why a non-cardiac cause leads to STE vary according to the etiology. In the case of subarachnoid hemorrhage and ischemic strokes, it is believed that the stress they induce on the central nervous system provokes a high catecholamine surge from local never endings in the heart leading to transient coronary vasoconstriction that causes ischemia and subendocardial myocardial damage.[6] Pulmonary conditions, on the other hand, lead to left and right ventricular strain which results in demand ischemia manifesting as EKG changes and elevated troponins.[7] In the case of abdominal etiologies including acute pancreatitis and cholecystitis, EKG changes are hypothesized to occur secondary to vagal nervous system changes and a catecholamine surge.[5,8] This explains why the changes seen are short lived and are most commonly located in the

inferior leads.[9,10] In certain cases, elevated troponins can also be seen. This results from pancreatic proteolytic enzymes release causing myonecrosis and coronary vasospasm.[11,12] Regardless of the etiology, these EKG changes will most likely resolve if the underlying condition is treated. We speculate that the STE seen in our case occurred secondary to gastritis induced vagal nervous system changes and a catecholamine surge. This is supported by the following facts: symptoms significantly improved after Mylanta and PPI, history of gastritis, STE in the inferior leads, and clean coronaries in the coronary angiogram.

Conclusion

In patients who present with acute MI with ST segment elevation, immediate reperfusion therapy will result in a better outcome. This requires rapid recognition of the STE and intervention. However, it is very important to know that not all STE are due to MI. Knowing that this condition exists will help prevent patients from undergoing unnecessary interventions.

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