

## **A concurrent presentation of nonspecific colitis as well as likely myocarditis**

Heavener T.<sup>A,B,D,E,F</sup>, Jepson M.<sup>A,B,D,E,F</sup>, Bushe B.<sup>A,B,D,E,F</sup>, Thotakura S.<sup>B,C,E,F</sup>, Chiles Ch.<sup>A,B,D,E,F</sup>

Scott & White Medical Center, Temple, TX, United States

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### **ABSTRACT**

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An ST-segment elevation myocardial infarction represents a time-sensitive cardiac pathology with utmost importance placed upon timely coronary angiography with percutaneous coronary intervention. While emphasis is placed on atherosclerotic or thrombotic coronary occlusion, it is important to recognize other etiologies which may present in a similar fashion. This case demonstrates a 71-year-old female patient with prior coronary artery disease and stenting who presented with acute abdominal pain and elevated cardiac biomarkers as well as ST-segment elevation on initial EKG. Coronary angiography revealed only mild to moderate coronary lesions and patent stents while echocardiography was essential unchanged from

prior evaluation. Computed tomography of the abdomen would show findings suggestive of infectious colitis and empiric antibiotics led to full resolution of symptoms. While no definitive cause for her cardiac manifestations was discovered, the authors propose coronary vasospasm or myopericarditis as likely etiologies in response to an overwhelming inflammatory state. The case underscores the importance of formulating a comprehensive differential diagnosis during the initial workup of a ST-segment elevation myocardial infarction.

**Keywords:** colitis, myocarditis, ST segment elevation, coronary vasospasm

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**\*Corresponding author:**

Trace Heavener, D.O.  
Scott & White Medical Center  
2401 S. 31st St.  
Temple, TX 76508  
Phone: 254-724-2364  
Fax: 254-724-4079  
Email: trace.heavener@bswhealth.org

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## INTRODUCTION

Myocardial ischemia is a mismatch between the oxygen demands of cardiac cells and the oxygen availability. If oxygen demand outstrips supply, the cardiac myocytes experience injury, called myocardial infarction (MI); this commonly manifests as the release of intracellular cardiac enzymes such as troponin. If the supply-demand mismatch is severe enough to cause transmural insult, ST segment elevation may be seen on electrocardiogram (ECG). Prompt diagnosis and treatment for ST-segment myocardial infarction (STEMI) is vital to the preservation of cardiac function. However, there are several known processes, other than MI, coronary atherosclerosis, or acute coronary thrombus, which may cause a presentation consistent with STEMI. These include, but are not limited to, coronary aneurysm [1], pericarditis [2], myocarditis [3], aortic dissection [4], subarachnoid hemorrhage [5], pneumonia [6], chronic obstructive lung disease [7], cholecystitis [8], and peritonitis. [7, 9] Furthermore, there are case reports on the correlations between ST segment elevation and ischemic colitis [10], as well as ST segment elevation and inflammatory (Inflammatory Bowel Disease) colitis. [11] In our review of the literature, we were unable to find any case reports of acute infectious colitis leading to a STEMI presentation.

## CASE REPORT

We present the case of a 71-year-old female who presented for right upper quadrant abdominal

pain, nausea and non-bloody non-bilious emesis. The patient had a medical history significant for coronary artery disease requiring prior drug eluting stent placement over ten years prior, uncontrolled type 2 diabetes mellitus, and heart failure with preserved ejection fraction. She had never smoked and had no known family history of MI. She also noted feeling anxious, short of breath, and had bilateral shoulder pain that was worse on the left.

In the emergency department, her vital signs were within normal limits. ECG revealed ST elevation in leads II, III and aVF, with a troponin level of 0.01 upon presentation (Fig. 1). Given the presentation, consistent with STEMI, she had emergent coronary angiography performed which revealed mild to moderate diffuse coronary disease with patent LAD stents (Fig. 2). An echocardiogram showed normal left ventricular systolic function with an ejection fraction of 50-55%, with no regional wall motion abnormalities, no valvular abnormalities, and no pericardial effusion. Subsequent laboratory work revealed cardiac troponin elevated up to 17.09 at six hours after admission and peaking at 27.44 at hour 14.

The following day she continued to have persistent abdominal pain and developed several loose non-bloody stools. Although a stool polymerase chain reaction test was negative for common diarrheal pathogens, an abdominal and pelvic computer tomography was obtained that showed diffuse inflammatory stranding and wall thickening involving the descending and sigmoid colon concerning for infectious, ischemic, or inflammatory colitis (Fig. 3).

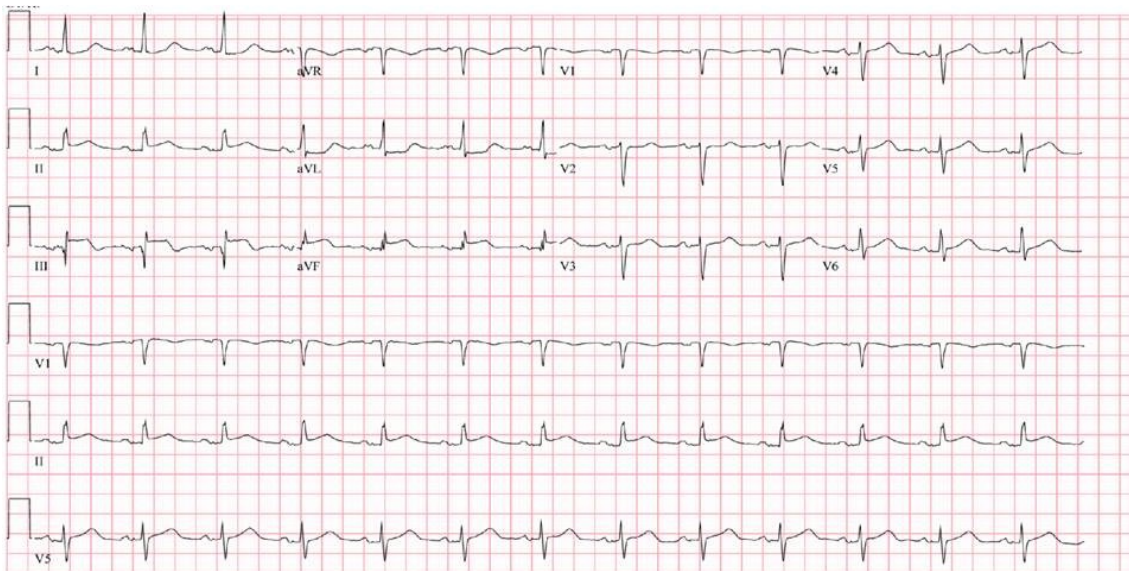
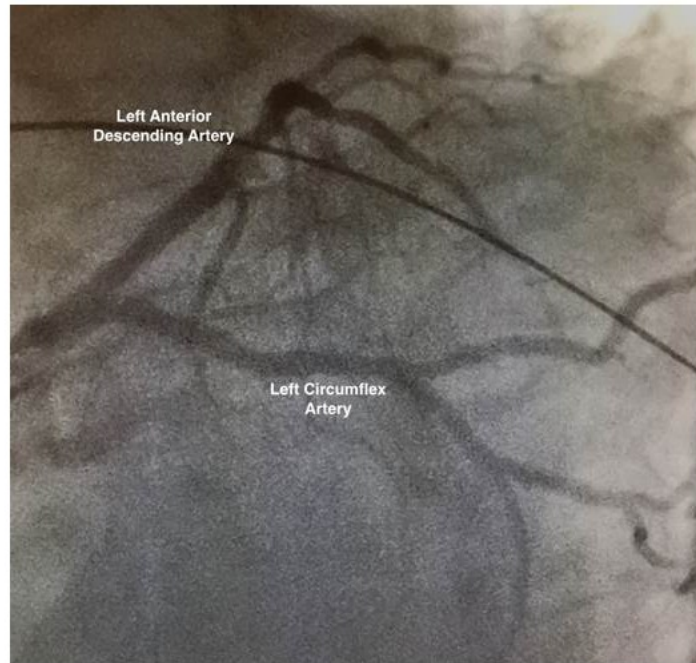


Fig. 1. Twelve lead ECG



**Fig. 2.** Angiogram of Left Coronary Artery from cranial view



**Fig. 3.** Axial cut of abdominal CT scan. Arrow denotes diffuse inflammatory stranding involving the sigmoid and descending colon up to the level proximal to splenic flexure with associated wall thickening throughout these segments

Subsequent computer tomography angiogram demonstrated no significant mesenteric stenosis. Ciprofloxacin and metronidazole were then started empirically. On the third day, the ECG abnormalities resolved and the troponin levels began

to decline. The patient's symptoms began to improve clinically on this day as well and thus no other laboratory, imaging or procedural tests were performed as it was felt they would not have impacted disease management.

## **DISCUSSION**

Acute MI is defined, based on the most recent guidelines, as a rise and/or fall of cardiac biomarkers with one of the following: symptoms of ischemia, new or presumed new significant ST-segment or T-wave changes or a new left bundle branch block, development of pathological Q waves, imaging evidence of new loss of viable myocardium or new wall motion abnormality, or identification of thrombus by angiography or autopsy. [12] These can be related to plaque rupture, vasospasm, fixed atherosclerosis with a supply/demand imbalance, or supply/demand imbalance alone.[13] Other criteria for acute MI include cardiac death with similar findings as previously stated, percutaneous coronary intervention, stent thrombosis, or coronary artery bypass grafting-related infarction with similar findings following surgery. [14]

ECG criteria have been established to include new ST segment elevation at the J point in two contiguous leads, with elevation being  $\geq 0.2$  mV in men  $\geq 40$  years old,  $\geq 0.25$  mV in men  $< 40$  years old, or  $\geq 0.15$  mV in women, in all leads other than V2-3. [15] In V2-3, the voltage criteria include  $\geq 0.1$  mV regardless of age or gender. While it has been well-documented that numerous disease processes can cause elevation of troponin, electrocardiographic evidence in the form of ST segment elevation is typically related with ischemic or inflammatory cardiac events, repolarization abnormalities, or electrolyte imbalances. In the only known primary study investigating incidence of diseases which mimic an acute MI, a 2008 retrospective chart review in the Netherlands revealed three presenting factors to be statistically significant in differentiating myocardial ischemia versus alternate diagnosis when having STEMI and elevated troponins: previous angina, smoking history and family history of MI at young age. [7] Although patients may not have these three risk factors, if they present with STEMI on ECG and elevated troponins, they may require rapid coronary angiography. [12] At the same time, providers should be mindful of disease processes other than MI that could have this presentation. During our review of the literature, there was a case report found of a myopericarditis related to non-specific colitis. [16] In that patient, after emergent cardiac evaluation, colonoscopy was performed which showed congested mucosal wall with scattered petechial involving the descending colon, sigmoid colon, and rectum. After ruling out obstructive coronary disease, that patient improved with conservative management and naproxen. Similar episodes of myopericarditis have been shown to

occur in conjunction with viral infections [17], certain medications [18], connective tissue diseases [19], or inflammatory bowel disease [20]. Viruses shown to be more common in having cardiotropic effects include Coxsackieviruses, adenoviruses, cytomegaloviruses, echovirus, influenza, Epstein Barr, Human Herpes Virus 6, Hepatitis C, and parvovirus B19 [21-22]. It has been postulated previously that a combination of these viruses, a predisposing genetic background, and the individual's immune responses ultimately contribute to both a direct viral-induced process as well as an indirect immuno-pathic process that both result in myocyte damage [23]. Diagnosis typically requires a consistent symptomatic syndrome, ECG and biomarker changes, as well as invasive testing such as endomyocardial biopsy as the gold standard. Imaging is typically obtained to include echocardiography evaluating for left ventricular function and pericardial effusion, and cardiac MRI to evaluate for pericardial thickening and myocardial inflammation. Guideline-based treatment includes high-dose NSAID therapy (1200 to 1800 mg of ibuprofen) [24]. If severe, then considerations for additional treatment would include systemic corticosteroids, adjunctive colchicine particularly for recurrent episodes [25], and possibly intravenous immunoglobulin, although there is still not enough evidence to support immunoglobulin as a recommendation [26].

Based on our literature review, the authors of this report conclude that this patient likely either had a STEMI related to a transient coronary vasospasm or myopericarditis, related to an acute infectious colitis that was likely viral in nature. This case highlights the importance of keeping a wide differential while managing emergent medical conditions and incorporating the entire clinical presentation in individualized patient care.

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## **Conflicts of Interest**

The authors have no conflicts of interest to mention

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None

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