

# A ST Elevation Myocardial Infarction in the Presence of Pneumoperitoneum



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## ABSTRACT

Myocardial infarction (MI) more commonly known as a “heart attack” is a relatively common occurrence with a wide array of potential etiologies. Many if not most MIs result in typical symptoms of angina (chest pain) or anginal equivalents which often is the primary driver to the diagnosis. In patients who are unable to communicate symptoms, such as those who are intubated and sedated, this can be challenging. In this case we discuss a patient who was intubated and sedated who develops a myocardial infarction in the setting of a pneumoperitoneum.

## Introduction

Acute myocardial infarction is the most common diagnosis in hospitalized patients in industrialized countries. An ST segment elevation myocardial infarction (STEMI) is due to the sudden decrease in coronary blood flow due to a thrombotic occlusion. Initially a plaque forms in the vessels mainly composed of lipids. This plaque development is secondary to risk factors such as hypertension, hyperlipidemia, and smoking. Though we are unable to predict when someone may suffer a STEMI, we can determine their likelihood to have a STEMI based on their risk factors and subsequently mitigate those risk factors.

STEMIs that occur in the inpatient setting typically occur in those older in age, female, and those with more comorbidities or risk factors. MIs are also known to occur in those with increased inflammatory states such as those who are critically or severely ill in the inpatient setting.

ST segment elevation in the critically ill is a relatively common finding, which may not always reflect a truly ischemic process. This is likely due to the elevated inflammatory state in these patients. The main difference in this population is the lack of the ability to verbalize chest pain which confers greater specificity to EKG findings in the non-critically ill.

## Case Report

A 83 year old female presented to the ED due to increasing abdominal pain in association with nausea, vomiting, and diarrhea. Patient has a past medical history significant for hypertension, diabetes mellitus type 2, and ESRD. Of note, patient was discharged two days prior to her most recent admission due to diverticulitis. She re-presented due to worsening symptoms. A clinical diagnosis of sepsis secondary to diverticulitis was established. Patient was subsequently started on empiric antibiotic treatment. Her initial CT did not show any evidence of free fluid or abscess.

As the patient’s clinical course progressed her symptoms did not subside and her WBC count continued to increase while on empiric antibiotic therapy. Infectious disease was consulted who broadened patient’s antibiotics to ertapenem and recommended a repeat CT abdomen/pelvis due to patient’s continued symptoms in the setting of a persistently increasing WBC count. Repeat imaging showed evidence of moderate pneumoperitoneum and free fluid in the right lower quadrant due to sigmoid colon perforation. General surgery subsequently performed an exploratory laparotomy with sigmoid colectomy.

Patient was also noted to incidentally have an elevated troponin at this time of 0.06. Patient’s post-operative EKG showed right bundle branch block with inferior ST segment elevations consistent with acute coronary syndrome. Once patient had undergone surgery, she was taken for emergent PCI in the setting of an inferolateral STEMI. Due to patient being intubated, it is unknown if patient was experiencing concomitant anginal symptoms. Patient was found to have a 99% occlusion of the left circumflex, significant ulcerated plaque with associated thrombus was noted. She underwent successful IVUS guided PCI with mechanical thrombectomy and placement of two drug eluting stents to the left circumflex artery.

Patient’s post-operative echocardiogram did show evidence of mild septal and inferior hypokinesis with a new reduced EF of 44%. After coronary intervention was performed, the patient was started on rectal aspirin and IV Cangrelor due to inability to use the upper GI tract in the post-operative period. The patient was transitioned from IV Cangrelor to oral Ticagrelor. She did well from a cardiac standpoint after suffering an acute coronary syndrome.

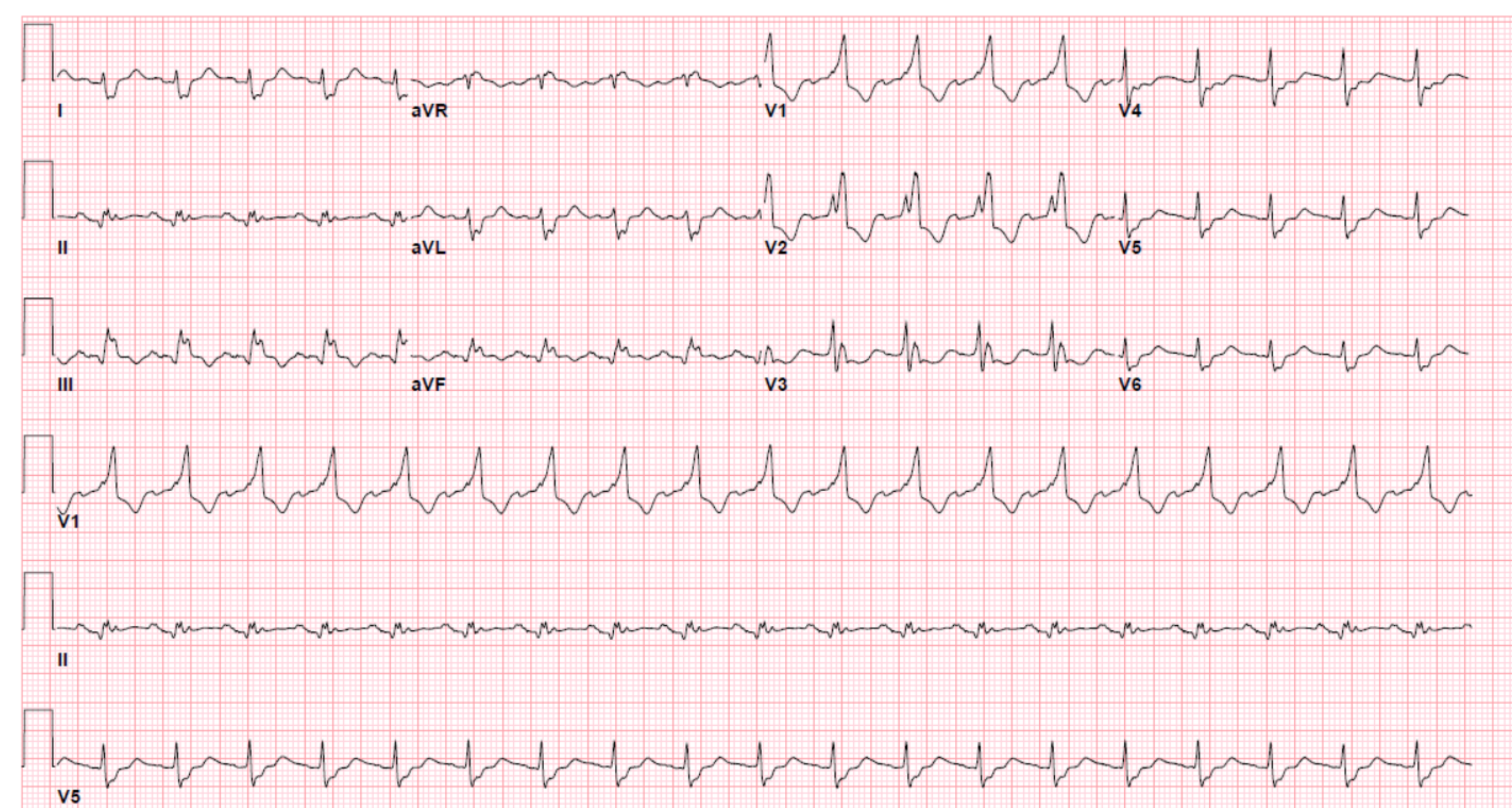


Figure 1

Figure 1: EKG showing findings of inferior ST segment elevation myocardial infarction. Right bundle branch block. Left posterior fascicular block. Vent. Rate: 116. PR int: 168. QRS duration: 148. QTc: 511.

Figure 2: Left heart catheterization. 99% proximal to mid occlusion of left circumflex artery; significant ulcerated plaque with associated thrombus noted.

Figure 3: Left heart catheterization. Post PCI image demonstrating excellent flow.

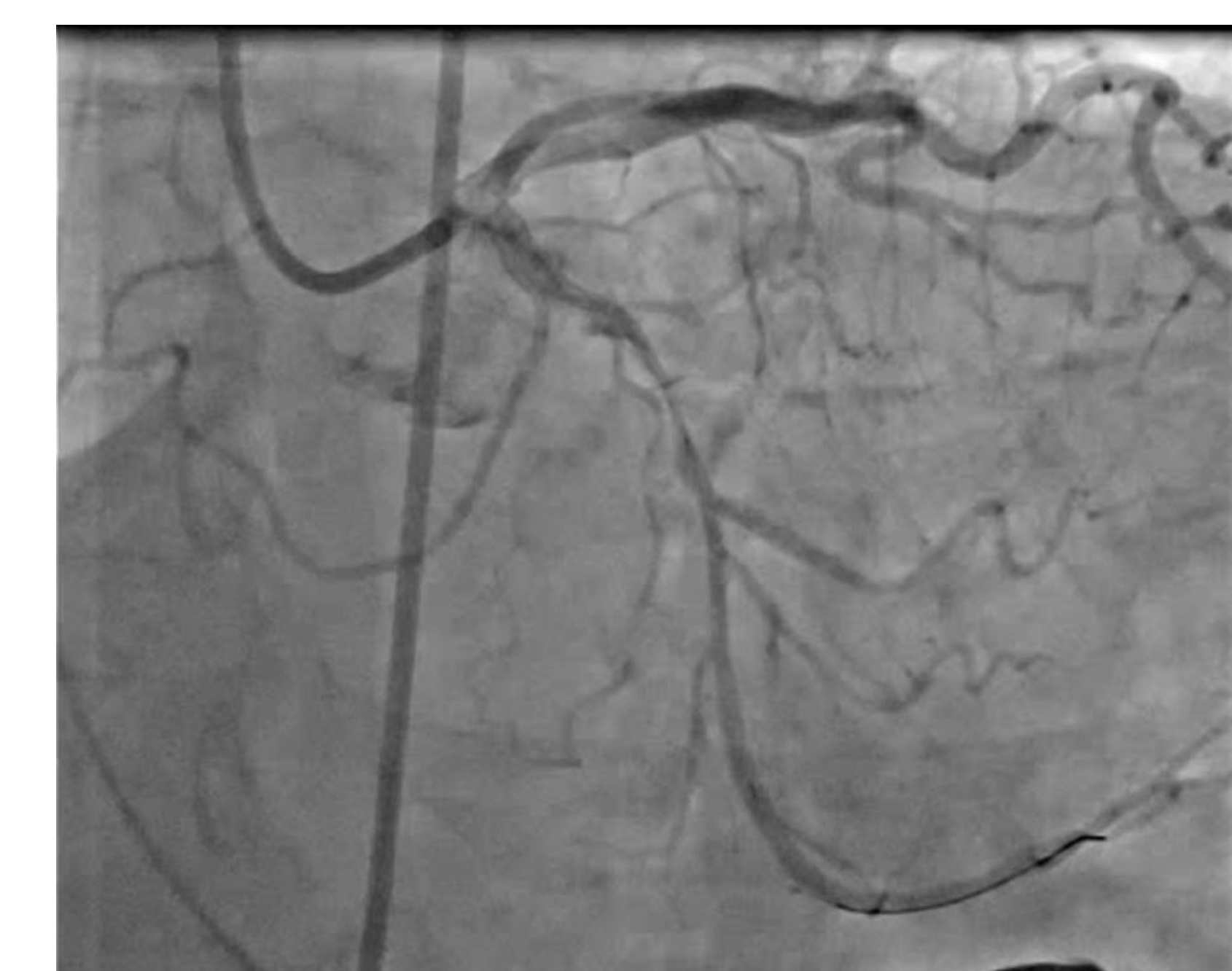


Figure 2



Figure 3

## CONCLUSION

This case illustrates the importance of having a high level of suspicion for myocardial ischemia in patients with multiple risk factors who are unable to report angina or anginal equivalents. Physiologic stress in the setting of pneumoperitoneum with feculent peritonitis was likely the culprit of this patient’s myocardial infarction.

It is not indicated to obtain daily troponins on patients who have not complained of chest pain, but checking troponin and EKGs in severely ill patients with multiple cardiac risk factors, especially in those who cannot verbalize anginal symptoms could potentially save patients from prolonged cardiac ischemia.

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