



Diagnostic Dilemma: Acute Mesenteric Ischemia may Mimic Acute ST-Segment Elevation Myocardial Infarction

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A 64-year-old man with no known cardiac history and no cardiovascular risk factors other than age presented with palpitations, respiratory distress, abdominal distension, hypotension, and sweating that started 3 h ago. The patient made no mention of chest or abdominal pain. On physical examination, the respiratory rate was 30 bpm, the heart rate was 138 bpm, the oxygen saturation on room air was 80% by pulse oximetry, and the blood pressure was 92/60 mmHg. The electrocardiogram (ECG) revealed ST-segment elevation in D1, inferior, V5-6 leads, and ST-segment depression in V1-3 leads with sinus rhythm (Figure 1a). With the diagnosis of acute inferoposterolateral ST-segment elevation myocardial infarction (STEMI), fibrinolytic, antiaggregant, and anticoagulant treatments were administered in the ambulance. However, the clinical findings did not improve, and abdominal distension worsened. The echocardiogram in the emergency department revealed normal left ventricular (LV) systolic function (LV ejection fraction by modified Simpson's method was 60% with no LV segmenter wall abnormalities), normal ascending aortic diameter and wall, and no significant valvular pathology or aortic dissection flap. Laboratory tests showed that lactic acidosis, C-reactive protein (130 mg/l), D-dimer (5 mcg/ml), white blood cell ($41.4 \times 10^3/\mu\text{l}$, neutrophil dominated), troponin T (408 pg/ml), and liver function tests were within normal limits. Cardiac, thoracic, and abdominal contrast-enhanced computed tomography (CT) angiography was performed. Cardiac CT revealed only non-critical stable plaque in the LAD, with no signs of intracoronary thrombus or plaque rupture. A thorax CT revealed no pathologic findings. Abdominal CT findings are shown in (Figure 2a, b, c). The patient was diagnosed with acute mesenteric ischemia (AMI) and had emergency surgery, as seen by the CT images. The patient underwent a successful operation and was discharged without any complications.

Acute abdomen and acute coronary syndrome are two crucial clinical conditions that may occasionally be intertwined in the emergency

department. Although the diagnoses of most patients are easy, a small subset of patients may require more careful attention. AMI is a rare acute abdominal presentation with a high mortality rate. It is responsible for the majority of emergency bowel resections, particularly in older patients with comorbidities that increase their susceptibility to thromboembolic events, such as atrial fibrillation (AF).^{1,2} Mesenteric ischemia is often caused by arterial thrombi in the mesenteric arteries, which results in reduced intestinal blood supply and cellular damage.³ Rapid diagnosis and early treatments are critical for lowering mortality rates exceeding 50%. The clinical presentation of AMI may differ depending on specific or non-specific laboratory and clinical findings. Patients with AMI may present with tachypnea,

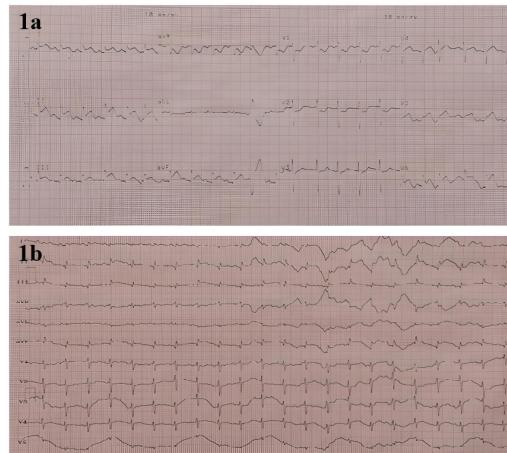


FIG. 1. (a) Patient's admission electrocardiogram (ECG) in the ambulance shows convex ST-segment elevation in the inferior and lateral leads and ST-segment depression in leads V1-3; (b) postoperative ECG showed regression of ST-segment elevations and depressions after laparotomy and 120-cm necrotic small bowel resection.

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Received: October 24, 2023 **Accepted:** November 22, 2023 **Available Online Date:** February 29, 2024 • **DOI:** 10.4274/balkanmedj.galenos.2023.2023-10-81

Available at www.balkanmedicaljournal.org

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Cite this article as: Özlek B, Dere Ö, Köseoğlu FD. Diagnostic Dilemma: Acute Mesenteric Ischemia may Mimic Acute ST-Segment Elevation Myocardial Infarction. Balkan Med J; 2024; 41(2):149-50.

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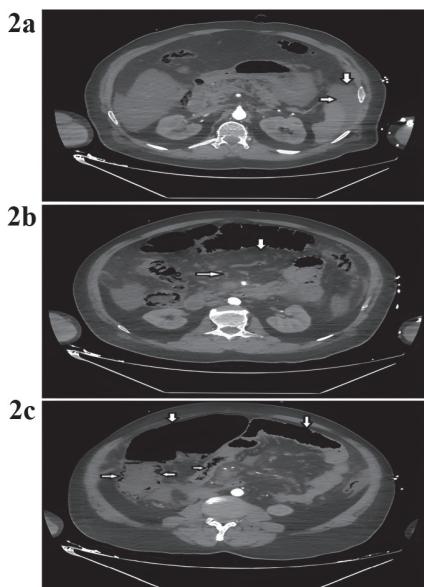


FIG. 2. On contrast-enhanced abdominal CT performed in the emergency department, (a) white arrowheads indicate peri splenic free fluid; (b) arrows indicate diffuse heterogeneous mesenteric fatty tissue; (c) this image shows intestinal dilatation and pneumatoisis intestinalis.

sweating, hypotension, and tachycardia as in the present case and generally with symptoms of nausea, vomiting, abdominal pain, and diarrhea. Approximately 90% of these patients had abnormally high leukocyte counts, metabolic acidosis, increased lactate, and high D-dimer levels.² Due to the scarcity of specific findings, the clinician must consider infection (e.g., mycobacterium-avium intracellular, cytomegalovirus, cryptosporidiosis, and giardiasis), inflammatory bowel diseases (e.g., Crohn disease, Celiac disease, diverticulitis, radiation enteritis, and graft-versus-host disease), neoplasm, infiltrative diseases (e.g., amyloidosis), scleroderma, intramural hemorrhage, and angioedema in the differential diagnosis of AMi.⁴ Another clinical emergency that can occur in adult patients of all ages is acute STEMI, which is typically diagnosed only by ECG and requires immediate intervention. It is uncommon for AMi to mimic acute STEMI on the ECG. So far, two cases have been reported in the literature.^{5,6} Typical ST-segment elevation on the ECG should initially suggest the clinician to a type I myocardial infarction (MI). However, evaluating the patient in conjunction with other clinical and laboratory findings in the differential diagnosis of AMi is crucial. Interestingly, after bowel resection, the present patient's ST-T abnormalities completely disappeared on ECG (Figure 1b).

It is unknown why mesenteric ischemia and inflammation might cause ST-segment elevation on the ECG. One possibility is that the widespread inflammation and tissue death in the mesenteric area might emerge as an inferior wall MI, as shown by the inferior leads of the ECG. The present patient is most likely suffering from type II MI caused by bowel ischemia. The ECG suggests a type I MI; however, type I MI only manifests with tachycardia, hypotension, and hypoxia if there is severe LV dysfunction (cardiogenic shock). In such a case,

there is poor LV function (or, much more rarely, good LV function with a severe valvular disorder). Thus, the clinical finding of normal LV systolic function raises the possibility that this clinical presentation is not the result of acute coronary occlusion. In our case, because the first medical team in the ambulance diagnosed the patient with acute STEMI based on ECG alone without an echocardiogram, the patient was initially treated based on this initial diagnosis and transferred to the emergency department. In this respect, the echocardiogram performed in the emergency department was useful in guiding the differential diagnosis. Type II MI with ST-segment elevation is uncommon,⁷ although it does occur. It is most prone to develop in the presence of a fixed coronary stenosis (which results in poor coronary flow, mainly when hypotension is present). Such stenosis may result in type II STEMI due to increased oxygen demand and reduced supply, but coronary stenosis is unnecessary. In this case, increased demand from tachycardia and decreased supply from hypotension and hypoxia could be sufficient to cause a type II STEMI. Assume a patient has shock and/or hypoxia but does not have a very low LV ejection fraction, even if the ECG reveals an acute occlusive MI. In that case, the ECG findings are likely due to the shock (rather than acute coronary occlusion, such as AMi). As the primary initiating event, another disorder causes that shock.

The risk of AMi is higher in adults with many cardiovascular risk factors or comorbidities, such as AF,¹ and the diagnosis of AMi in these patients may be more comfortable because it is easier to think of the diagnosis. Nevertheless, it may be life-saving for the clinician to keep the possibility of AMi in mind even in patients with no apparent risk factors, as in the present case.

Informed Consent: The patient signed a written informed consent to publish this case report.

Authorship Contributions: Concept- B.Ö., F.D.K.; Design- B.Ö., F.D.K.; Data Collection or Processing- B.Ö., Ö.D., F.D.K.; Analysis or Interpretation- B.Ö., Ö.D., F.D.K.; Literature Search- B.Ö., Ö.D., F.D.K.; Writing- B.Ö. Ö.D. F.D.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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