

A myocardial infarction detected in an unusual way

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Abstract

An 86-year-old man presented to the emergency department with abdominal pain in the left iliac fossa and constipation in the previous three days. Laboratory tests revealed an increase in C-

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reactive protein levels (22.37 mg/dL), white blood cell count ($12.38 \times 10^9/L$), neutrophil count ($10.07 \times 10^9/L$), and hyperglycemia (237 mg/dL). An abdominal contrast-enhanced computed tomography scan ruled out acute abdominal pathological findings but showed a marked hypoperfusion of the mid-apical anterior wall and the apex of the left ventricle. A subsequent electrocardiogram revealed an excessive ST elevation (>5 mm) and a negative QRS complex in precordial leads (from V1 to V6). Serial assays of high-sensitivity troponin I showed a plateau phase (from 15696.4 to 15479.3 ng/L). Echocardiography revealed a left ventricle dilated and severely hypokinetic (ejection fraction of approximately 15-20%). A diagnosis of subacute myocardial infarction with severe dysfunction of the left ventricle was therefore made.

Introduction

A contrast-enhanced computed tomography (CT) scan is not typically included in the evaluation of myocardial infarction.¹ However, many patients may present with atypical symptoms, leading them to undergo an emergency contrast-enhanced CT scan to rule out other suspected acute conditions. In some cases, this scan may ultimately reveal a myocardial infarction.² We report a case of a subacute myocardial infarction detected by an abdominal contrast-enhanced CT scan.

Case Report

An 86-year-old man presented to the emergency department with abdominal pain in the left iliac fossa and constipation in the previous three days. His medical history included cholecystectomy for lithiasis, radical prostatectomy for prostate cancer, dual-chamber pacemaker implantation for second-degree atrioventricular block, drug-eluting stent implantation in the left anterior descending artery for acute myocardial infarction, chronic ischemic heart disease, hypertension, and type 2 diabetes mellitus. He was under treatment with low-dose aspirin, irbesartan, hydrochlorothiazide, bisoprolol, pantoprazole, metformin, silodosin, and amlodipine. On physical examination, his vital signs were normal, and his abdomen was tender in the left iliac fossa with no signs of peritonism. Laboratory tests revealed a marked increase in C-reactive protein levels (22.37 mg/dL), a modest increase in white blood cell count ($12.38 \times 10^9/L$) and neutrophil count ($10.07 \times 10^9/L$) and hyperglycemia (237 mg/dL). An abdominal contrast-enhanced CT scan ruled out acute pathological findings in the abdominal cavity but caught a few slices of the heart showing a marked hypoperfusion of the mid-apical anterior wall and the apex of the left ventricle (Figure 1); the CT scan showed no thrombus in the left ventricular lumen. A subsequent 12-lead electrocardiogram (ECG) revealed spontaneous atrial rhythm followed by ventricular paced rhythm with an excessive ST elevation (>5 mm) and a negative

QRS complex in precordial leads (from V1 to V6) (Figure 2). These alterations were not compatible with repolarization alterations secondary to ventricular pacing, but they were strongly suspicious for ischemia. Serial assays of high-sensitivity troponin I showed a plateau phase (levels ranged from 15696.4 to 15479.3 ng/L; normal values < 19.8 ng/L). B-type natriuretic peptide levels were 678 pg/mL (normal values < 100 pg/mL). Echocardiography revealed a left ventricle dilated, hypokinesia of the anterior and anterolateral wall of the left ventricle, and akinesia of mid anteroseptal and apical septal segments of the left ventricle (ejection fraction of approximately 15-20%). Based on all of these findings, a diagnosis of subacute myocardial infarction with severe

dysfunction of the left ventricle was made. A dual antiplatelet therapy (aspirin and clopidogrel) was undertaken because of patient frailty and subacute presentation of myocardial infarction. A few days later, the patient presented with melena and severe anemia (hemoglobin 5.7 g/dL); dual antiplatelet therapy was suspended, and the patient underwent esophagogastroduodenoscopy, which revealed some ulcerative lesions in the gastric body and antrum. After some transfusions of red cell concentrates, aspirin was resumed. Clinical conditions improved, and the patient was finally discharged from the hospital with home therapy (low-dose aspirin, folic acid, atorvastatin, bisoprolol, furosemide, pantoprazole, metformin, dapagliflozin, canrenone, and silodosin).

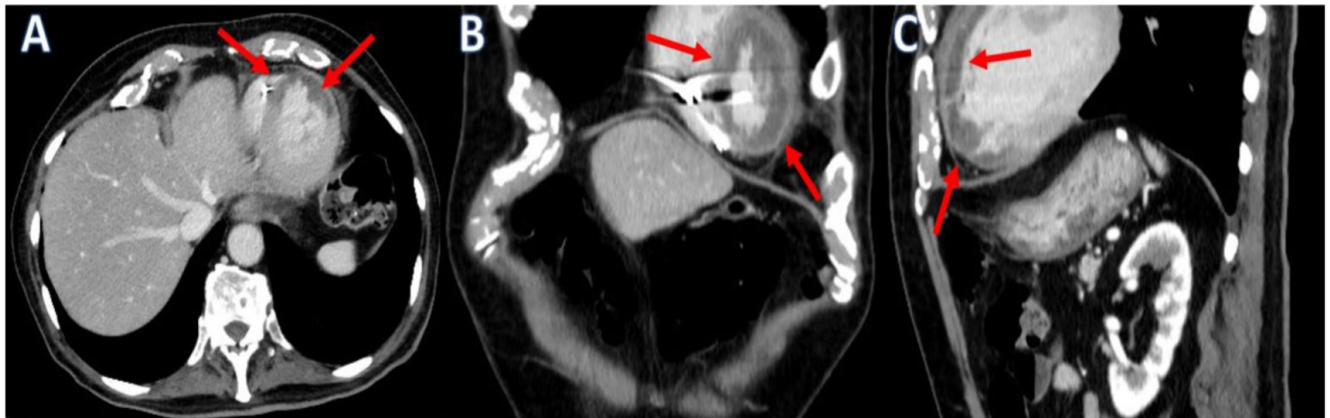


Figure 1. Axial (A), coronal (B), and sagittal (C) contrast-enhanced computed tomography scan of the abdomen showing a marked hypoperfusion of the mid-apical anterior wall and the apex of the left ventricle (red arrows).

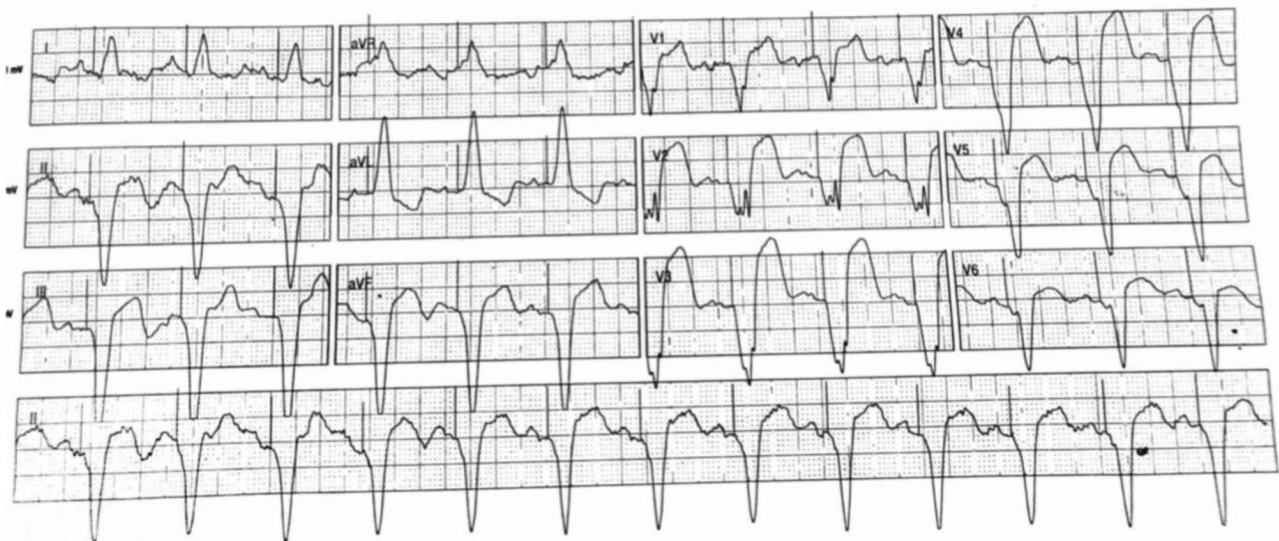


Figure 2. 12-lead electrocardiogram showing spontaneous atrial rhythm followed by ventricular paced rhythm with an excessive ST elevation (>5 mm) and a negative QRS complex in precordial leads (from V1 to V6). A notch in the ascending limb of the S wave in lead V3, and less evident in the ascending limb of the S wave in lead V4, is also present (the so-called "Cabrera's sign"). These alterations were not compatible with repolarization alterations secondary to ventricular pacing, but they were strongly suspicious for ischemia.

Discussion and Conclusions

The prevalence of myocardial infarctions presenting with atypical symptoms is high (nearly 34%).³ The atypical symptoms tend to occur more commonly among older, female, diabetic (possibly due to autonomic neuropathy), hypertensive, and with prior heart failure patients.⁴ Atypical presentations frequently cause delays in seeking care, suspecting or diagnosing by the treating physician, resulting in a poor prognosis.⁵

One of the most common atypical presentations of myocardial infarction is gastrointestinal discomfort (21.43%).⁴ As a result, some patients undergo an emergency CT scan of the abdomen because of suspected abdominal pathologies.⁶ Abdominal CT scans frequently cover the caudal parts of the heart, allowing the evaluation of myocardial contrast attenuation and possibly suspecting ischemia.⁷ The pattern of coronary perfusion is centripetal, with the subepicardium of the left ventricle better perfused and thus more resistant to ischemia than the subendocardium. Perfusion abnormalities, therefore, usually manifest first and most profoundly in the subendocardium. The subepicardium is usually less affected by perfusion abnormalities, but transmural ischemia can also occur. Ischemic defects follow specific coronary territories.⁶

As a result, radiologists could help shorten delays to myocardial infarction diagnosis in some cases by taking a quick look at myocardial contrast attenuation. However, a recent retrospective single-center study assessed that radiologists regularly miss myocardial hypoperfusion during the evaluation of abdominal CT scans, probably because they do not think about the eventuality of myocardial infarction.⁷

Nevertheless, it is important to underline that radiological visualization of myocardial hypoperfusion alone does not typically reveal the onset, apart from cases where calcifications or fatty atrophy of the myocardium are present: medical history, ECG, echocardiography, and laboratory parameters can therefore help to decide whether an acute or a chronic situation is present or not.⁷

In conclusion, emergency radiologists should look at myocardial contrast attenuation during the evaluation of abdominal CT scans, especially in those patients who present to the emergency

department with gastrointestinal discomfort, cardiovascular risk factors, and no pathological findings in the abdominal cavity on CT images. Emergency physicians should think about atypical manifestations of myocardial infarction in those patients who present to the emergency department with gastrointestinal discomfort and cardiovascular risk factors, especially if CT scan rules out pathological findings in the abdominal cavity.

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