

Uncommon Cause of ST Elevation

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A 73-year-old man presented with severe postprandial epigastric pain that did not radiate. He had 6 episodes of emesis followed by retching. The character of the pain was different from past anginal symptoms. At his local emergency department, a chest roentgenogram revealed air presumably within a large intrathoracic hiatal hernia (Figure 1). Computed tomography was notable for the stomach being almost entirely intrathoracic and distended with an organo-axial volvulus configuration (Figure 2). A nasogastric tube was inserted, and he was transferred to our institution. His medical history included hiatal hernia, hypertension, dyslipidemia, diabetes mellitus, and chronic kidney disease. He had undergone coronary artery bypass grafting 26 years previously with vein grafts to his left anterior descending artery and right coronary artery. Ten years ago, he had redo surgery with a left internal mammary artery to the left anterior descending artery. His medications included aspirin, β -blocker, angiotensin receptor blocker, statin, and nitrates. He was afebrile; his pulse was 73 bpm and blood pressure was 169/87 mm Hg. Chest examination revealed a significant systolic upper sternal lift with normal chest auscultation. His abdomen was not tender. He had normal leukocyte count, creatinine of 1.5 mg/dL (normal values, 0.8 to 1.3 mg/dL),

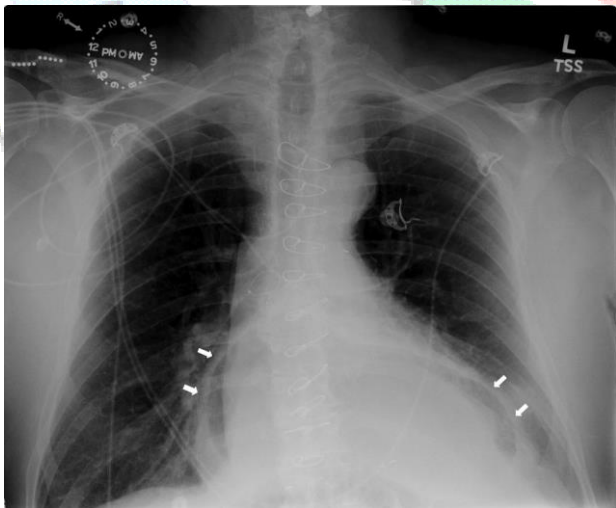


Figure 1. Portable anterior-posterior chest x-ray with arrows delineating air within the intrathoracic stomach.

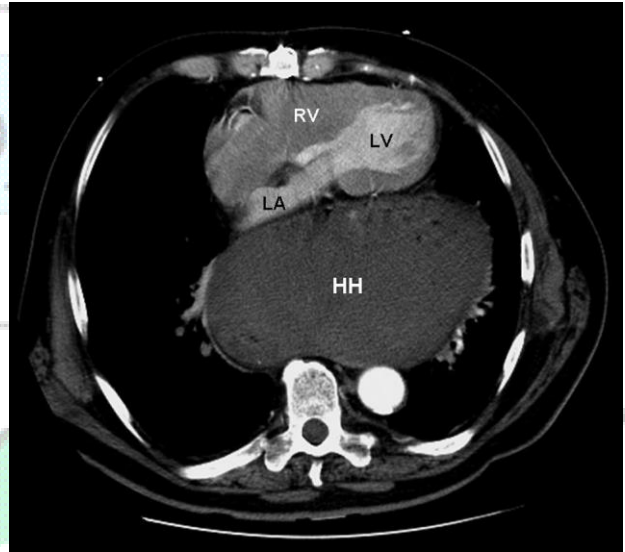


Figure 2. Chest computed tomography demonstrating the hiatal hernia (HH) compressing cardiac structures: left atrium (LA), left ventricle (LV), and right ventricle (RV).

and lactate of 2.92 mg/dL (0.6 to 2.3 mmol/L). Initial troponin I was 0.02 ng/mL (0.01 ng/mL). A telemetry alarm alerted for ST-segment elevation (STE), and ECG revealed prominent anterior STE (Figure 3). The patient denied dyspnea or chest pain. Intravenous heparin was initiated and aspirin therapy was continued. Emergency bedside transthoracic echocardiogram demonstrated left ventricular geometric distortion with asynchronous contraction pattern and hyperdynamic left ventricular function with ejection fraction of 70% without regional wall motion abnormalities (Movie I in the online-only Data Supplement). Serial troponin assessment remained flat without significant delta. Serial ECG assessment revealed persistent STE, and the patient remained asymptomatic. Repeat transthoracic echocardiogram was unchanged. Heparin was discontinued. An upper endoscopy was performed with suctioning of a large amount of retained liquid. The examination demonstrated a massive hiatal hernia with a large antral ulceration and smaller ulcerations within the gastric body and fundus. Selected ulcers were treated endoscopically, and intravenous proton pump inhibitor started. Thirty-six hours after admission and after stomach

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The online-only Data Supplement is available with this article at <http://circ.ahajournals.org/cgi/content/full/123/9/e259/DC1>.

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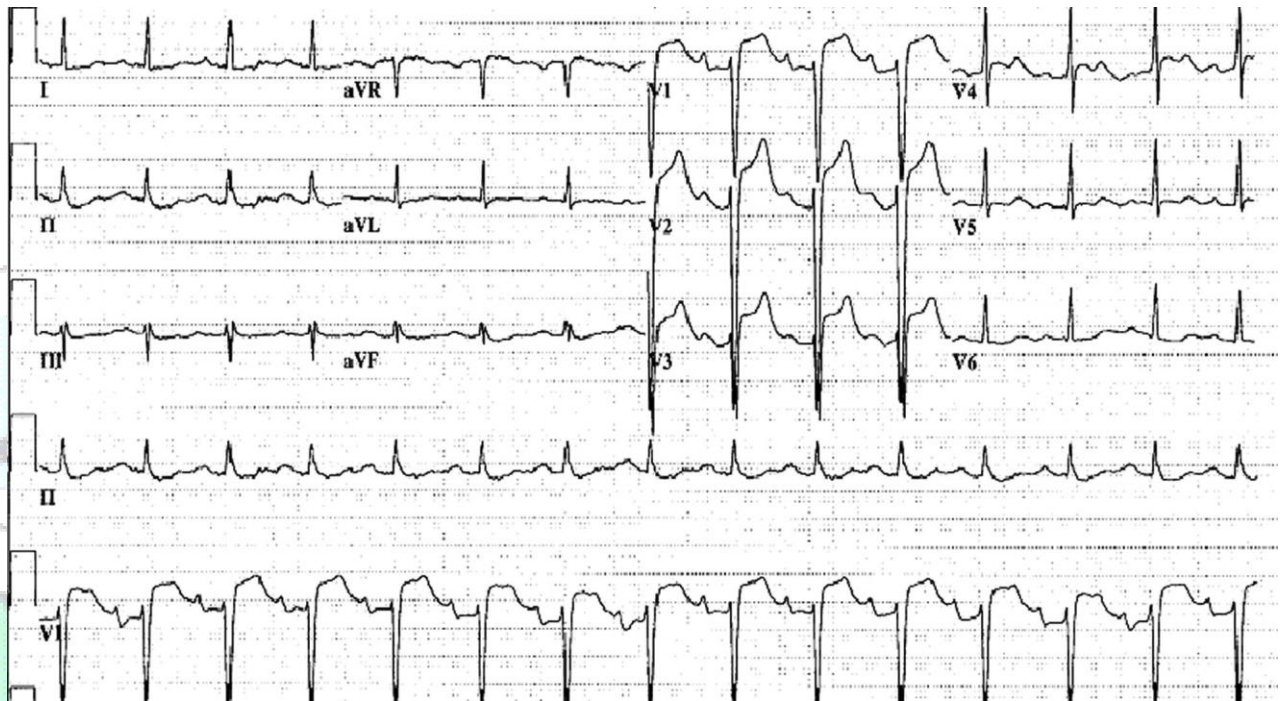


Figure 3. ECG demonstrating 5-mm anterior ST elevation (V₁ through V₃) and less prominent elevation in V₄.

decompression, his electrographic changes completely resolved (Figure 4), and the upper sternal lift was no longer present. The patient subsequently underwent laparoscopic hiatal hernia repair, wedge gastropasty, and Nissen fundoplication. His postoperative course was uneventful.

Discussion

STE myocardial infarction is a life-threatening emergency with outcomes depending on timely revascularization by

thrombolytics or percutaneous coronary intervention. The ECG is an essential component of its diagnosis. If the clinical presentation is consistent with acute coronary syndrome, the presence or absence of STE directs the treatment approach. Thus, proper interpretation of the ECG is fundamental. Echocardiographic wall motion assessment can increase the diagnostic specificity in selected cases that remain unclear because wall motion abnormalities are a later occurrence within the ischemic cascade.¹ However, this must be balanced

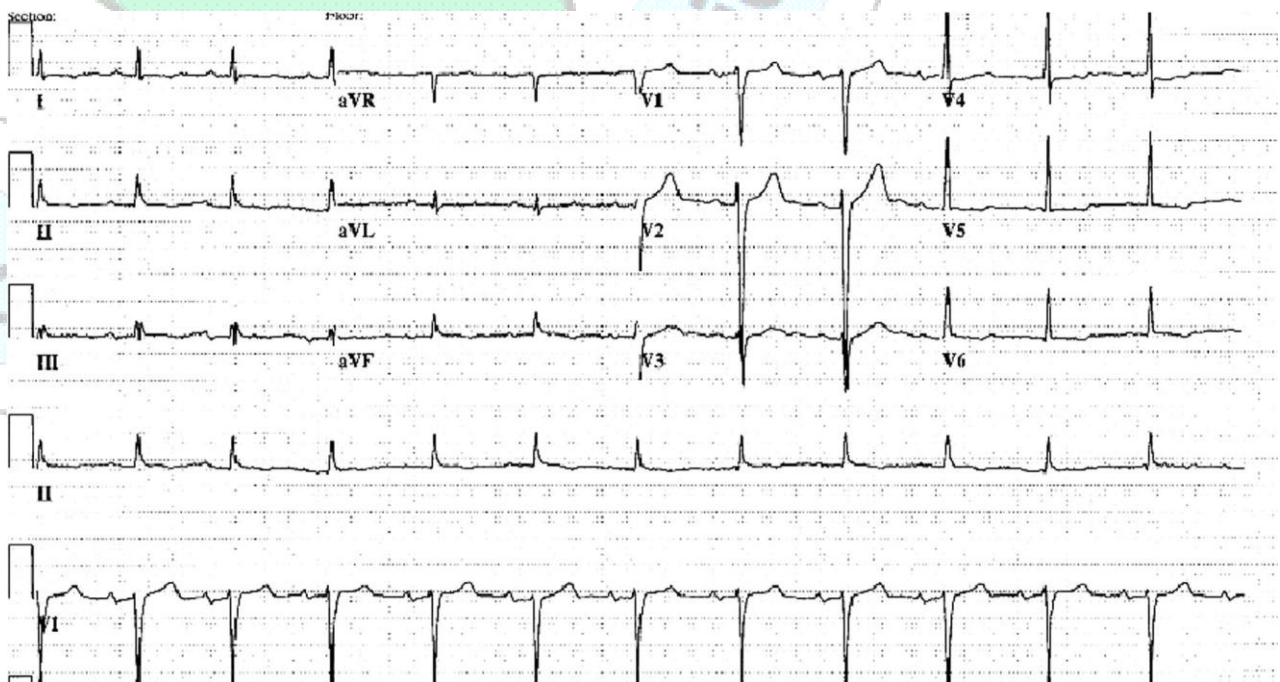


Figure 4. ECG after an upper endoscopy shows resolution of the STE and lower QRS voltage.

against the time delay necessary to obtain a transthoracic echocardiogram and the potential ongoing myocardial injury. The dissimilar character of the symptoms compared with previous angina and the presence of a large hiatal hernia with volvulus suggested that our patient's epigastric symptoms were related to 1 pathological process. However, abrupt 5-mm precordial STE with a "positive first troponin" and known coronary disease posed an important diagnostic challenge because 2 different pathological processes can coexist, and epigastric symptoms are not uncommon in STE myocardial infarction. Thus, concern for left internal mammary artery compression or primary epicardial vessel thrombosis was real. The contour of the ST segment, however, was concave upward rather than the convex "tombstone" pattern typically observed in STE myocardial infarction. Furthermore, there was no typical reciprocal ST depression in the inferior leads except for a scooped-out ST appearance with flattened T waves, particularly in lead II. Repeat transthoracic echocardiogram confirmed no acute wall motion abnormality, and we speculate that STE was related to cardiac compression, particularly because it resolved completely after endoscopic stomach decompression. Mild troponin elevation without significant change on serial measurements suggests his chronic kidney disease as the underlying cause of the biomarker abnormality.

The differential diagnosis of STE includes STE myocardial infarction but also coronary artery spasm, acute pericarditis,

J-point elevation, left ventricular hypertrophy, myocarditis, Brugada syndrome, hyperkalemia, pulmonary embolism, and repolarization abnormalities in the setting of bundle-branch blocks.² STE has also been reported in various intra-abdominal conditions, including acute pancreatitis,³ and in a woman with suspected pericarditis in the setting of a large hiatal hernia in whom STE developed after stomach decompression.⁴ In our case, the STE resolved after stomach decompression, suggesting that cardiac compression was likely the causative factor. Hiatal hernia is thus a condition to be considered in the differential diagnosis of STE.

Disclosures

None.

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