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CASE REPORT | STOMACH

Hiatal Hernia Presenting with Recurrent Non-ST Elevation Myocardial Infarction and Cardiac Tamponade

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ABSTRACT

Hiatal hernia is a common pathology, particularly among the elderly or obese populations. Occasionally, markedly dilated hernias can impinge on surrounding structures, notably the heart or lung. In such cases, morbidity can be considerable. We present a case of an enlarging hiatal hernia that compressed the heart, leading to recurrent non-ST elevation myocardial infarction with cardiac tamponade. The patient was successfully managed with nasogastric decompression and surgical repair. We recommend that extrapericardial pathology be considered in tamponade patients with concurrent hiatal hernia and that surgery should be considered the definitive treatment modality.

INTRODUCTION

Hiatal hernia is frequently encountered by gastroenterologists, typically as an incidental finding. Associated gastroesophageal reflux disease may be managed with proton pump inhibitor (PPI) therapy, although resistant cases may require fundoplication repair of the hernia.[1] We describe an unusual case of a patient with paraesophageal hiatal hernia presenting with recurrent non-ST elevation myocardial infarction (NSTEMI) and cardiac tamponade secondary to extrinsic cardiac compression by the hernia. After the comprehensive clinical and radiologic evaluation, she underwent nasogastric tube (NGT) decompression of the stomach, which resolved the tamponade. The hernia was subsequently surgically repaired.

CASE REPORT

An 87-year-old woman presented to the emergency department with severe unremitting substernal chest pain of several hours duration, which radiated to her back. The pain was associated with nausea and dysphagia and was unrelieved by nitroglycerin and aspirin. The patient reported being asymptomatic from chest pain at her baseline, except for intermittent reflux episodes, and was experiencing no symptoms of chronic heart failure. Two years previously, she was hospitalized with an episode of chest pain and elevated troponin (peaking at 1.3 ng/mL) caused by cardiac compression secondary to a large paraesophageal hiatal hernia. During that episode, she underwent nasogastric decompression of the dilated hernia but declined surgical repair.

On this admission, the physical examination was significant for heart rate 100 beats/min and blood pressure 90/61 mm Hg in the setting of nitroglycerin administration, with rapid spontaneous improvement to 130/88 mm Hg. All other vital signs were within normal limits. The body mass index was 21 kg/m^2. Her abdomen was soft, nondistended, and non-tender with normal bowel sounds in all quadrants. A holosystolic murmur was auscultated throughout the precordium, loudest at the right upper sternal border. Jugular venous pressure was elevated to 4 cm above the sternal angle. Lungs were clear bilaterally.

Electrocardiogram showed ST segment depression in leads V5 and V6. Serial laboratory readings showed raised troponin peaking at 2.12 ng/mL and brain natriuretic peptide of 83 pg/mL, collectively indicating NSTEMI without heart failure. Echocardiography revealed a large hernia partially effacing the left atrium and left ventricular free wall, compressing the heart, and causing tamponade

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physiology. Bedside ultrasound demonstrated an absence of pericardial effusion or abdominal aortic pathology. Thoracic computed tomography without contrast revealed a large hiatal hernia containing nearly the entirety of the stomach with mass effect on the heart (Figure 1). Gastric volvulus was not observed. Significant gastric dilation was visible on the chest x-ray (Figure 2).

A NGT was placed, and urgent gastric decompression was performed to relieve pressure on the heart and avert impending hemodynamic instability. NGT insertion was challenging owing to the presence of a massive hiatal hernia and required 6 attempts. The following day, the patient reported her symptoms had alleviated. In total, she underwent 2 days of continuous wall suction, which resolved her ST segment depression. The NGT was removed in the setting of a diminished output and clinical improvement. Postdecompression laboratory readings showed downtrending troponin values. Repeat imaging was not performed.

The patient subsequently consented to definitive repair of the hernia. The surgical risk was evaluated using diagnostic cardiac catheterization, which indicated stable coronary disease and was judged to be acceptable based on this workup. The patient underwent a robotic paraesophageal hernia repair with mesh and Toupet fundoplication. The postoperative course was uneventful, with the patient reporting no recurrence of chest pain. Troponin values returned to normal, and electrocardiogram findings remained at baseline. She was discharged with a cardiology follow-up 2 days later. Cardiac (aspirin, statin, antihypertensive) and gastroprotective PPI medication regimens were continued.

DISCUSSION

Hiatal hernias can present across a spectrum of disease severity, with the majority either undetected or incidentally discovered.

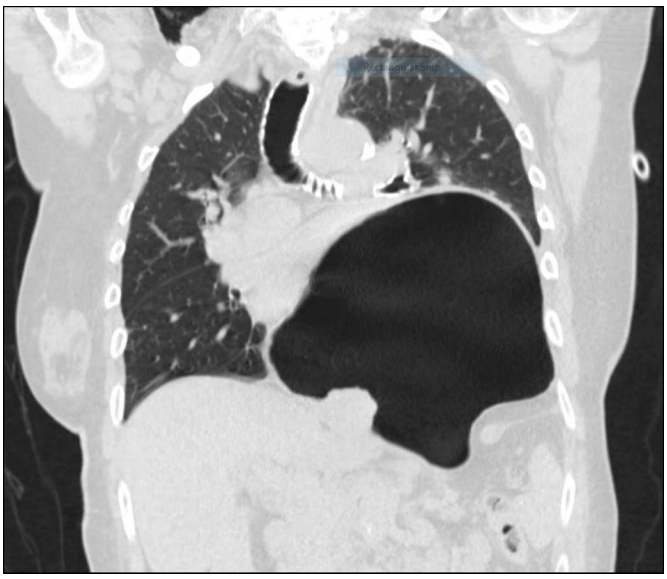

Figure 1. Computed tomography scan on admission showing dilation of the stomach with compression of the heart.

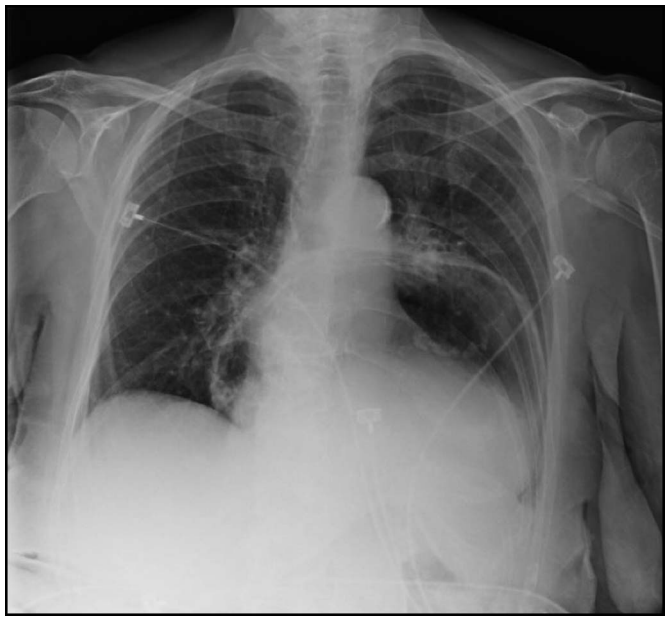

Figure 2. Chest x-ray on admission showing dilation of the stomach.

In patients who present with reflux symptoms, prescription of PPIs for symptomatic relief and regular screening for dysplastic changes are essential components of management.[2] However, complications can arise because of mass effects from the displaced portion of the stomach, for example, on the heart; in such cases, the burden of morbidity can be considerable, and surgical intervention is warranted. Toupet fundoplication is associated with lower reported rates of postoperative dysphagia and is similarly effective in controlling reflux compared with Nissen fundoplication, hence the decision to undertake the former procedure.[3]

Cardiac compression is a recognized complication after hiatal hernia repair.[4,5] Immediate postoperative hernia recurrence or hematoma formation within the hernial sac, secondary to trauma during surgery, can cause mass effects leading to a cardiac compromise.[6] Tamponade can also arise as a consequence of direct injury to the heart or pericardium during hernia repair.[7] This prompted a recent review of the surgical technique with a view to reducing the occurrence of life-threatening tamponade.[8] The authors report an association between the use of tacks in the diaphragm and increased incidence of penetrating cardiac complications during hernia repair and caution surgeons against this fixation approach. Coronary artery bypass surgery has also been associated with tamponade in the setting of pre-existing hiatal hernia[9,10]—this likely relates to gastric dilation, a common finding in the early postoperative period after cardiac procedures, although rarely symptomatic.[11,12] The impact of large hernias on ventilation has also been characterized—exertional dyspnea was identified in 83% of patients with hiatal hernia, despite intrinsically normal pulmonary function, and significantly improved after surgical repair.[13] Sahin et al described a case of postprandial dyspnea because of pulmonary congestion

caused by left atrial and right pulmonary vein compression by a large hiatal hernia.[14] In a report that parallels our case, Schummer described a patient with previously undiagnosed large hiatal hernia that led to an isolated episode of NSTEMI.[15] There has also been a previous report of strangulated gastric volvulus leading to cardiac compression with electromechanical dissociation in a patient with previously demonstrated rolling hiatal hernia.[16]

We describe a case of cardiac tamponade associated with progressively enlarging, long-standing hiatal hernia, in the absence of preceding intra-abdominal/intrathoracic intervention. The patient's initial decision to opt against surgical repair permitted a recurrence of hernial dilation, which culminated in a repeat episode of symptomatic cardiac compression. To our knowledge, this is the first report of recurrent cardiac compression from a large hiatal hernia and emphasizes that in the patient with acceptable surgical risk, surgical repair is the definitive treatment.

DISCLOSURES

Author contributions: All authors contributed equally to this manuscript. A. Arvind is the article guarantor.

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Informed consent was obtained for this case report.

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