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# The Unexpected Squeeze: When a Hiatus Hernia Induces Cardiac Tamponade and Causes a Myocardial Infarction

**Rashed AlBanna*, Aysha Husain, Ahmed Alhalwachi and Rami Gabani**

Cardiology Department, Salmaniya Medical Complex, Manama, Bahrain

***Corresponding author:** Rashed AlBanna, Cardiology department, Salmaniya Medical Complex, Manama, Bahrain.
E-mail: RAbdulla1@hospitals.gov.bh

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## Abstract

**Background:** Hiatus hernia is linked to cardiac complications such as pulmonary edema and postprandial syncope. In severe cases, extracardiac compression can simulate cardiac tamponade physiology, impairing diastolic ventricular filling and reducing coronary perfusion pressure, potentially precipitating myocardial ischemia.

**Case Summary:** We present a case of a large hiatus hernia presenting with acute coronary syndrome without coronary atherosclerosis. The treatment strategy was to decompress the hernia and augment intravascular fluid volume.

**Discussion:** Compression from a large hiatus hernia can cause tamponade physiology, as documented in the literature following cardiac surgery. Elevated left ventricular filling pressure during tamponade leads to ischemia, progressing from the subendocardium to the subepicardium. In this case, sepsis and hypotension further reduced blood flow, worsening ischemia. This case highlights a rare cardiac complication of hiatus hernia and emphasizes the importance of understanding its pathophysiology to effectively address treatment challenges.

**Keywords:** Hiatus hernia; Myocardial infarction; Cardiogenic shock; Tamponade

## Introduction

Hiatus hernia is linked to cardiac complications such as pulmonary edema and postprandial syncope. In severe cases, extracardiac compression can simulate cardiac tamponade physiology, impairing diastolic ventricular filling and reducing coronary perfusion pressure, precipitating myocardial ischemia.

## Case Presentation

A woman in her sixties presented with a three-day history of progressive shortness of breath and central chest pain radiating to the left arm. The pain was associated with palpitations and paroxysmal episodes of shortness of breath, partially alleviated with oral analgesics and proton pump inhibitors. The patient denied ankle swelling or claudication. She was noticed by her daughter to be drowsy and confused on the day of her admission.

She also experienced nausea with frequent emesis, intermittent fevers and burning micturition, which was attributed to recurrent urinary tract infections. On initial presentation, her hemodynamics were significant for profound hypotension with a blood pressure 70/40 mmHg. Tachycardia was present, with a heart rate of 110 beats per minute. Her temperature was within normal limits. Clinical assessment revealed signs of distress, in addition to asthenia and dulled sensorium. Precordial auscultation was unremarkable. Respiratory examination revealed diminished breath sounds on the left lung base, with prominent bowel sounds auscultated in the same region.

Past medical history was significant for hypothyroidism managed with thyroxine replacement, with no documented diabetes or essential hypertension. Notably, she had a history of recurrent abdominal pain requiring multiple laparotomies, the most recent was three years prior for lysis of adhesions. The patient was an active smoker, widowed, and mother of two sons and one daughter.

The initial diagnostic evaluation included an electrocardiogram which revealed sinus tachycardia with mild ST segment depression in multiple leads (Figure 1).

A chest X-ray revealed a cardiothoracic ratio within normal limits. A large retrocardiac opacity with an air-fluid level was identified (Figure 2). An echocardiogram revealed normal left ventricular systolic function, with no overt regional wall motion abnormalities or pericardial effusion.

An urgent CT pulmonary angiogram was negative for significant pulmonary artery filling defects suggestive of acute pulmonary embolism. Of note, a large hiatal hernia was visualized. This hernia involved herniation of the gastroesophageal junction, stomach, segments of small and large intestines, and mesenteric fat into the thoracic cavity (Figure 3).

The relevant blood results at presentation and discharge are summarized in Table 1.

**Table 1:** Laboratory data on admission and on discharge.

| Laboratory parameter | On admission | On discharge |
|---|---|---|
| Troponin I | 10.3 ng/mL with normal range <1.5 ng/mL | 0.060 ng/mL |
| Brain Natriuretic peptide | 321.4 pmol/L normal range of <47.7 pmol/L | 11 pmol/L |
| C Reactive protein | 124.40 mg/L (Normal range 0 – 3 mg/L) | 12.3 mg/L |
| Procalcitonin | procalcitonin 21.41 µg/L (Normal range <0.5 µg/L) | 0.34 µg/L |
| Total White blood cell count | 23.67 x 10^9/L | 6.80 x 10^9/L |
| Platelet count | 80 x 10^9/L | 327 x 10^9/L |
| Prothrombin time | 16.60 s | 12.10 s |

Midstream urine analysis revealed pyuria and hematuria with a positive nitrite test, suggestive of a urinary tract infection. Subsequent urine and blood cultures confirmed the presence of Escherichia coli.

Based on the initial clinical presentation, the provisional diagnosis was acute non-ST-elevation myocardial infarction (NSTEMI) complicated by cardiogenic shock. Consequently, she was admitted to the coronary care unit (CCU) and managed with bed rest, dual antiplatelet therapy, and heparin infusion. Inotropic support and intravascular volume repletion with normal saline were initiated and titrated based on hemodynamic parameters. Given the positive urine culture and clinical picture, broad-spectrum intravenous antibiotics were commenced and adjusted based on culture sensitivity results.

The surgical team recommended gastric decompression via nasogastric tube. Her clinical course showed progressive improvement: decreasing dyspnea, resolving chest pain, normalization of tachycardia and blood pressure, and adequate diuresis. She remained afebrile throughout her admission. Serial electrocardiograms demonstrated ST segment normalization and progressive decline in cardiac troponin and brain natriuretic peptide levels (Figure 1). Additionally, her inflammatory and coagulation parameters normalized.

By the fifth post-admission day, she was transferred out of the CCU and gradual mobilization was initiated. On the tenth day, she was discharged home with recommendations for continued dual antiplatelet therapy for three months, followed by life-long aspirin and statin therapy. To complete the 14-day treatment course as advised by infectious disease services, oral antibiotics were prescribed.

A cardiac computed tomography angiography (CCTA) scan was preformed after discharge and revealed normal coronaries and a total calcium score of zero.

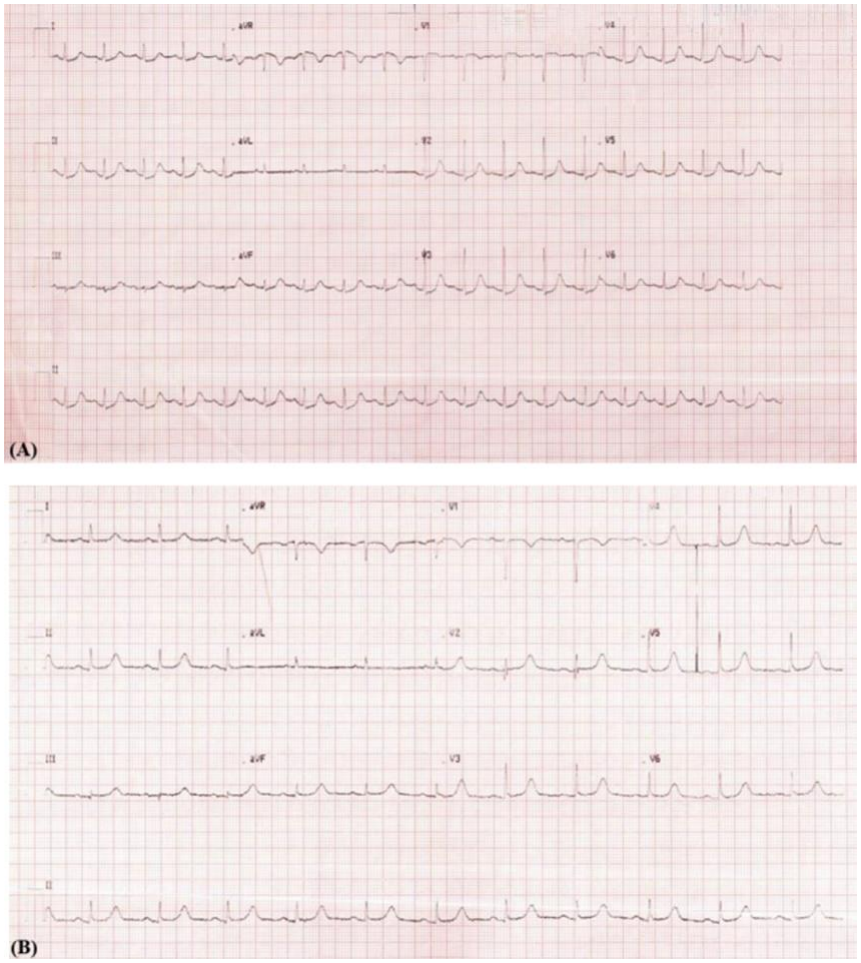


**Figure 1:** ECG of the patient.

**(A):** ECG on admission. The ECG shows sinus tachycardia with 1mm ST segment depression in leads V2-V6, I, II and aVF and 1mm ST elevation in aVR.

**(B):** ECG before discharge showed resolution of the ischemic changes.

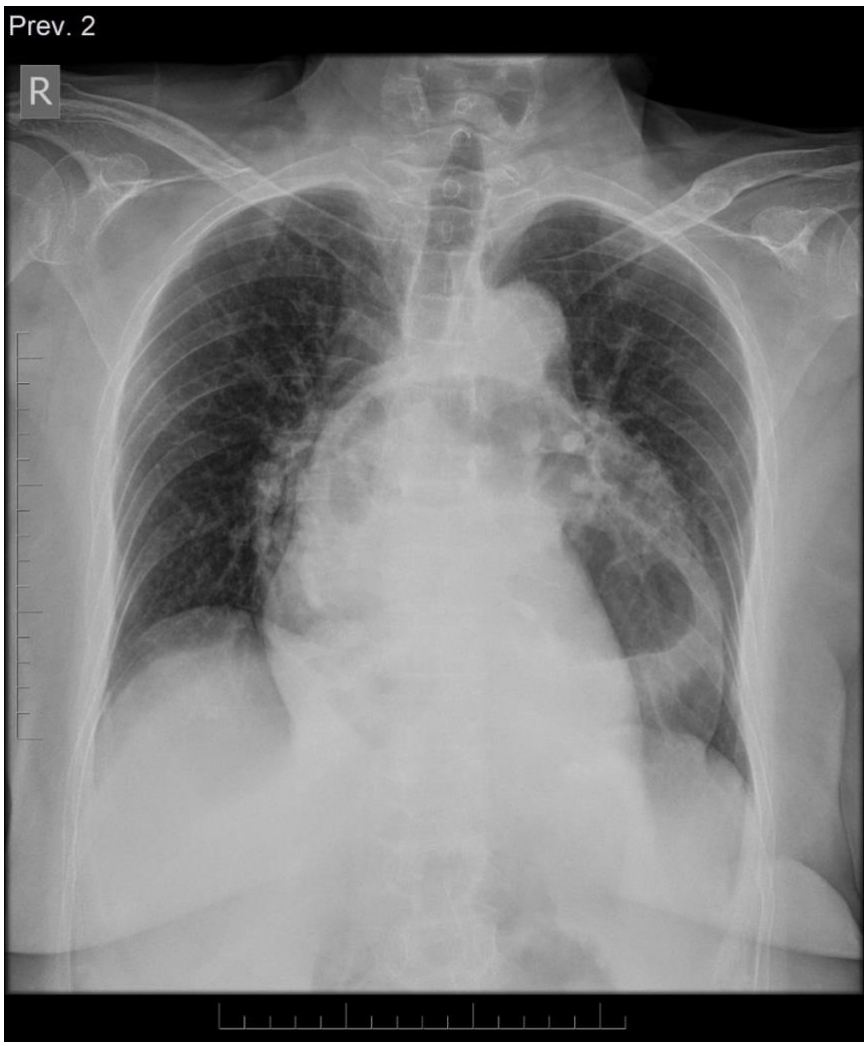


**Figure 2:** Chest X-ray on admission.

Chest X-ray shows a large round retrocardiac opacity with an air-fluid level.

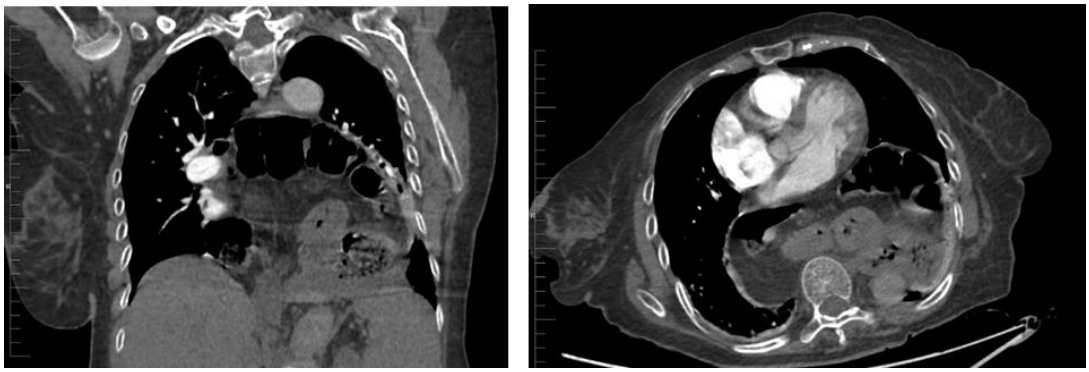

**Figure 3:** Computerized tomography (CT) of the chest.

Coronal and axial CT scan view of the chest demonstrating a large paraesophageal hernia in the thoracic cavity containing bowel loops and abutting the heart.

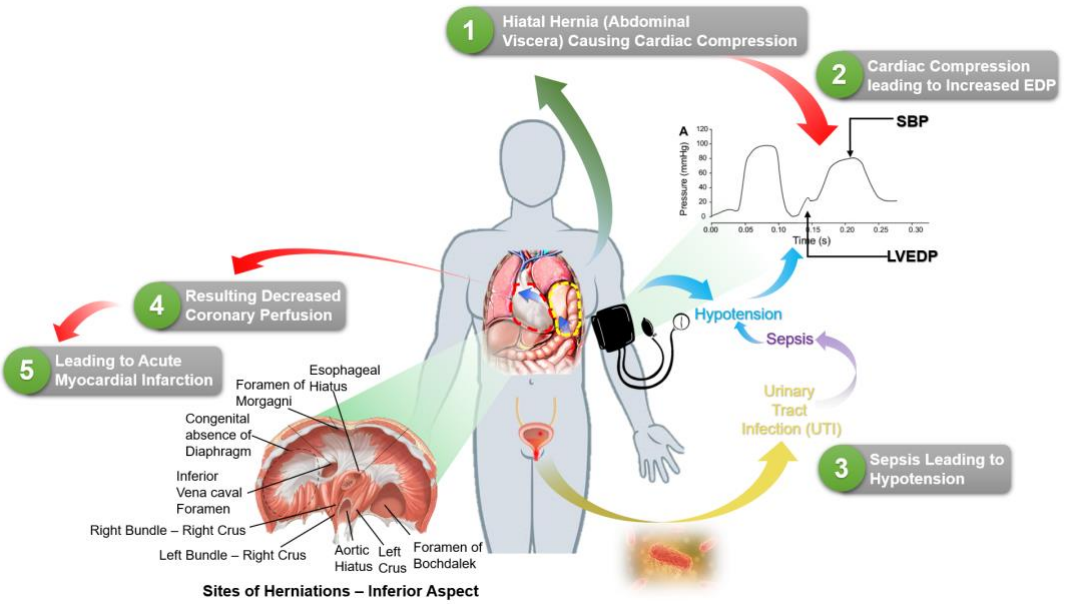


**Figure 4:** Mechanisms linking hiatal hernia and acute myocardial infarction in the patient.

This figure depicts the pathophysiological mechanisms by which a hiatal hernia, through cardiac compression and tamponade physiology, coupled with sepsis and resultant hypotension, can lead to decreased coronary perfusion, culminating in acute myocardial infarction.

## Discussion

Hiatus hernia is a condition characterized by herniation of abdominal contents, primarily the stomach, through the diaphragm into the mediastinum [1]. The literature highlights several cardiac complications associated with hiatal hernia. These include acute pulmonary edema, attributed to elevated pulmonary capillary wedge pressure during recumbency, potentially caused by sac compression of the left atrium by the herniated contents [2]. Electrocardiographic changes that can mimic ST elevation myocardial infarction have been well reported [3]. Ventricular tachycardia of unclear pathogenesis has also been described [4]. Postprandial syncope can occur secondary to left atrial obstruction by a large hernia [5]. While pericardial fluid accumulation is the most common cause of cardiac tamponade, any mass effect on the heart can lead to similar hemodynamic alterations [6]. Extracardiac compression mimicking cardiac tamponade can be one of the rare manifestations of a large hiatus hernia, particularly post-cardiac surgery [7]. In this case, the patient's acute coronary ischemia was attributed to cardiac tamponade physiology induced by the large hiatal hernia (Figure 4). During the early stages of tamponade, a significant increase in cardiac contractility occurs due to augmentation of intrinsic sympathetic state, a compensatory mechanism to impaired diastolic filling. The impaired diastolic filling and elevations in LV filling pressure will lead to disproportionate decrease in subendocardial blood flow compared with subepicardial flow. In late tamponade, however, there is a further reduction in coronary blood flow, resulting from a proportionately similar decrease in blood flow to both the subendocardium and the subepicardium. This in turn leads to a large decrease in contractility [8].

In summary, failure to restore normal intrapericardial pressure will perpetuate myocardial ischemia through a cascade of events:

**1.** Reduced coronary perfusion pressure due to hypotension and elevated filling pressure.

**2.** Impaired subendocardial perfusion due to aforementioned pressure-dependent distribution changes.

**3.** Increased oxygen demand from compensatory efforts.

**4.** Potential neurohormonal activation further burdening the already stressed myocardium.

This vicious cycle can lead to irreversible damage if not promptly addressed [9]. This aligns with the reported case of pulseless electrical activity associated with hiatal hernia [10].

In our case, the patient presented with hypotension, likely exacerbated by an active septic process. Subsequent decompression of the hernia sac and intravascular volume augmentation resulted in hemodynamic restoration, normalization of electrocardiographic changes, and a decline in cardiac marker levels. Moreover, cardiac computed tomography angiography (CCTA) revealed an absence of significant underlying atherosclerotic burden. This constellation of findings strongly suggests acute type 2 myocardial infarction secondary to acute cardiac tamponade, which compromised myocardial perfusion by impairing the delicate balance between oxygen demand and supply.

On follow up the patient continued to exhibit symptoms attributed to her hiatus hernia, with no recurrence of cardiovascular life-threatening events. Both aspirin and statins were discontinued. Surgical repair of the hiatal hernia was recommended.

## Conclusion

This case report illustrates a rare cardiac complication associated with hiatus hernia. The initial presentation of chest pain, hypotension, and elevated cardiac markers suggested acute non-ST-segment Elevation Myocardial Infarction (NSTEMI) with cardiogenic shock. However, the subtle electrocardiogram changes and absence of apparent regional wall motion abnormalities on echocardiography raised suspicion of alternative etiologies contributing to the clinical picture.

One hypothesis is that the hypotension associated with septic shock could have precipitated coronary ischemia in the presence of occult coronary artery disease. However, after evaluating the coronary anatomy and confirming the presence of a grade 4 hiatus hernia, the latter emerged as the more likely explanation for the myocardial infarction. The literature supports the potential for hiatal hernia to induce cardiac complications through a mechanism akin to cardiac tamponade.

In this scenario, the low diastolic pressure and elevated left ventricular filling pressure (evidenced by elevated BNP) likely contributed to a further reduction in coronary perfusion pressure, leading to myocardial ischemia. Additionally, the literature suggests that hiatal hernia can manifest as supraventricular arrhythmias and pulseless electrical activity, both characteristic presentations of pericardial tamponade. This case supports the potential extrapolation of tamponade physiology to explain the observed clinical features. This case also highlights the importance of considering atypical causes of cardiac presentations, especially in individuals with existing anatomical predispositions such as hiatal hernia.

## Abbreviations

**NSTEMI:** Non-ST-elevation myocardial infarction; **CCU:** Coronary care unit; **BNP:** Brain natriuretic peptide; **CCTA:** Cardiac computed tomography angiography

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## REFERENCES

1. Roman S, Kahrilas PJ. The diagnosis and management of hiatus hernia. BMJ. 2014; 349: 6154.
2. Siu C-W, Jim M-H, Ho H-H, et al. Recurrent acute heart failure caused by sliding hiatus hernia. Postgrad Med J. 2005; 81: 268-269.
3. Gard JJ, Bader W, Enriquez-Sarano M, et al. Uncommon cause of ST elevation. Circulation. 2011; 123.
4. Gnanenthiran SR, Naoum C, Kilborn MJ, et al. Posterior cardiac compression from a large hiatal hernia-A novel cause of ventricular tachycardia. HeartRhythm Case Rep. 2018; 4: 362-366.
5. Vanerio G. Syncope caused by huge hiatal hernia. Case Rep Cardiol. 2011; 2011: 560734.
6. McGuire SS, Thaden JJ, Goyal DG, et al. Cardiac tamponade caused by gastrointestinal obstruction: A rare complication in esophagectomy patients. CASE. 2022; 6: 173-177.
7. Papoulidis P, Beatty JW, Dandekar U. Hiatal hernia causing extrapericardial tamponade after coronary bypass surgery. Interact Cardiovasc Thorac Surg. 2014; 19: 716-717.
8. Wechsler AS, Auerbach BJ, Graham TC, et al. Distribution of intramyocardial blood flow during pericardial tamponade. Correlation with microscopic anatomy and intrinsic myocardial contractility. J Thorac Cardiovasc Surg. 1974; 68: 847-856.
9. Heward SJ, Widrich J. Coronary Perfusion Pressure. StatPearls Publishing. 2023.
10. Ohira S, Okada Y, Ishida Y, et al. Cardiopulmonary arrest secondary to compression of the heart owing to esophageal hiatal hernia: a case report. Acute Med Surg. 2020; 7.