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# **Left atrial compression by a large hiatal hernia: a rare cause of cardiac dysfunction**

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# **Left atrial compression by a large hiatal hernia: a rare cause of cardiac dysfunction**

## **Abstract**

Symptomatic hiatal hernia (HH) is most often revealed by gastroesophageal reflux disease, but there are atypical presentations some of which are life-threatening. We report the case of a 57-year-old woman brought to the emergency department with isolated shortness of breath for 24 hours. Initial explorations revealed unexplained hyperlactatemia (6.4 mmol/L) without clinical or biological evidence of hypovolemia, distributive, obstructive or cardiogenic shock. Two hours after admission, we observed a decreased of blood pressure and an increase of lactate level to 7.9 mmol/L. A bedside echocardiography revealed an extra-cardiac left atrial compression and thoracoabdominal computed tomography showed a large sliding HH compressing the left atrium. After an upper gastrointestinal endoscopy permitting the aspiration of gastric contents, a repair surgery was performed without complications and patient was discharge three days later. Emergency physicians should be aware that HH can be a rare cause of cardiac symptoms by heart compression and certainly use echocardiography for unexplained hemodynamic failure.

## **Keywords**

Hiatal hernia; Heart failure; Left atrium; Compression; Shortness of breath

## **1. Introduction**

Hiatal hernia (HH) is defined as the protrusion of abdominal organs, most often the stomach, into the mediastinum through the diaphragmatic esophageal hiatus. Its prevalence is unknown and difficult to determine as HH remains asymptomatic in most situations. The main risk factors for HH are older age and obesity. Even when symptomatic, HH can be difficult to

diagnose due to its various clinical presentations. The most common symptom is gastroesophageal reflux (heartburn, regurgitation) but there are other rarer signs such as epigastric discomfort, chest pain or anemia [1, 2]. There are also atypical presentations some of which can be life threatening. We hereby report the case of a woman with cardiac dysfunction due to a left atrial compression by a large HH.

## **2. Case presentation**

A 57-year-old woman is brought to the emergency department (ED) by a mobile intensive care unit with isolated shortness of breath for 24 hours. She reported no other symptoms, particularly no fever, cough, chest pain or digestive trouble. Her medical history included pulmonary embolism, esophagitis, obesity, intellectual disability and alcohol use disorder. Her significant medications were rivaroxaban, lansoprazole, baclofen and alimemazine. Because of tachypnea and wheezing on pulmonary auscultation, the initial management before her transport to the hospital was oxygen and nebulization of terbutaline and ipratropium. On ED presentation, the patient was tachycardic to 109 beats per min, mildly hypertensive (154/99 mmHg) and afebrile (36,2°C). Her respiratory rate (RR) was 36 breaths per min with diaphragmatic breathing and her saturation was 98 % on 2L/min oxygen. Pulmonary examination found decreased breath sounds at lung bases and wheezing. A first arterial blood gas (ABG) analysis revealed hyperlactatemia (6.4 mmol/L) with compensated acidosis (pH 7.45, PaCO<sub>2</sub> 24mmHg, PaO<sub>2</sub> 113mmHg, HCO<sub>3</sub> 17 mmol/L). Because this could have been a side effect of terbutaline, we decided a simple monitoring. Chest X-ray showed cardiomegaly without pleural or parenchymal abnormalities (Figure 1). Two hours after admission, as the patient spontaneously improved her breathing (RR 20 breaths per min), we observed a reduced blood pressure to 86/41mmHg, improving after a fluid bolus with 500ml of NaCl 0.9%. A second ABG analysis found an increase of lactate level to 7.9

mmol/L. Complete blood count was normal (including white blood cells count at  $7\,010/\text{mm}^3$ ), inflammatory markers (C-reactive protein and procalcitonin) were negative and cardiac enzymes (troponin, brain natriuretic peptide and D-dimer) were normal. A bedside echocardiography revealed an extra-cardiac left atrial compression, resulting in a thoracoabdominal computed tomography (CT). It showed a large sliding HH containing most of the stomach, measuring  $98 \times 170\text{mm}$  and compressing the left atrium (Figures 2 and 3). An upper gastrointestinal endoscopy (UGIE) allowed aspiration of gastric contents and found a Barrett's esophagus with no active bleeding. After this procedure, a second echocardiography showed the disappearance of left atrial compression. The next day, a surgical HH repair with Toupet fundoplication was performed, without complications. The patient was discharged three days later and had no complications at one month.

### **3. Discussion**

Symptomatic HH is most often revealed by a gastroesophageal reflux, but the diagnosis can be challenging in front of atypical symptoms. In our patient, this could have been suspected given the history of esophagitis and the presence of the two main risk factors: age above 50 years-old and obesity. Others risk factors are male sex, history of abdominal surgery or trauma and spinal deformities [1, 2]. HH is a rare cause of cardiac compression which can be revealed by various symptoms such as chest pain, dyspnea or syncope [3–5]. An anamnestic data to remember, these symptoms are often increased or triggered by exercise or ingestion of food. The prevalence of atrial fibrillation has been shown to be higher in patients with HH compared to the general population [6]. Paroxysmal ventricular tachycardia has also been reported as rare cases of ST-segment elevation acute coronary syndrome [7, 8]. One case of cardiac arrest has even been described [9]. Our patient was in shock with no obvious cause. There was no reason to suspect septic, hypovolemic or anaphylactic shock. In this situation,

when a cardiac or obstructive etiology is suspected, echocardiography appears to be a useful and reliable tool. Bedside echocardiography can rapidly inform the emergency physician on volume status, contractility, right chamber dilatation, pericardial effusion or other rarer causes of extra cardiac compression, as in our patient. In this last case, the diagnosis is specified by CT, followed by UGIE if necessary. The treatment of a compressive HH consists of gastric aspiration and sometimes a surgical repair. Outcome is most often favorable.

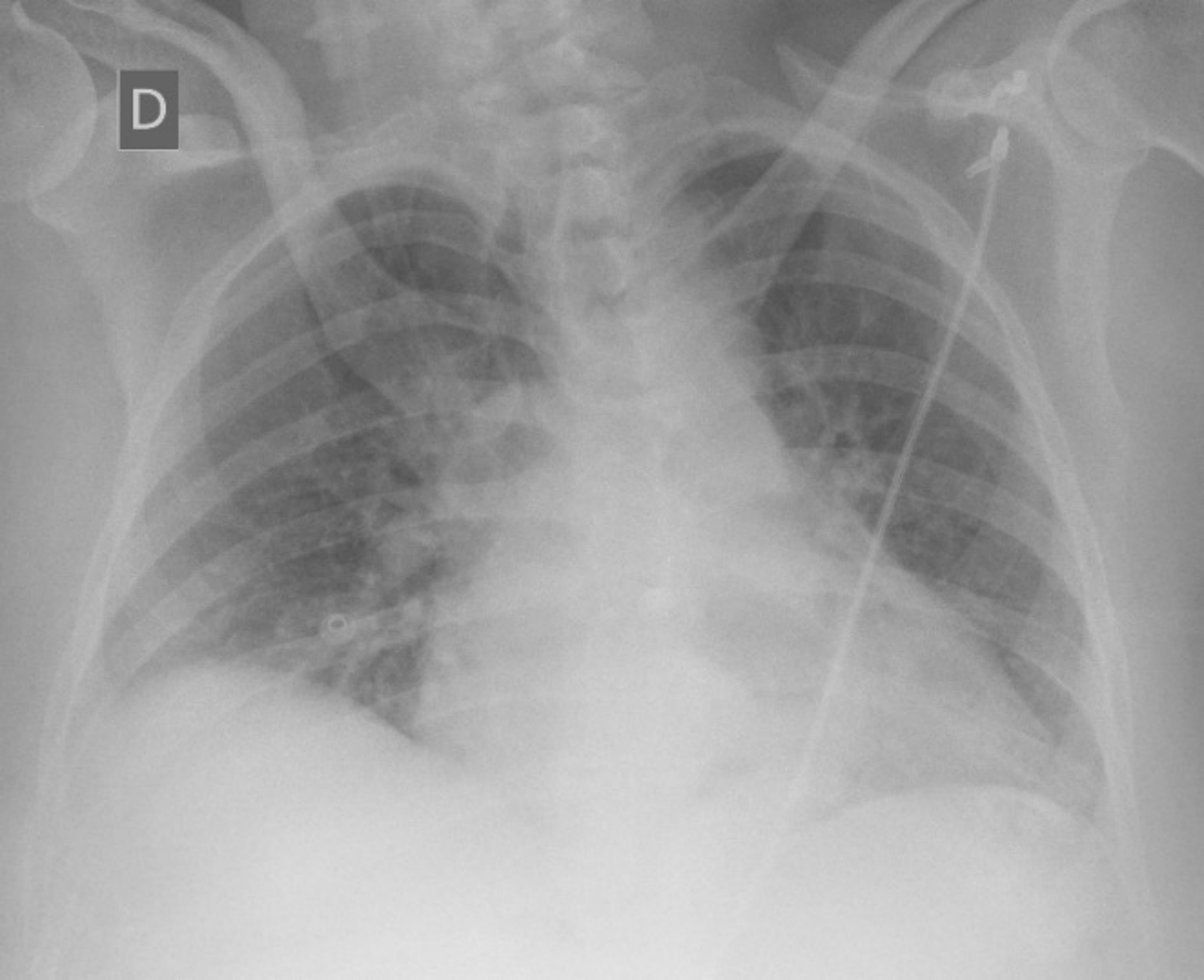
#### **4. Conclusion**

We have reported the case of a 57-year-old woman with heart failure due to left atrial compression by a large HH. Emergency physicians should be aware that HH can be a rare cause of cardiac symptoms and certainly use echocardiography for unexplained hemodynamic failure.

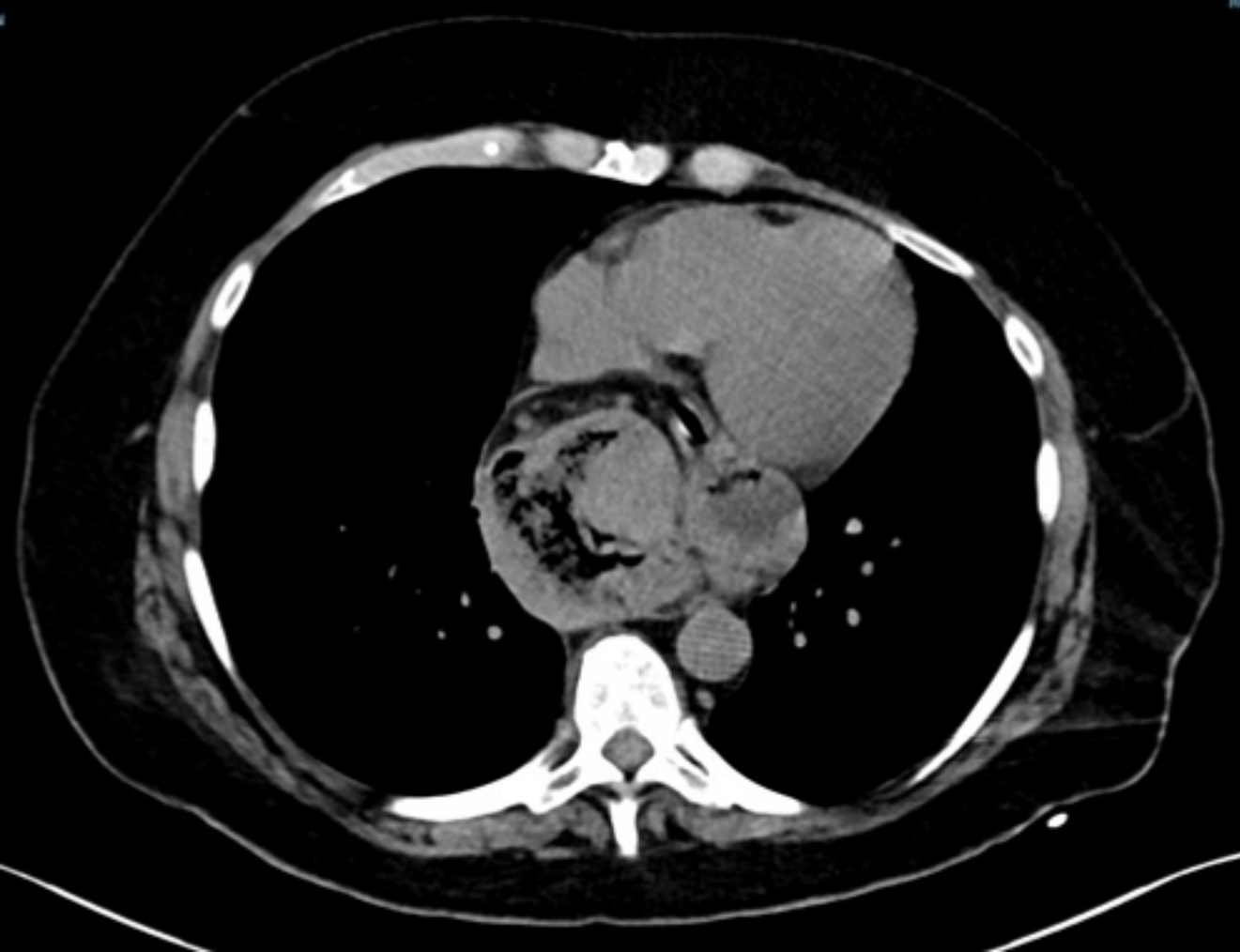
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## **Legends**

Figure 1. Chest X-ray: cardiomegaly without pleural or parenchymal abnormalities.

Figure 2. CT-scan, axial section: large hiatal hernia compressing the left atrium.

Figure 3. CT-scan, coronal section: large hiatal hernia compressing the left atrium.