



Cardiac Tamponade Caused by Gastrointestinal Obstruction— A Rare Complication in Esophagectomy Patients

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Abstract

Cardiac tamponade is diagnosed when fluid builds up within the pericardial sac, exerting pressure on the heart from the outside, reducing cardiac output and threatening the body's blood supply.¹ Despite the vague nature of the symptoms, patients typically report dyspnea and chest discomfort. ² Low blood pressure, fast heart rate, shallow breathing (tachypnea), elevated venous pressure in the jugular vein (pulsus paradoxus), and muffled heart sounds on cardiac auscultation are other clinical symptoms. But it's rare to be seen in post esophagectomy patients as a result of gastrointestinal Obstruction.

Keywords: Cardiac tamponade, Gastrointestinal obstruction, Esophagectomy tamponade-physiology, Echocardiogram, Point-of-care ultrasound.

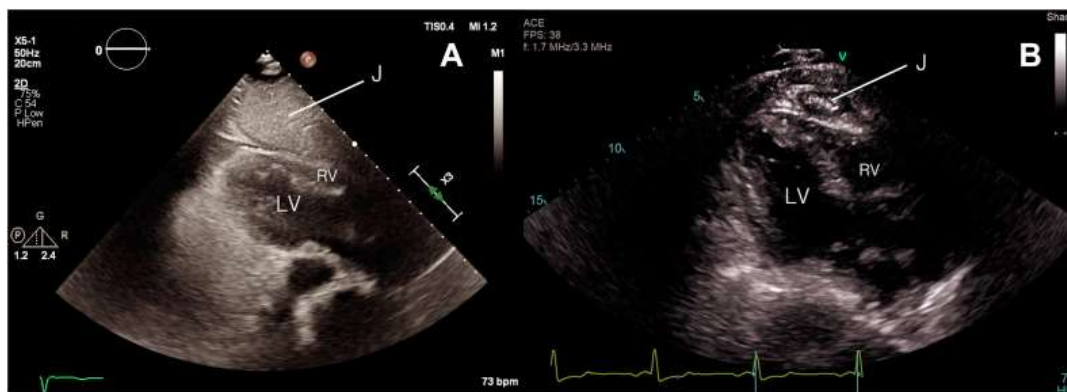
1. Introduction

The ability to quickly diagnose cardiac tamponade is essential for doctors treating very unwell patients. The optimal tool for quick evaluation and identification of tamponade in situations where it is thought in view of history or actual test is echocardiography at the mark of care, which may also be used during later pericardiocentesis. Pericardial effusion, pronounced respirophasic variation in in-flow velocities at mitral and tricuspid valves, invagination of the right atrial wall during systole and diastolic right ventricular breakdown, a plethoric sub-par vena cava (IVC) with insignificant respiratory variety, and diastolic hepatic vein stream inversion during lapse are all important echocardiographic findings.^{3,4}

Here, we report on a rare instance of cardiac tamponade in a patient who had just had an Ivor Lewis esophagectomy. Echocardiography revealed that he had a pericardial effusion, although the patient's clogged mediastinal gastrointestinal (GI) tract was the major tamponade mechanism.

2. Case Presentation

A 64-year-elderly person with a background marked by metastatic adenocarcinoma of the gastroesophageal intersection arrived to the ED with hypotension 3 years after his Ivor Lewis esophagectomy (Figure 1).



Seven days before to presenting to the ED, he had undergone CT chest angiography, which uncovered a critical (10-20 mm or 100-500 mL) pericardial emission, and he had a past filled with cardiovascular tamponade requiring crisis pericardiocentesis 10 months earlier. On the other hand, this result was considered chronic and stable based on earlier imaging. The patient experienced GI blockage for 3–4 days, and his jejunum in the mediastinum, which had been surgically rerouted, bulged visibly from his left upper neck during the physical examination. He had gone to the gastroenterology short term center for an upper GI endoscopy because of his blockage, but he was taken straight to the emergency department (ED) after his systolic blood pressure (SBP) was measured at 80 mm Hg without any decompression. The patient's SBP was 90 mm Hg yet he or she was not tachycardic when they arrived at the ED. After his surgeries, he had persistent dyspnea and chest discomfort, although they had not suddenly become worse. Urgently, a targeted, non-comprehensive transthoracic echocardiography (TTE) was done using a Philips EPIQ 7 ultrasound hardware at the place of care, and the outcomes showed a critical heterogeneous assortment in the foremost mediastinum that most probable compared to the patient's as of now known expanded jejunum.

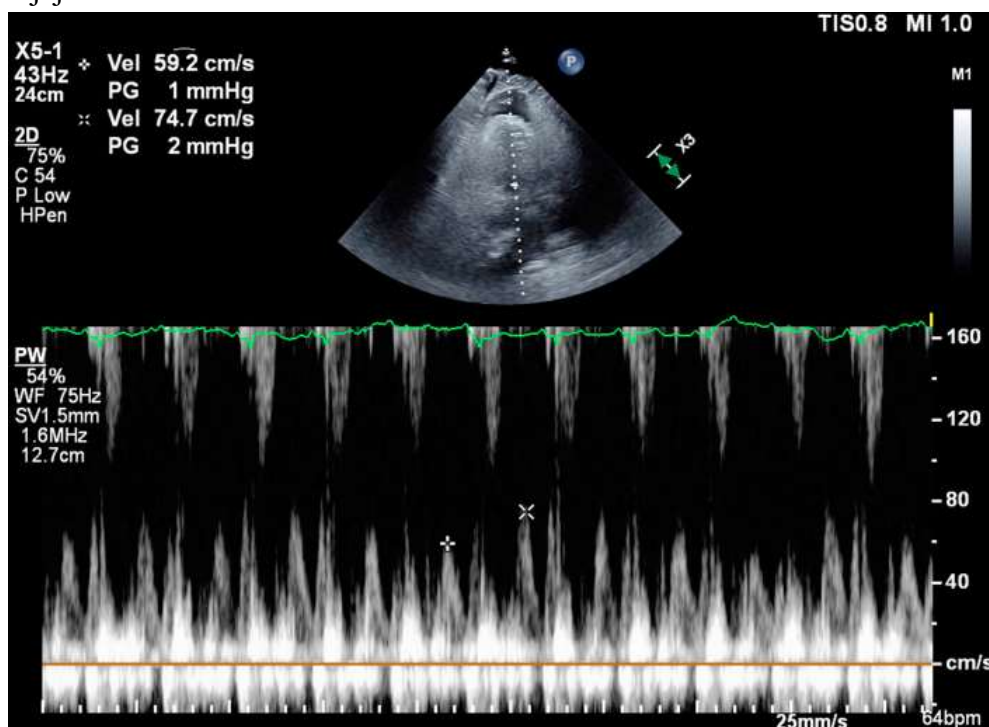


Figure 2A is a parasternal long-pivot picture from the ED show, whereas Figure 2B is from a research conducted 12 months earlier. There was an increase in IVC size and a decrease in inspiratory collapse {50%} consistent with extraneous pressure of the right ventricle and outpouring parcel brought about by the assortment.



Figure 3 shows a 20% drop in top inflow speeds via the mitral valve during inspiration, and Figure 4 shows evidence of flow reversal in the hepatic veins in conjunction with a dulled respiratory variety in the predominant vena cava. It was also possible to make out a minor pericardial effusion, with the greatest pocket found apically and along the right ventricular edge (100 mL). It is likely that the patient's expanded gastrointestinal parcel in the mediastinum, as opposed to the pericardial emission, was the primary source of tamponade physiology, as seen by the obvious mass impact of the gut loop on the right heart. As seen in



Figures 5, the patient underwent repeat CT chest angiography, which revealed a uniquely enlarged mediastinal and upper stomach jejunum that was causing superfluous tension of the right heart and genuinely keeping diastolic filling of the right ventricle, left chamber, and left ventricle. The right chamber extended and contrast refluxed into the inferior vena cava (IVC) because to right cardiac malfunction. The CT scan was repeated 7 days later, and it was found that the pericardial effusion had shrunk in size. This led doctors to decide against doing a pericardiocentesis and instead perform endoscopic decompression via gastroenterology after fluid replacement. Hemodynamic improvement and an increase in SBP to 135 mm Hg resulted with the removal of roughly 3 L of food debris and the subsequent installation of a nasojejunal tube under fluoroscopic supervision. After the patient had been decompressed, his care team conferred with his family and other involved parties. After it was decided that, due to the complexity of his illness, any surgical treatments would not improve his condition, he was sent home to receive hospice care.

3. Discussion

It is frequently taught that cardiac tamponade happens when liquid gathers in the pericardial sac, coming down on the heart from outside and keeping it from filling properly. Exudate, transudate, or blood that collects around the heart (the pericardium) for any number of causes may lead to cardiac tamponade. Pericardial volume may rapidly rise in response to haemorrhage caused by, for example, an entering sore to the heart or ventricular wall crack after a MI. Other gamble factors, like disease (tuberculosis [TB], myocarditis), autoimmune illnesses, neoplasms, uremia, and other inflammatory diseases (pericarditis), tend to create a slower-growing effusion. Patients tend to fare better with modest accumulations of pericardial fluid as opposed to those that happen all at once. Accordingly, pericardial effusions from traumatic causes (hemopericardium) need modest amounts to create hemodynamic shakiness, however pericardial radiations from clinical reasons (like harm) may make immense volumes of liquid gather in the pericardial sac before patients become symptomatic. The pericardium typically contains an unobtrusive amount of liquid that is physiologically important to pad the heart. The heart's chambers get pressed when liquid collects rapidly enough, and tamponade physiology occurs swiftly even with very tiny quantities. Hemo-pericardium, the outcome of a severe heart damage, is a prototypical case in point. Because the heart's chambers can't relax, venous return, filling, and cardiac output all suffer.

Effusions that expand slowly, as those caused by autoimmune illness or neoplasms, enable the pericardium to extend, and the effusions may get fairly big before they cause tamponade.[8]

Hemorrhagic, serosanguineous, or chylous fluids might be present. Reduced diastolic filling is at the root of cardiac tamponade, causing a drop in cardiac output. Tachycardia is an early compensatory indication that occurs in response to decreased production. As a result, the right atrium and ventricle are not filled to their full potential because of the compression. While this is valid in by far most of circumstances, it is essential to remember that any tension applied remotely to the heart will ultimately bring about the well known course of tamponade physiology, which includes the inhibition of cardiac filling and the reduction of output.7,8 This is also the situation with tamponade caused by issues in the gastrointestinal

system, as is the case with pneumopericardium⁹, another uncommon cause. Clinicians need to be aware that twisted life systems will adjust prior lessons of well established sickness pathology even as contemporary medicine continues to produce novel treatments and therapies to enhance and lengthen patients' lives. Whether they are placed there surgically or the diaphragm herniates, echocardiographers have new obstacles when examining patients with intrathoracic gastrointestinal organs. Firstly, following an Ivor Lewis surgery, features in the anterior mediastinum might severely restrict typical echocardiographic windows, particularly in blockage with air-fluid levels. It is possible for stomach or intestinal contents to masquerade as a lump in the chest.¹⁰ When the architecture of the digestive system is unclear on an echocardiogram, fizzy drinks and other forms of oral echo contrast may help shed light on the situation.¹¹ In this particular instance, however, the patient showed symptoms of gastrointestinal blockage that ruled out oral nutrition. Our instance illustrates how individuals with surgically changed anatomy may have non-typical illness manifestations. Without echocardiographic supervision, doing a pericardiocentesis on this patient may have been very dangerous, if not fatal.

This example further highlights the need of having echocardiographic imaging easily accessible to facilitate the rapid identification, treatment, and discharge of critically sick patients. Clinical symptoms of tamponade heart and a history of a large pericardial effusion necessitating emergency removal of pericardial effusion did not exclude the need of echocardiographic and CT imaging in this case. Although the patient's persistent effusion likely contributed to the outward cardiovascular pressure on the right half of the heart, the undeniable deformation seen on imaging of the right ventricular free wall because of the expanded jejunum was reasonable the essential supporter of this patient's basic condition.

4. Conclusion

Cardiovascular tamponade is brought about by outside pressure of the heart and is lethal because it stops the heart from filling correctly, leading to a decrease in cardiac output and cardiogenic shock. Even though pericardial effusion is the most frequent cause of tamponade, and pericardiocentesis is the standard procedure for relieving symptoms, tamponade may be caused by any mass action on the heart. This case report details the effective use of esophagogastroduodenoscopy to relieve cardiac tamponade due to a gastrointestinal blockage. Medical professionals must be aware of the potential for anatomical alteration after thoracic surgeries. The usefulness of echocardiography as a diagnostic tool for these uncommon aetiologies of cardiac tamponade and as a guide for the safest route to pericardiocentesis is highlighted by our case.

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