Postsurgical Left Atrial Compression due to Hemopericardium Resulting in Liver Failure: A Rare Cause of Cardiac Tamponade

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INTRODUCTION

Left atrial cardiac tamponade is an uncommon phenomenon. We present a case of mechanical aortic valve replacement that developed posterior pericardial effusion causing left atrial compression, cardiac tamponade, and liver failure.

CASE PRESENTATION

A 50-year-old woman with a history of quadricuspid aortic valve underwent mechanical aortic valve replacement with a no. 21 On-X valve. Immediate postoperative course was unremarkable, and the patient was discharged on anticoagulation on postoperative day 5. They presented 18 days later with upper abdominal pain, severe nausea, and dyspnea. They were noted to be in acute liver failure and were transferred to our hospital for further management. On arrival, the blood pressure was 111/74 mm Hg, heart rate was 104 beats per minute, and SpO_2 was 100% on nasal canula at a flow of 2 L/min. Physical exam revealed an anxious woman with epigastric and right upper quadrant abdominal tenderness to palpation, bibasilar pulmonary crackles, and trace bilateral pedal edema. Neck veins were difficult to visualize due to obesity. Pertinent laboratory analysis revealed Hgb, 7.2 g/dL; international normalized ratio, 9; Pro-BNP, 4,348 pg/mL; lactate, 2.4; creatinine, 0.66 mg/dL; AST, 1,304 U/L; ALT, 1,064 U/L; alkaline phosphatase, 254 U/L; and total bilirubin, 0.8 mg/dL.

Transthoracic echocardiogram (TTE) showed large posterior pericardial effusion (diameter 50 mm; Figure 1), without obvious hemodynamic effect on the right atrium or the right ventricle. However, the left atrium (LA) was small and partially collapsed, and the left ventricle (LV) exhibited pseudodyskinesis (Figure 2; Video 1 and Video 2). The inferior vena cava (IVC) was dilated with minimal respiratory variation noted (Figure 3). Respiratory variation in the left ventricular outflow tract and mitral Doppler tracings were consistent with cardiac tamponade physiology. Prosthetic aortic valve was well seated without significant perivalvular leak. Right ventricular systolic pressure and estimations of left-sided filling pressures using Doppler hemodynamics were not obtained as the echocardiogram was done in an urgent, limited fashion. Contrast-enhanced computed tomography (CT) of the abdomen demonstrated posterior hemopericardium with compression of the LA (Figure 4) and heterogenous attenuation throughout the liver compatible with passive hepatic congestion. The pericardial effusion measured 40 Hounsfield units, compatible with subacute blood. Initially, primary hepatic disease was considered as an explanation for liver failure. Acute hepatitis and autoimmune panels were negative, and right upper quadrant ultrasound showed nonspecific gall bladder wall thickening and passive hepatic congestion. Gastroenterology was consulted and concurred that the liver dysfunction was likely due to passive congestion. The patient was diagnosed with cardiac tamponade due to left atrial compression with resultant liver failure.

Supratherapeutic international normalized ratio was reversed with prothrombin complex concentrate and intravenous vitamin K. Central venous catheter was placed, and central venous pressure (CVP) was 22 mm Hg. The patient was taken to the operating room by cardiothoracic surgery, and a subxiphoid pericardial window was performed with 700 mL of dark blood immediately evacuated from the pericardial space. It was noted that the effusion was under high-pressure containment, as it had spurted forcefully out upon pericardial window placement. Postoperatively, CVP decreased to 6 mm Hg, blood pressure normalized, and nausea and abdominal pain almost immediately resolved. The pericardial drain was removed on postoperative day 2. Postoperative TTE showed resolution of pericardial effusion, and left atrial size immediately normalized (Figure 5). Anticoagulation was restarted on postoperative day 1 once hemostasis was confirmed. Liver function tests improved over the next several days, and the patient was eventually mobilized and discharged home on postoperative day 6 (Table 1). The patient was seen at follow-up 6 weeks later and was doing clinically well. Repeat TTE showed no pericardial effusion with normal left arial size.

DISCUSSION

This case demonstrates a postoperative posterior pericardial effusion causing left atrial compression and tamponade resulting in hemodynamic decompensation, hepatic congestion, and liver failure.

Cardiac tamponade is a life-threatening condition due to fluid accumulation in the pericardium inhibiting cardiac chamber filling, leading to decreased cardiac output.[1] Postcardiac surgery tamponade can be classified as early (<24 hours) and late (>5-7 days).[1] Although classically affecting right-sided chamber filling, focal or loculated hematomas can result in compression of left-sided chambers as well.

Focal left atrial diastolic collapse has been described as an uncommon etiology of postoperative cardiac tamponade.[2,3] Left atrial compression can result in diminished LV diastolic filling and low cardiac output. Elevated filling pressures, pulmonary edema, and subsequently elevated right heart pressures can result in hepatic congestion.

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Keywords: Left atrial compression, Left atrial cardiac tamponade, Liver failure

Conflicts of interest: The authors reported no actual or potential conflicts of interest relative to this document.

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2468-6441

https://doi.org/10.1016/j.case.2022.06.012

VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE parasternal long-axis view demonstrates a small compressed LA (*arrows*) with large posterior pericardial effusion. The LV demonstrates pseudodyskinesis.

Video 2: Two-dimensional TTE apical 4-chamber view demonstrates the focal nature of the pericardial effusion compressing the LA (*arrows*).

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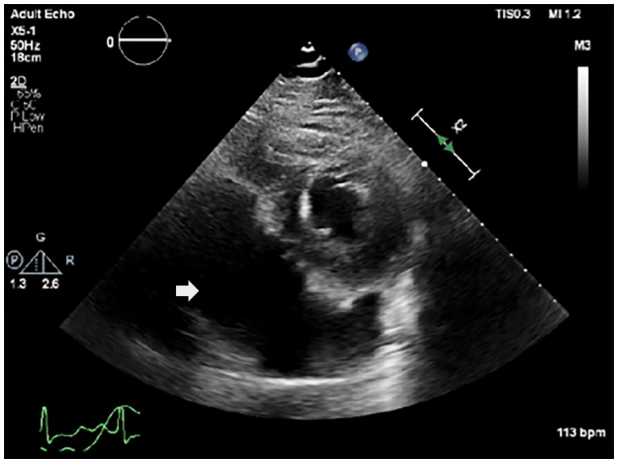

Figure 1 Two-dimensional TTE parasternal short-axis view at the midventricular level demonstrates a large loculated posterior pericardial effusion (*arrow*).

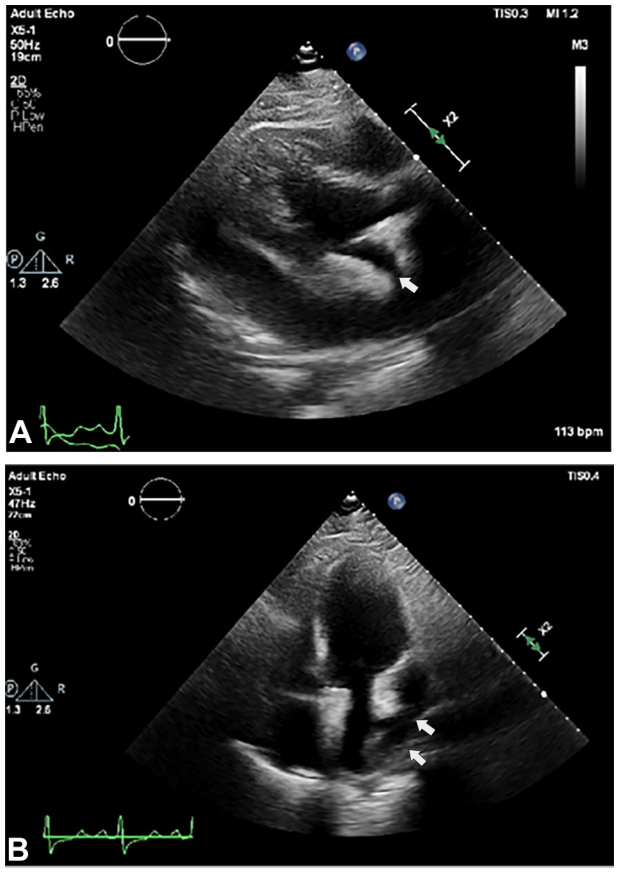

Figure 2 Two-dimensional TTE parasternal long-axis **(A)** and apical 4-chamber views **(B)**, systolic phase, demonstrate a small compressed LA due to posterior pericardial effusion. *Single arrow (top)* indicates a small compressed LA, and *double arrows (bottom)* point to the effusion compressing the LA.

Precise clot location dictates chamber compression and subsequent hemodynamic effects. Descriptions of other left-sided structures being compressed are uncommon but are also seen in the literature, including focal left ventricular outflow tract obstruction, diastolic collapse of the LV, and isolated left atrial appendage tamponade.[4-6] Even a large left pleural effusion can cause left atrial diastolic collapse and mimic cardiac tamponade physiology.[7] Our case also demonstrated an abnormal motion of the LV, consistent with LV pseudodyskinesis. Although classically caused by extrinsic diaphragmatic or subdiaphragmatic compression of the inferior wall, pseudodyskinesis was demonstrated in our case, likely from local compression from the focal pericardial effusion.[8]

Early TTE is an indispensable tool for immediate diagnosis and to guide treatment of focal cardiac tamponade. In many postcardiac surgical patients, transesophageal echocardiography is required as TTE may be technically difficult due to positive pressure ventilation, location of effusion in the far field of the transducer, and limitations due to chest wounds and tubes.[1] In our case, however, TTE was able to provide adequate diagnostic images and allowed timely mobilization of the surgical team for definitive treatment.

Classic echocardiographic findings of cardiac tamponade include right-sided chamber collapse, respiratory changes in Doppler flow velocities, reduced Doppler-estimated stroke volume and cardiac output, and a dilated IVC. However, 1 or more of these findings may be absent with focal postsurgical tamponade. As seen in our patient, LA compression and respiratory changes in Doppler flow velocities were present, but right-sided chamber collapse was not apparent. This may have been due to lack of direct right heart compression as the clot was posterior and focally impinging on the LA. In addition, subsequently elevated right heart pressures may have prevented right

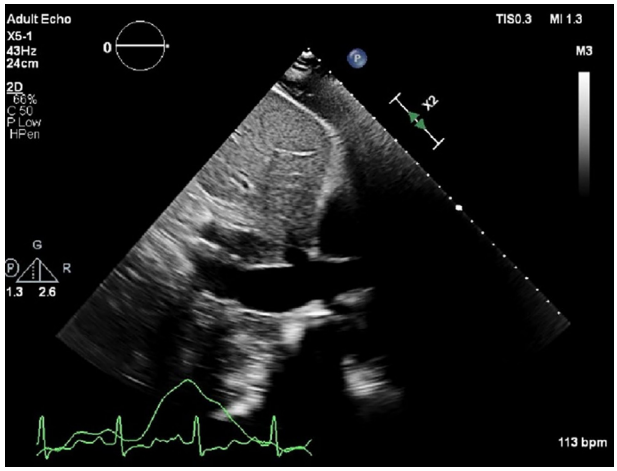

Figure 3 Two-dimensional TTE subcostal view demonstrates a dilated IVC with minimal respiratory variation (IVC plethora).

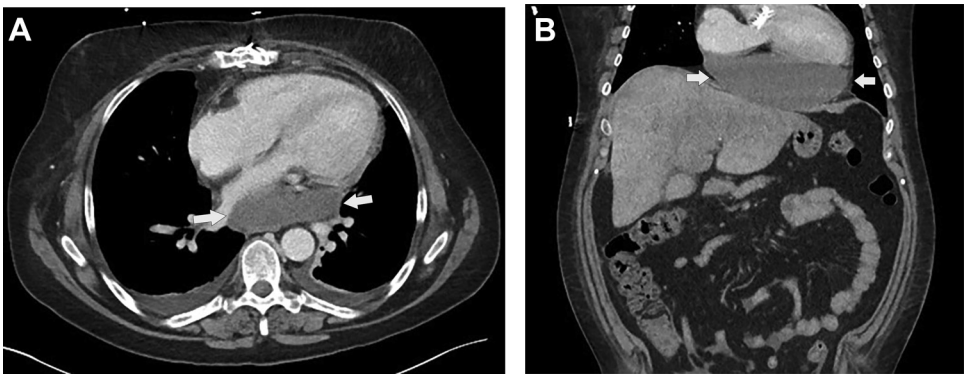

Figure 4 Contrast-enhanced thoracoabdominal CT scan, axial **(A)** and coronal **(B)** displays, demonstrates a large focal pericardial effusion with inferior extension and LA compression *(arrows)*.

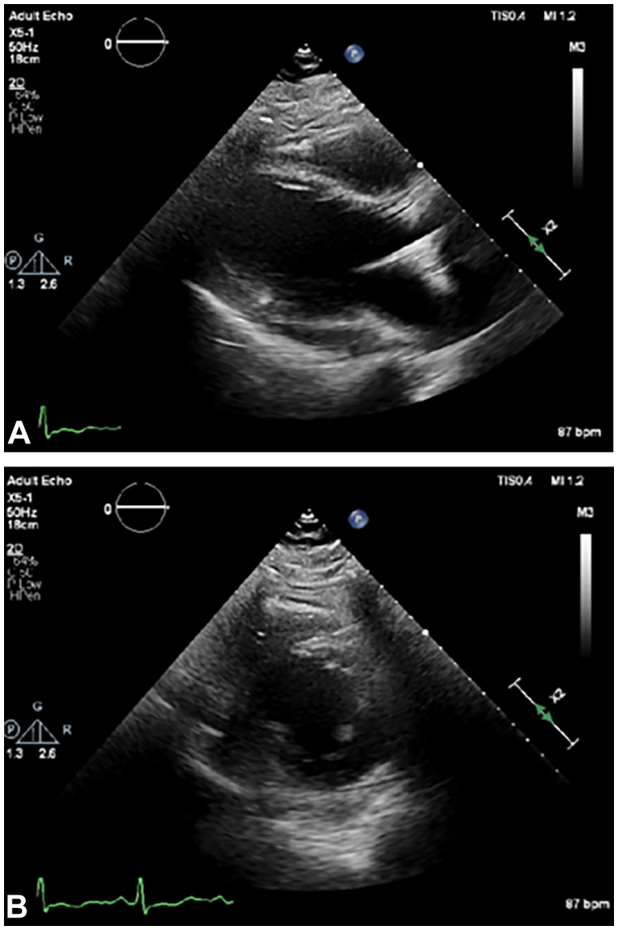

Figure 5 Two-dimensional TTE parasternal long **(A)** and short **(B)** axis displays in early systolic phase obtained after evacuation of the pericardial effusion demonstrate normal LA size and a small focal pericardial effusion.

heart chamber collapse, as evidenced by the high CVP and hepatic congestion in our case. When the clinical picture is unclear, further imaging including CT can also aid in diagnosis, as was seen in our case.

Signs and symptoms of classic tamponade may also be absent in regional focal tamponade, and a high index of suspicion is needed based on the clinical scenario. The classic "Beck's triad" of cardiac tamponade (jugular venous distention, hypotension, muffled heart sounds) has been shown to have poor sensitivity (0%-19%) for diagnosis.[9] Our patient's initial presenting of symptoms of severe abdominal pain and nausea was ambiguous, and initially a primary hepatic disease was considered. Severe hepatic congestion and subsequent stretching of the liver capsule have been seen in cardiac tamponade and may explain our patient's abdominal symptoms.[10] Worsening liver function tests and elevated prothrombin time were also a clue to the diagnosis due to passive hepatic congestion. In one case series in postcardiac surgical patients with late pericardial effusions, all patients with tamponade physiology had posterior effusions with abnormal liver function studies.[11] It is important to consider focal cardiac tamponade in the differential of new unexplained liver failure after cardiac surgery.

Table 1 Liver function tests at admission and discharge

	At admission	At discharge
Total bilirubin, mg/dL	0.8	0.3
AST, U/L	1,304	60
ALT, U/L	1,064	217
Alkaline phosphatase, U/L	254	252

After pericardial decompression and the patient's clinical improvement, timing of anticoagulation initiation is also an important consideration. The decision is based on clinical judgment after weighing the risks of rebleeding with potential valve thrombosis. After multidisciplinary discussions with the surgeons and confirming postoperative hemostasis, we initiated anticoagulation on postoperative day 1 and had no further bleeding complications thereafter.

CONCLUSION

Postcardiac surgical left atrial tamponade is an uncommon but important diagnosis that can be made with TTE. Focal tamponade should be considered with new abdominal pain and postoperative liver failure. If TTE is inadequate due to postoperative imaging limitations transesophageal echocardiography may need to be pursued promptly. A high index of suspicion is required to diagnose this life-threatening but treatable disorder.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2022.06.012.

REFERENCES

1. Klein AL, Abbara S, Agler DA, Appleton CP, Asher CR, Hoit B, et al. American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr 2013;26:965-101215.
2. Torelli J, Marwick TH, Salcedo EE. Left atrial tamponade: diagnosis by transesophageal echocardiography. J Am Soc Echocardiogr 1991;4:413-4.
3. Thompson D, Ali I, Gilbert M, Zaidi A. Left atrial cardiac tamponade complicating radical nephrectomy: case report. J Cardiothorac Surg 2020;15:165.
4. Luong C, Kim JM, Wong GC, Klein R, Brunner N. Loculated pericardial effusion: an uncommon cause of left ventricular outflow tract obstruction. JACC Case Rep 2021;3:128-32.
5. Mars T, Mikolavcic H, Salobir B, Podbregar M. Echocardiography of isolated subacute left heart tamponade in a patient with cor pulmonale and circumferential pericardial effusion. Cardiovasc Ultrasound 2010;8:27.
6. Yamano T, Nakatani S, Nakamura T, Sawada T, Azuma A, Yaku H, et al. Left atrial appendage collapse as a sole feature of cardiac tamponade after cardiac surgery: a case report. J Am Soc Echocardiogr 2007;20:1415.e1-2.
7. Khaja M, Santana Y, Rodriguez Guerra MA, Rehmani A, Perez Lara JL. Isolated left atrial cardiac tamponade caused by pleural effusion. Cureus 2020;12:e11578.
8. Yosefy C, Levine RA, Picard MH, Vaturi M, Handschumacher MD, Isselbacher EM. Pseudodyskinesis of the inferior left ventricular wall: recognizing an echocardiographic mimic of myocardial infarction. J Am Soc Echocardiogr 2007;20:1374-9.
9. Stolz L, Valenzuela J, Situ-LaCasse E, Stolz U, Hawbaker N, Thompson M, et al. Clinical and historical features of emergency department patients with pericardial effusions. World J Emerg Med 2017;8:29-33.
10. Khalid U, Favot M, Ubaid F. Pericardial tamponade masquerading as abdominal pain diagnosed by point-of-care ultrasonography. Clin Pract Cases Emerg Med 2017;1:403-6.
11. Borkon AM, Schaff HV, Gardner TJ, Merrill WH, Brawley RK, Donahoo JS, et al. Diagnosis and management of postoperative pericardial effusions and late cardiac tamponade following open-heart surgery. Ann Thorac Surg 1981;31:512-9.