Cardiac Tamponade Causing Predominant Left Atrial and Ventricular Compression After Left Ventricular Assist Device Placement

ABCDEF 1 Samjeris Victor
E 2 J.W. Awori Hayanga
E 3 John S. Bozek
E 3 Justin Wendel
B 2 Luigi F. Lagazzi
ABCD 3 Heather K. Hayanga

Corresponding Author: Heather K. Hayanga, e-mail: heather.hayanga@wvumedicine.org

Financial support: None declared
Conflict of interest: None declared

Patient: Male, 47-year-old
Final Diagnosis: Left-sided cardiac tamponade
Symptoms: Back pain • dyspnea • hypotension
Medication: —
Clinical Procedure: —
Specialty: Anesthesiology

Objective: Unusual clinical course

Background: Cardiac tamponade is a life-threatening condition that occurs when pericardial fluid accumulates in the pericardial sac, causing compression of the heart and obstructive shock. This hemodynamic event typically occurs in right-sided cardiac chambers due to the low pressures of the right atrium and right ventricle. Patients undergoing left ventricular assist device (LVAD) placement are at particularly high risk of pericardial effusion development and potential cardiac tamponade because of the need for postoperative anticoagulation.

Case Report: A 47-year-old man underwent LVAD placement for deteriorating biventricular function. After several days of stability postoperatively, he experienced dyspnea and had evidence of increasing hemodynamic compromise. He was immediately taken to the operating room, where transesophageal echocardiography showed near-complete collapse of the left atrium and left ventricle with preservation of the right heart chamber sizes in the setting of a large heterogenous posterior pericardial effusion. With swift surgical intervention, the cardiac tamponade was successfully evacuated and the patient regained hemodynamic stability.

Conclusions: Cardiac tamponade can present overtly or covertly, and should be high on the list of differential diagnoses in a patient with deterioration in hemodynamic status after cardiac surgery, especially after LVAD placement. Although cardiac tamponade usually affects right-sided cardiac chambers, the left-sided chambers can also be involved. Isolated left-sided cardiac tamponade is rare but can occur in the presence of a loculated posterior pericardial effusion, as seen in this patient.

Keywords: Anesthesia, Cardiac Procedures • Cardiac Tamponade

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/938115
Background

Cardiac tamponade is a life-threatening condition that occurs when pericardial fluid accumulates in the pericardial sac, causing compression of the heart, which leads to a decrease in cardiac output and obstructive shock [1]. Malignancy, pericarditis, and iatrogenic causes (post-surgery and cardiovascular invasive procedures) are the most common etiologies of cardiac tamponade [2]. Pericardial effusion can occur following open-heart surgery due to the increased risk of intrapericardial inflammation and bleeding [3]. Patients undergoing left ventricular assist device (LVAD) placement are at particularly high risk of pericardial bleeding and cardiac tamponade because of the need for postoperative anticoagulation agents [3]. Cardiac tamponade typically affects right-sided cardiac chambers because of the lower pressures of the right atrium and right ventricle [4]. Cardiac tamponade of the left-sided cardiac chambers is rare and typically only seen in situations such as those in patients with severe pulmonary arterial hypertension (PAH) or loculated posterior pericardial effusion [4]. This case report describes a patient who developed left-sided cardiac tamponade after LVAD placement and his subsequent hospital course.

Case Report

A 47-year-old man with a past medical history of non-ischemic dilated cardiomyopathy requiring automatic implantable cardioverter defibrillator placement, hypertension, hyperlipidemia, atrial fibrillation, chronic kidney disease stage III, and hypothyroidism presented to an outside hospital with 2 weeks of worsening lower-extremity edema, dyspnea on exertion, and paroxysmal nocturnal dyspnea. The patient reported an unintentional 3.6-kg weight gain during this time despite taking extra doses of his prescribed furosemide. His initial blood pressure was 83/61 mmHg, troponin level was 0.03 ng/mL, B-type natriuretic peptide (BNP) was 2037 pg/mL, and creatinine was 4.28 mg/dL. He was negative for COVID-19 and his chest radiograph was within normal limits. Initially, a norepinephrine infusion was started and a total of 3 L of intravenous fluids and colloids were given during the outside hospital stay in an attempt to optimize volume status. Urine output remained low at approximately 10 mL/h; therefore, diuresis was initiated with a furosemide infusion. Transthoracic echocardiography (TTE) revealed a severely dilated left ventricle with severely depressed left ventricular function with estimated ejection fraction of 20%. A diagnosis of cardiogenic shock was made and dobutamine and vasopressin infusions were added to his medication regimen. A temporary dialysis catheter was placed on hospital day 6 to initiate continuous renal replacement therapy (CRRT). Improvements in his urine output and creatinine were noted; however, due to continued hypotension requiring inotropic and vasopressor support, he was transferred to our hospital for evaluation for LVAD and/or heart transplantation.

On presentation to our facility, the patient’s blood pressure was 104/62 mmHg and he had a troponin level of 60 ng/mL, BNP of 3294 pg/mL, and creatinine of 2.81 mg/dL. He was weaned off of all vasoactive infusions upon admission, although the furosemide infusion was continued. TTE revealed a severely dilated left ventricle with global hypokinesis and ejection fraction of 20%, as well as a mildly dilated and depressed right ventricle with severe tricuspid regurgitation. Right heart catheterization revealed biventricular failure and a high mean right atrial pressure (23 mmHg) along with a low pulmonary artery pulsatility index (0.4). He was evaluated by an interdisciplinary team for LVAD Heartmate 3™ placement and was deemed to be a surgical candidate. He underwent successful LVAD placement several days later with no intraoperative complications and without need for additional mechanical right ventricular support. He was transferred to the intensive care unit (ICU) in stable condition on inhaled nitric oxide and intravenous epinephrine, vasopressin, and norepinephrine infusions with the LVAD speed at 5400 revolutions per minute (rpm) and flow at 4 L per minute (lpm).

The patient was extubated on postoperative day 1. Inotropic and vasopressor modifications were made based on evaluation of point-of-care cardiac ultrasound, pulmonary artery catheter assessment, LVAD settings, and hemodynamic evaluation. He was started on a milrinone infusion in the setting of moderate right ventricular dysfunction; the epinephrine infusion was maintained while norepinephrine and vasopressin infusions were weaned. On postoperative day 2, TTE illustrated dilated right and left ventricles with a midline interventricular septum. The LVAD flow was increased to 6000 rpm, but this caused significant bowing of the septum toward the left ventricle; therefore, the speed was decreased to 5800 rpm. Low-dose dobutamine eventually replaced epinephrine, as is standard practice at our institution. A heparin infusion was started on postoperative day 2, and a transition to warfarin was commenced on postoperative day 4. He remained hemodynamically stable and all inotropic and vasopressor infusions except low-dose dobutamine were stopped on postoperative day 5.

On postoperative day 6, He developed shortness of breath and lower back pain. He was tachycardic and hypotensive by Doppler-derived blood pressure assessment, with mean arterial pressure gradually declining to 50 mmHg, requiring reinitiation of the dobutamine infusion and then a low-dose epinephrine infusion, as well as inhaled nitric oxide. The patient’s central venous pressure was elevated at 21 mmHg, his mixed venous oxygen saturation was 41%, cardiac output using the Fick equation was 2.0 l pm, and systemic vascular

This work is licensed under Creative Common Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PAN] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Victor S. et al:
Cardiac tamponade causing predominant left atrial and ventricular compression
© Am J Case Rep, 2022, 23: e938115

Cardiac tamponade causing predominant left atrial and ventricular compression
resistance was 996 mmHg·min·mL⁻¹. His LVAD flow had decreased to 2.7 l pm, pulsatility index had increased from 2 to 8, and speed and power remain unchanged. Point-of-care cardiac ultrasound showed a questionable pericardial effusion; however, the study was limited as it was difficult to visualize the cardiac structures and function in the setting of the patient’s body habitus and the LVAD. With initial concern for potential right ventricular failure in the setting of interventricular septum interdependence, the LVAD flow was decreased to 5600 rpm. Cardiac surgery was updated. Given the limited point-of-care ultrasound findings and the patient’s perceived relative hemodynamic stability, urgent computed tomography was ordered and revealed a moderate-to-large, dense pericardial effusion. The decision was made to perform emergent chest exploration and assessment of LVAD function.

The patient was alert and oriented, communicating appropriately, and lying supine on initial evaluation by the anesthesiologist. Intraoperatively, an arterial line was placed before induction and standard monitors were used. A judicious approach to general endotracheal anesthesia was instituted using ketamine and fentanyl, and then etomidate and rocuronium. The patient was placed in Trendelenburg positioning immediately after induction due to a decrease in mean arterial pressure. Transesophageal echocardiography (TEE) revealed near-complete collapse of the left atrium and left ventricle, with preservation of the right heart chamber sizes in the setting of a large heterogeneous posterior pericardial effusion. There was no evidence of right atrial systolic free wall or right ventricular diastolic free wall collapse (Figures 1, 2). No significant deterioration in right ventricular function was noted, and the interventricular septum was undiscernible given the

Figure 1. (A) Midesophageal four-chamber view and (B) transgastric mid-papillary short-axis view of near-complete collapse of the left atrium and left ventricle with preservation of the right heart chamber sizes in the setting of a large heterogeneous posterior pericardial effusion in a patient with left ventricular assist device.

Figure 2. (A) Midesophageal four-chamber view and (B) transgastric mid-papillary short-axis view after surgical evacuation of a large heterogeneous posterior pericardial effusion in a patient with left ventricular assist device.
Cardiac tamponade is a potentially dangerous complication after implantation of LVADs [9]. Patients with LVADs are at increased risk of bleeding due to the risk of von Willebrand syndrome, platelet dysfunction, angiodysplasia, and arteriovenous malformation [10]. LVADs also pose a significant risk of pump thrombosis; therefore, patients require aggressive anticoagulation, further increasing bleeding risk. Studies report that 40% of patients with ventricular assist devices experienced early postoperative bleeding and 20% developed tamponade requiring surgical intervention [11]. Thus, early recognition and management in suspected patients is essential. The patient described in this report had multiple risk factors for developing cardiac tamponade, including recent LVAD surgery and initiation of heparin and warfarin therapies.

Isolated left-sided cardiac tamponade is a rare finding given the increased wall thickness and higher pressures of the left-sided chambers compared to the right [12]. Thus, cardiac tamponade usually affects the right side of the heart given its low chamber pressures and comparably thinner walls. However, after LVAD implantation and without significant valvular heart disease, there is an associated decrease in left atrial pressure, end-diastolic left ventricular pressure, and left ventricular diameter [13]. Most cases of isolated left-sided cardiac tamponade involve patients with severe pulmonary hypertension or postoperative loculated effusions after cardiac surgery [14]. Common symptoms associated with cardiac tamponade may be masked in patients with LVADs [15]. Thus, high clinical suspicion of cardiac tamponade is warranted, not only if right atrial pressure quickly increases, but also if a LVAD pump flow decreases [15]. Studies have demonstrated that although right-sided cardiac compression tends to have more pronounced acute hemodynamic effects than left-sided cardiac compression, left-sided cardiac tamponade can also lead to significant hemodynamic instability [16]. While cases of cardiac tamponade in patients with recent LVAD placement exist, cases in the literature of isolated left-sided collapse in this patient population
are scarce, but possible. Thus, it is imperative to be cognizant of a more indolent presentation of cardiac tamponade in patients with LVADs, especially when left-sided heart chambers are primarily involved.

Conclusions

This case presentation illustrates how a change in clinical status several days after LVAD implantation presents a clinical conundrum best deciphered through multiple data points, with TEE being an important resource in differentiating etiologies. Cardiac tamponade can have various presentations, and typically involves the right-sided cardiac chambers. However, the left atrium and left ventricle can also be affected. Isolated left-sided cardiac tamponade is rare but can occur in the setting of a posterior loculated pericardial effusion. Patients with recent cardiac surgery, especially LVAD placement, who start to show evidence of hemodynamic deterioration, require swift comprehensive evaluation and early management.

Declaration of Figures’ Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Rahim Khan HA, Gilani JA, Pervez MB, et al. Penetrating cardiac trauma: A retrospective case series from Karachi. J Pak Med Assoc. 2018;68(8):1285-87
2. Jenson J, Poulsen S, Molgaard H. Cardiac tamponade: A clinical challenge. European Society of Cardiology. 2017(4):107-13
3. Zalawadiya SK, Lindenfeld J, DiSalvo T. Rapid diagnosis of cardiac tamponade using pulsatility index variability in a patient with a HeartWare ventricular assist device. Circulation. 2015;131(13):e387-88
4. Kumar B, Kodilwadmath A, Singh A, et al. Left ventricular tamponade—pathophysiology determines the therapeutic approach: A case series. Eur Heart J Case Rep. 2020;5(2):ytaa502
5. Ariyarajah V, Spodick DH. Cardiac tamponade revisited: A postmortem look at a cautionary case. Tex Heart Inst J. 2007;34(3):347-51
6. Muralidhar K. Utility of perioperative transesophageal echocardiography. Ann Card Anaesth. 2016;19(Suppl.):S2-55
7. Essa RA, Ahmed SK. Life-threatening cardiac tamponade secondary to COVID-19 treated with unipolar video-assisted thoracoscopic surgery: A case report. Am J Case Rep. 2022;23:e935839
8. Fowler NO, Gabel M, Buncher CR. Cardiac tamponade: A comparison of right versus left heart compression. J Am Coll Cardiol. 1988;12(1):187-93
9. Kohmoto T, Oz MC, Naka Y. Late bleeding from right internal mammary artery after HeartMate left ventricular assist device implantation. Ann Thorac Surg. 2004;78(2):689-91
10. den Exter PL, Beeres SLMA, Eikenboom J, et al. Anticoagulant treatment and bleeding complications in patients with left ventricular assist devices. Expert Rev Cardiovasc Ther. 2020;18(6):363-72
11. Jett GK. ABIOMED BVS 5000: Experience and potential advantages. Ann Thorac Surg. 1996;61(1):301-4; discussion 311-13
12. Poorsattar SP, Maus TM. Isolated left-sided heart tamponade on echocardiography in severe pulmonary hypertension and right heart failure. J Cardiothorac Vasc Anesth. 2020;34(11):3172-74. Erratum in: J Cardiothorac Vasc Anesth. 2021;35(3):977-78
13. Noly PE, Pagani FD, Noiseux N, et al. Continuous-flow left ventricular assist devices and valvular heart disease: A comprehensive review. Can J Cardiol. 2020;36(2):244-60
14. Klein AL, Abbara S, Agler DA, 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(9):965-1012.e15
15. Topilsky Y, Price TN, Atchison FW, Joyce LD. Atypical tamponade hemodynamic in a patient with temporary left ventricular assist device. Interact Cardiovasc Thorac Surg. 2011;12(5):832-34
16. Saltzman AJ, Paz YE, Rene AG, et al. Comparison of surgical pericardial drainage with percutaneous catheter drainage for pericardial effusion. J Invasive Cardiol. 2012;24(11):590-93