Case Report Article

Late-Onset Type I Left Ventricular Rupture Following Double Valve Replacement: An Unexpected Cause of Cardiac Tamponade

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Highlights

- Cardiac tamponade due to late-onset type I left ventricular wall rupture is a very rare condition that has been reported only infrequently.
- A multimodality cardiac imaging approach is essential to confirm and characterize the diagnosis of left ventricular wall rupture.

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ABSTRACT

Background: Cardiac tamponade is a life-threatening emergency caused by substantial pericardial accumulation of fluid, blood, or pus. This buildup compresses the cardiac chambers, resulting in hemodynamic compromise, shock, and possible death. Postoperative cardiac tamponade incidence ranges from 0.1% to 6%. We describe a case of late-onset type I left ventricular (LV) rupture after double-valve replacement (DVR), presenting as cardiac tamponade.

Case Presentation: A 58-year-old woman was referred to our hospital with a 1-month history of progressive breathlessness and orthopnea. She had undergone DVR surgery, specifically an aortic and mitral valve replacement, 3 months prior to admission. Echocardiography performed at the referring hospital revealed a large, loculated pericardial mass, suspected to be a hemopericardium, that was compressing the LV structure and causing cardiac tamponade. Further imaging with cardiac computed tomography (CT) demonstrated contrast extravasation at the atrioventricular groove adjacent to the prosthetic mitral valve, which confirmed a type I LV rupture.

The patient was diagnosed with a late-onset type I LV wall rupture following DVR. Urgent pericardiocentesis was performed, followed by an open thoracotomy, the creation of a pericardial window, and the surgical repair of the rupture site.

Conclusions: Multimodal cardiac imaging, such as echocardiography and cardiac computed tomography, is essential for comprehensive assessment and characterization of the underlying etiology of postoperative cardiac tamponade.

Keywords: Cardiac Tamponade; Type I LV Rupture; Echocardiography; Cardiac CT; Pericardial Window; Heart Valve Surgery

RHYTHM
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Introduction

Cardiac tamponade is a progressive accumulation of fluid (effusion), blood, or pus within the pericardial cavity, leading to increased intracardiac pressure.

This pressure compresses the heart chambers, resulting in hemodynamic instability, circulatory shock, and potentially cardiac arrest.[1] Epidemiological data on the incidence and prevalence of cardiac tamponade in the general population remain limited. Reported incidence rates include approximately 0.05% (115,638 cases) in the United States, 0.8% (46 of 6,015 cases) in Malaysia, and 5.3% in Japan.[2–4] The leading etiologies include chronic kidney disease (18.4%), malignancy (17.0%), infections (16.0%), connective tissue diseases (4.0%), chest trauma (2.1%), and iatrogenic causes (<2%).[2,5]

Despite advancements in cardiac surgical techniques, postoperative cardiac tamponade remains a recognized complication, with an estimated incidence ranging from 0.1% to 6%.[6,7] The condition may arise due to post-cardiac injury syndrome or direct cardiac chamber perforation. In most cases, significant pericardial effusion occurs within the first 48 hours postoperatively and is typically associated with oozing or microvascular bleeding. Late-onset cardiac tamponade due to ventricular wall rupture following cardiac surgery is rare. Herein, we present a rare case of late-onset type I left ventricular (LV) rupture following double-valve replacement (DVR), presenting with cardiac tamponade.

Case Presentation

A 58-year-old woman was referred to our hospital with a 1-month history of progressive exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. Her symptoms had worsened over the week preceding admission. She had undergone DVR surgery (aortic and mitral) using a Medtronic Aortic Mechanical Heart Valve No. 16 (Medtronic Inc., Minneapolis, MN) and a St. Jude Masters Mitral Mechanical Valve No. 27 (Abbott, California, USA) 3 months prior. Additionally, she had a history of a permanent pacemaker implantation (Vitatron G20A2 SR, Medtronic PTE. LTD., Singapore) in VVIR mode, performed 2 years earlier at the referring hospital due to sinus node dysfunction.

On physical examination, the patient exhibited tachypnea and tachycardia. Her blood pressure was 114/61 mm Hg with norepinephrine support at 0.25 µg/kg BW/min. Her heart rate was 113 bpm, and auscultation revealed muffled metallic heart sounds. A 12-lead ECG showed atrial fibrillation with a rapid ventricular response and low-voltage QRS complexes.

Chest radiography revealed cardiomegaly (cardiothoracic ratio >0.70) with suspected pleuropericardial effusion, two visible mechanical heart valve silhouettes, and a pacemaker generator located in the left subclavian region with the distal lead projecting to the right ventricle (Figure 1).

Transthoracic echocardiography demonstrated a large, loculated pericardial effusion causing significant compression of the LV (Figure 2, Video 1). Cardiac computed tomography (CT) revealed a fistula with contrast extravasation from the LV, adjacent to the prosthetic mitral valve, into the intrapericardial cavity, consistent with a diagnosis of type I LV rupture (Figure 3).

The patient had undergone an initial urgent pericardiocentesis on the second day of hospitalization at the referring hospital, with drainage of 900 mL of hemorrhagic fluid. Despite this procedure, her clinical condition did not improve significantly, prompting a transfer to our hospital the next day. Over the course of a 10-day hospitalization at our facility, an additional 1,323 mL of hemorrhagic fluid was evacuated via a pericardial drain. Laboratory testing revealed a hemoglobin level of 9.4 g/dL, a leukocyte count of 6.2×10^3/µL, and a platelet count of 116×10^3/µL. The patient's International Normalized Ratio (INR) was elevated at 5.53. Two 5 mg doses of vitamin K were administered consecutively, and warfarin was withheld for 2 days. A subsequent INR level was in the therapeutic range at 2.20. Pericardial fluid analysis showed a protein concentration of 2.47 g/dL, a negative Rivalta test, lactate dehydrogenase of 271 IU/L, and a predominance of erythrocytes (132,360 red blood cells with 27% polymorphonuclear cells and 73% mononuclear

cells). Cultures for bacteria, acid-fast bacilli, and fungi were negative, as was cytology for malignant cells. The patient received two units of packed red cells during hospitalization.

Following consultation with the cardiothoracic surgery team, an urgent open thoracotomy via subxiphoid access was performed on the 12th day of hospitalization. The pericardial layers were opened, and approximately 1,000 mL of pericardial fluid (hematoma) was evacuated. A 5.0 mm rupture was identified at the atrioventricular junction near the mitral prosthetic valve. The LV rupture was repaired using an external approach, based on the surgeon's preference and the accessibility of the rupture site. A pericardial window was created, followed by the insertion of a water-sealed drainage.

Postoperatively, the patient showed significant clinical improvement. A follow-up cardiac CT scan performed 10 days after surgery revealed no further contrast extravasation from the LV into the intrapericardial cavity and a marked reduction in pericardial hematoma volume.

The patient was discharged on the following medications: warfarin (3 mg once daily), bisoprolol (2.5 mg once daily), candesartan (4 mg once daily), codeine (10 mg twice daily), N-acetylcysteine (200 mg three times daily), and ciprofloxacin (500 mg twice daily).

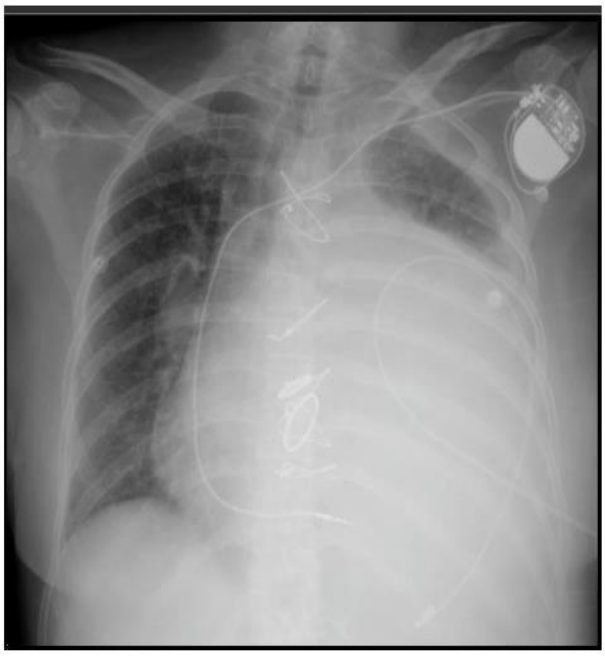


Figure 1. Chest X-ray image of patient showed cardiomegaly with cardiothoracic ratio >0.70 with water-bottle sign appearance, permanent pacemaker with single lead implanted in the projection of right ventricular. Two mechanical prosthetic valves and four sternal wires were seen.

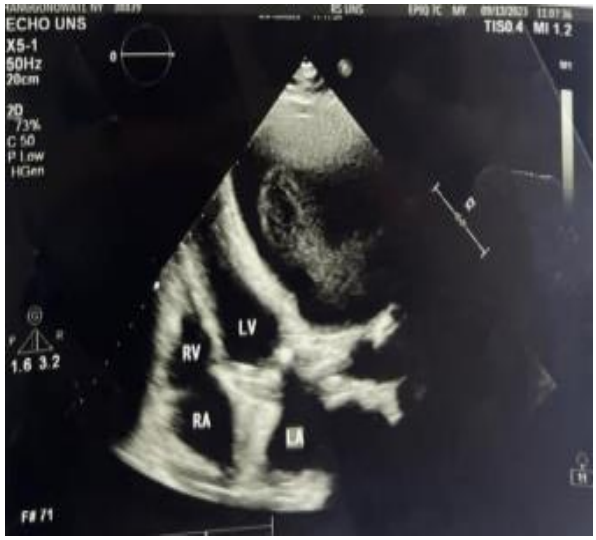
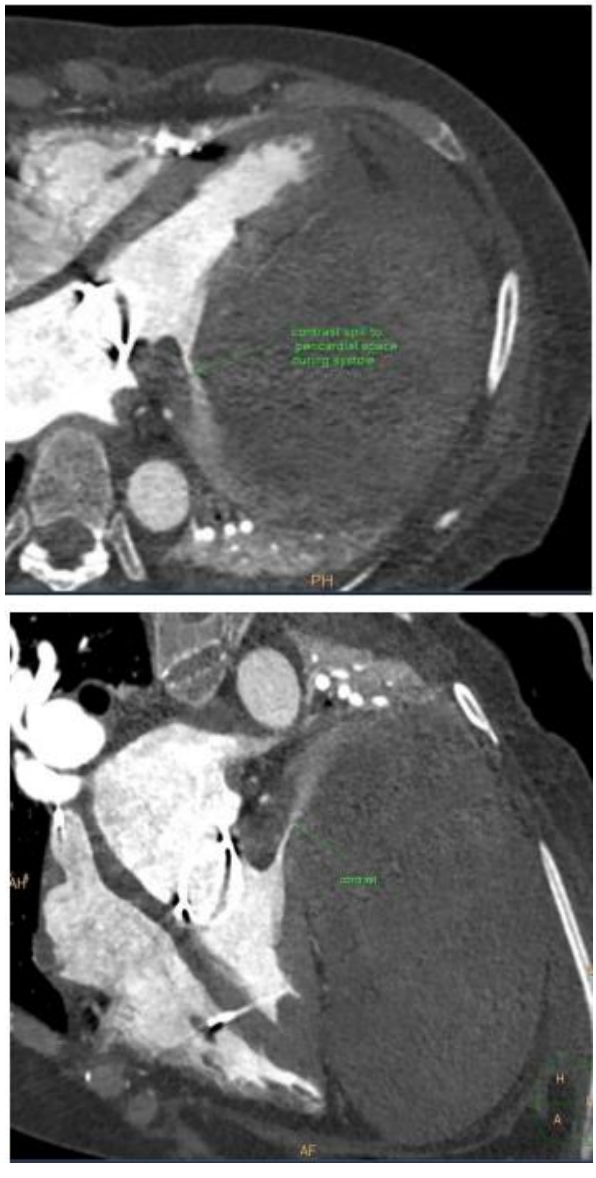


Figure 2. Transthoracic echocardiography with apical 4-chamber view revealed large loculated pericardial effusion (white arrow) which compressed LV. LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle.



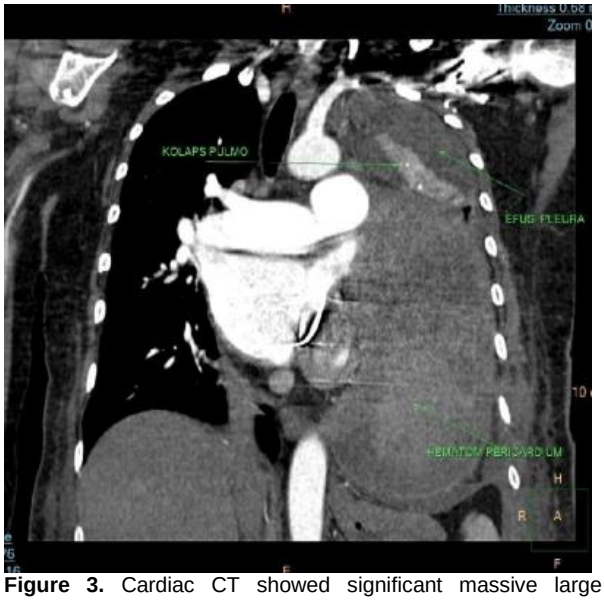


Figure 3. Cardiac CT showed significant massive large pericardial effusion due to LV wall rupture (contrast extravasation from intrachamber to pericard) near to prosthetic mitral valve (Type I LV Wall rupture).

Discussion

Postoperative cardiac tamponade occurs in 0.1% to 6% of cases.[6,7] A South Korean study reported that, among 556 patients undergoing cardiac surgery, 10 (1.8%) developed cardiac tamponade within 48 hours after surgery (immediate phase), 17 (3.1%) within 30 days (early phase), and 7 (1.2%) more than 30 days postoperatively (late-delayed phase).[8]

The pathophysiology of postoperative pericardial effusion and tamponade may involve post-pericardiotomy syndrome, in which inflammation plays a central role, or mechanical rupture of the ventricular wall following valve surgery.[9,10] Albeit rare, late delayed-onset cardiac tamponade secondary to LV wall rupture after cardiac surgery has been described. A retrospective study by Deniz et al.[11] involving 2,560 patients identified 23 cases (0.8%) of ventricular wall rupture, with 48% occurring immediately or before sternal closure. Biyikoglu et al.[12] reported a similar case of late-onset (2 years after surgery) type I LV wall rupture, specifically located in the posterior left atrioventricular groove, following mitral valve replacement. The rupture was identified using transesophageal echocardiography and cardiac CT. Surgical closure was subsequently performed using a Dacron patch via an internal approach through the left atrium.

To our knowledge, only one case of LV wall rupture following DVR surgery has been reported, by Argiriou et al.[13] In that report, the rupture occurred immediately in an octogenarian patient with comorbidities of myasthenia gravis and myocarditis. Several risk factors that might predispose to type I LV wall rupture include extensive annular calcification, small prostheses, aggressive or deep annular decalcification, excessive tissue debridement after bacterial endocarditis, deep subannular sutures, and upward malposition of the heart after prosthetic valve implantation.[11]

Types of LV wall rupture after cardiac valve surgery have been classified into four categories based on anatomical location.[14] Type I is defined as a rupture located at the atrioventricular groove, which is the most common type. Type II is defined as a rupture at the base of the papillary muscles. Type III is defined as a rupture located between Type I (atrioventricular groove) and Type II (base of the papillary muscles). Type IV is defined as a rupture located between the papillary muscles and the apex.

Multimodality cardiac imaging plays a crucial role in evaluating postoperative tamponade. It helps identify the rupture location, elucidate the underlying cause, and guide surgical intervention while providing enhanced visualization of adjacent structures (e.g., the mediastinum, lungs, and vena cava). Although chest radiography may suggest tamponade (e.g., the water bottle sign), findings can appear normal in acute cases. Echocardiography remains the first-line imaging modality for pericardial diseases, including cardiac tamponade, as recommended by several societies.[15–18]

In this case, echocardiography revealed a large, loculated pericardial effusion compressing the LV, while valve function remained intact. The compression impaired both systolic and diastolic functions, resulting in low cardiac output physiology.

The European Society of Cardiology (ESC) considers cardiac CT a second-line diagnostic tool.[19] It is particularly useful for evaluating pericardial fluid characteristics, detecting hematomas, localizing rupture sites, and aiding in

preoperative planning.[17] Nonetheless, in cases requiring urgent pericardiocentesis, cardiac CT is not typically indicated as the initial imaging modality.

In our case, cardiac CT was performed post-pericardiocentesis to identify the etiology and assess the feasibility of definitive surgical repair. The cardiac CT imaging confirmed a type I LV wall rupture, characterized by contrast extravasation from the structure around the atrioventricular groove, with an estimated rupture size of 4.8 mm.

Floerchinger et al.[20] emphasized the value of cardiac CT in diagnosing delayed tamponade after cardiac surgery, citing its superior sensitivity and specificity compared with echocardiography. Various surgical techniques for LV wall rupture repair have been described, including internal and external approaches.[10,21] In our case, an off-pump open thoracotomy via subxiphoid access was performed, with pericardial hematoma evacuation, creation of a pericardial window, and external repair of the rupture. This approach was chosen for several reasons, including the surgeon's preference and the accessibility of the rupture site. Nevertheless, a previous study found no statistically significant difference between the two approaches.[11] The procedure was successful, and the patient recovered without complications. She was discharged home 15 days postoperatively.

Conclusion

In summary, late-onset LV wall rupture should be considered in patients presenting with cardiac tamponade weeks or months after heart valve surgery, even if the initial recovery was uneventful. This case underscores the importance of a multimodal cardiac imaging approach and a multidisciplinary team in ensuring an accurate diagnosis and optimal treatment outcomes.

Declarations:
Ethical Approval

This study was conducted in accordance with the Declaration of Helsinki. Written consent was obtained from the patient.

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Conflict of Interest

The authors declare no conflicts of interest.

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