JACC: CASE REPORTS VOL. 29, 2024

HEART FAILURE AND CARDIOMYOPATHIES

CASE REPORT: CLINICAL CASE

Acute Heart Failure With Unilateral Pulmonary Edema Due to Aortic Insufficiency During LVAD Support

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ABSTRACT

Unilateral pulmonary edema (UPE) is a rare manifestation of cardiogenic pulmonary edema that is often confused with other causes of unilateral pulmonary infiltrates. A 47-year-old female with a HeartWare left ventricular assist device (LVAD) presented with dyspnea and UPE. Right heart catheterization revealed inadequate left ventricular unloading in the setting of aortic insufficiency and facilitated LVAD speed adjustment leading to resolution of symptoms. Timely diagnosis of UPE is critical because it is related to an independent increased risk of mortality, likely due to initial misdiagnosis and delayed proper treatment. The increasing use of LVADs in patients with advanced heart failure necessitates a thorough understanding of potential device complications and their management. (JACC Case Rep. 2024;29:102584)

HISTORY OF PRESENTATION

A 47-year-old female supported by a HeartWare left ventricular assist device (LVAD) (Medtronic) was admitted to the cardiac care unit after presenting to the emergency department with severe shortness of breath. She reported being in her usual state of health until the previous day when she began experiencing progressively worsening dyspnea while shopping. Overnight, she also developed nonbloody, nonbilious emesis and a productive cough. On admission, the patient was afebrile with a heart rate of 58 beats/min, Doppler opening pressure of 110 mm Hg, respiratory rate of 24 breaths/min, and oxygen saturation of 92% on a 15L non-rebreather mask. Her physical examination was notable for tachypnea, diffuse crackles on auscultation, absence of a palpable pulse, and abdominal tenderness at the driveline insertion site without guarding.

TAKE-HOME MESSAGES

- Thorough hemodynamics evaluation is essential in clarifying the final diagnosis of cardiogenic UPE in LVAD patients, ensuring appropriate and effective management.
- Timely adjustment of LVAD parameters based on hemodynamic studies can significantly improve outcomes, emphasizing the need for clinicians to be vigilant for atypical manifestations such as UPE and promptly adjust treatment protocols.

PAST MEDICAL HISTORY

The patient has a history of end-stage heart failure and was on HeartWare LVAD support for 6 years. Transplant candidacy had been declined due to

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received June 18, 2024; revised manuscript received July 16, 2024, accepted July 22, 2024.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2024.102584

ABBREVIATIONS AND ACRONYMS

AI = aortic insufficiency

LVAD = left ventricular assist device

MR = mitral regurgitation

UPE = unilateral pulmonary edema

multiple medical high-risk findings with several adverse events, very significant human leukocyte antigen sensitization, and an elevated Stanford Integrated Psychosocial Assessment for Transplant (SIPAT) score of 57 (indicating a poor candidate). During LVAD support, she experienced numerous clinical complications including multiple cerebrovascular accidents without residual deficits, gastrointestinal bleeding, chronic driveline infection treated with incision and drainage as well as chronic antibiotics, and episodes of ventricular arrhythmia.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for acute dyspnea in this patient includes several potential etiologies, including LVAD failure from pump thrombosis, cannula obstruction, motor failure, or inappropriately low pump speed leading to acute pulmonary edema from inadequate left ventricular (LV) unloading. Non-LVAD-related causes include new or worsening valvular insufficiency, ventricular arrhythmia, severe anemia caused by gastrointestinal bleeding, pulmonary embolism, and pneumonia.

INVESTIGATIONS

Electrocardiogram results showed sinus tachycardia. Chest x-ray constituted unilateral, right-sided infiltrates with a moderate size pleural effusion concerning for underlying pneumonia (**Figure 1A**). Laboratory testing showed decreased levels of hemoglobin (10.4 g/dL, reference, 12.2-15.3 g/dL), a subtherapeutic international normalized ratio of 1.6 (goal: 1.8-2.2), an elevated brain natriuretic peptide (3,007 pg/mL, reference <100 pg/mL) and mildly increased transaminase levels (aspartate aminotransferase 108 U/L, alanine aminotransferase 83 U/L, and alkaline phosphatase 173 U/L). White blood cell and platelet count were normal. Plasma free hemoglobin of 24 mg/dL (<40 mg/dL), lactate dehydrogenase of 493 IU/L (<600 IU/L), and absence of hemoglobinuria or bilirubinemia, along with LVAD interrogation showing no acute deviations in pump flow (3.2 L/min), power (3.1 W), and speed (2,460 rpm) from the patient's baseline made the diagnosis of pump thrombosis unlikely.[1] Interrogation of the implanted cardioverter-defibrillator (ICD) revealed a normal functioning single-chamber ICD and climbing intrathoracic impedance (OptiVol fluid index) but no recent arrhythmic events to correlate with the presentation. Transthoracic echocardiography depicted severe LV systolic dysfunction with closed aortic valve on all observed beats, moderate-severe aortic insufficiency (AI), moderate-severe tricuspid regurgitation, and severe mitral regurgitation (MR). A point-of-care chest ultrasound to assess pleural effusion revealed unilateral, right-sided small pleural effusion and lung consolidation representing atelectasis or pneumonia. A further infectious work-up with blood cultures, viral and methicillin-resistant *Staphylococcus aureus* swabs, and *Streptococcus pneumonia* urine antigens was ordered.

MANAGEMENT

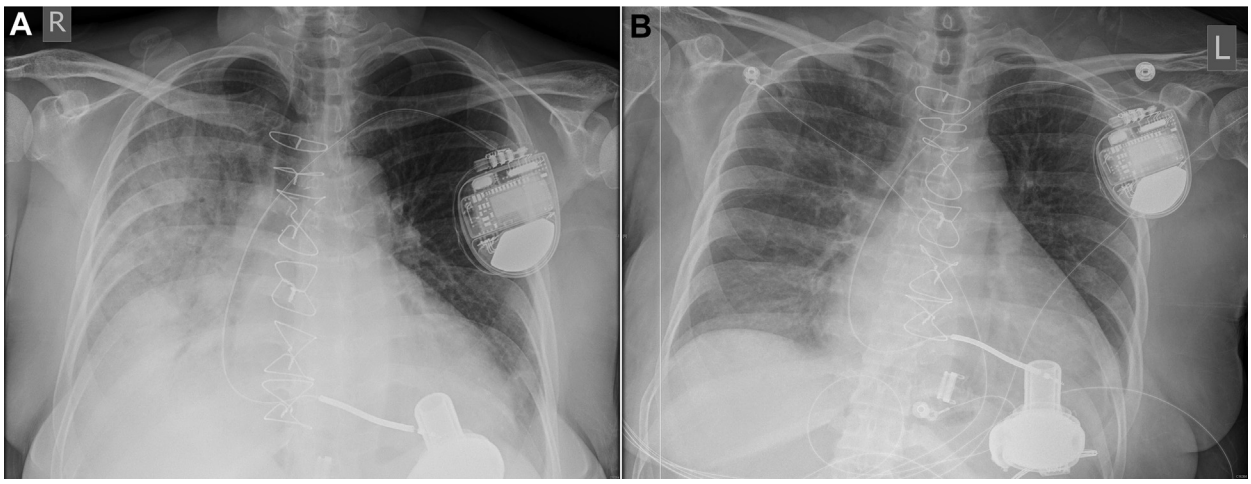
Bi-level positive airway pressure treatment for hypoxemic respiratory failure was attempted, but it was transitioned back to high-flow nasal cannula 50 L/40% due to intolerance. In addition to continuing levofloxacin (Levaquin) 750 mg/d for chronic driveline infection, the patient was started on empiric broad-spectrum intravenous antibiotics piperacillin/tazobactam (Zosyn) 4.5 g/d, intravenous vancomycin 1 g/d, and intravenous hydrocortisone 200 mg/d for severe pneumonia. To differentiate between pneumonia and unilateral pulmonary edema from inadequate LV unloading, a hemodynamic ramp study with right-heart catheterization was performed. An increase in speed from 2,460 to 2,860 rpm led to a notable reduction of opening pulmonary capillary wedge pressure (PCWP) from 24 to 15 mm Hg and mean pulmonary arterial pressure from 38 mm Hg to 28 mm Hg (**Table 1**). Oxygen saturation remained 100% after weaning the patient from high-flow nasal cannula 50 L/40% to 5 L nasal cannula on the day after the procedure. Given the drastic improvement in oxygenation following LVAD speed adjustment and negative infectious work-up, the patient was diagnosed with unilateral pulmonary edema due to aortic insufficiency in the setting of inadequate LV unloading.

DISCUSSION

We report on an atypical presentation of acute unilateral pulmonary edema (UPE) in a patient with moderate-severe AI during LVAD support and provide guidance for further management.

Typically, acute cardiogenic pulmonary edema presents as bilateral symmetrical perihilar opacities on chest radiography. However, in approximately 2% of cases, it can manifest unilaterally, predominantly affecting the right upper lobe.[2] Although cardiogenic UPE is strongly related to severe MR, its unilateral appearance on chest x-rays can lead to misdiagnoses, such as pneumonia, neoplasm, lung infarction, atelectasis, and aspiration.[3] For instance, a large retrospective analysis by Attias et al[2] involving 869

FIGURE 1 Chest Radiography



(A) Unilateral, right-sided infiltrates with a moderate size pleural effusion on admission day. (B) Significant improvement of the unilateral pulmonary edema with no pleural effusion 1 day after hemodynamic ramp study.

patients with acute cardiogenic pulmonary edema reported delay in diagnosis and treatment in 33% of cases with UPE.

In our case, the patient had been on LVAD support for 6 years, adding to the diagnostic complexity by necessitating consideration of both LVAD-related and non-LVAD-related causes. The patient's history of chronic driveline infection, unilateral pulmonary opacities, and respiratory distress initially led to the initiation of empiric antibiotics for suspected pneumonia. However, clinical signs of left heart failure, along with echocardiographic findings of moderate-severe AI and severe MR, and markedly elevated brain natriuretic peptide levels without fever or leukocytosis helped differentiate cardiogenic etiology from other diagnoses.[4] A subsequent hemodynamic ramp study served both diagnostic and therapeutic purposes showing an elevated PCWP, which was significantly improved with increased LVAD speed. These speed adjustments have previously been shown to alleviate the increase in LV pressure due to significant AI during LVAD support.[5,6] The development of moderate to severe AI following LVAD implantation exacerbates heart failure, increases rehospitalization rates, and reduces survival, particularly when the device is used as a destination therapy.[7] Transcatheter aortic valve replacement (TAVR) has emerged as an effective off-label treatment for selected high-risk patients with de novo AI while on LVAD support, leading to lasting improvements in AI severity, functional status, and quality of life.[8,9] Because AI was the main cause of our patient's decompensation and recognizing its significant impact on clinical outcomes during LVAD support, especially given the anticipated extended duration of device support, we consulted our multidisciplinary structural heart team for TAVR evaluation. Timely diagnosis is critical, because UPE carries a nearly 7-fold increased mortality risk compared to bilateral pulmonary edema, likely due to delays in proper treatment.[2] This case underscores the importance of thorough hemodynamics evaluation to distinguish cardiogenic causes from other potential diagnoses, ensuring appropriate and effective management.

FOLLOW-UP

A follow-up chest x-ray 1 day after the pump speed was increased showed significant improvement of the

TABLE 1 Hemodynamic Data During Ramp Test Phases I, II, and III

	I: Baseline	II: Maximal Speed	III: Maintenance Speed
Pump speed, rpm	2,460	3,000	2,860
MAP, mm Hg	104	90	100
Heart rate, beats/min	75	70	70
RAP, mm Hg	13	N/A‡	N/A
s/d/mPAP, mm Hg	45/25/38	40/20/33	42/19/28
PCWP, mm Hg	24	13	15
Fick cardiac index, L/min/m^2	1.92	2.44	2.50
SVR, dynes/s/cm^5	2,074	N/A	N/A

MAP = mean arterial pressure; N/A = not available; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure; s/d/mPAP = systolic/diastolic/mean pulmonary arterial pressure; SVR = systemic vascular resistance.

right-sided pulmonary edema with no pleural effusion (Figure 1B). After 2 days, the patient was breathing comfortably on room air and was discharged on day 4 after admission. Because of the complexity and the need for careful consideration after multimodality imaging, our structural heart team recommended outpatient evaluation for TAVR to address the identified valvulopathy.

CONCLUSIONS

The increasing use of LVADs in patients with advanced heart failure necessitates a thorough understanding of potential device complications and their management. This knowledge is essential for ensuring safe long-term care and improving patient outcomes.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS acute heart failure, aortic valve, cardiac assist devices, hemodynamics, insufficiency, pulmonary edema