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CASE REPORT

CLINICAL CASE

Impact of VA-ECMO on Dynamic LV Outflow Obstruction After Transcatheter Aortic Valve Replacement

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ABSTRACT

Dynamic left ventricular outflow obstruction is a rare but severe complication of transcatheter aortic valve replacement. It presents as a paradoxical hemodynamic collapse after relieving the left ventricular afterload. Considering its unique pathophysiology, this entity dictates counterintuitive treatments. We describe a case of left ventricular outflow obstruction treated with venoarterial extracorporeal membrane oxygenation and discuss its management principles. (J Am Coll Cardiol Case Rep 2024;29:102157) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

An 80-year-old man presented to the emergency department with shortness of breath. On admission, his blood pressure (BP) was 186/64 mm Hg, heart rate was 82 beats/min, respirations were 25 breaths/min, oxygen saturation was 96% (on a nonrebreather mask), and his temperature was 98.5 °F. Examination revealed acute respiratory distress with bilateral wet crackles, jugular venous distention, and a late peaking 3/6 holosystolic ejection murmur with a diminished aortic component. An electrocardiogram showed left ventricular (LV) hypertrophy and chest x-ray pulmonary edema with bilateral pleural effusions (Figure 1). He was rapidly placed on continuous positive airway pressure and diuretics. Because his hemodynamic condition was likely aggravated by his chronic kidney disease, hemodialysis was initiated to further improve his fluid status. Echocardiography revealed mildly reduced left ventricular ejection fraction (LVEF) of ~45%, a small LV cavity (left ventricular internal diastolic diameter = 4.2 cm), and severe aortic stenosis (AS, mean gradient = 45.7 mm Hg, peak aortic jet velocity = 4.2 m/s, aortic valvular

LEARNING OBJECTIVES

- To recognize dynamic LVOTO as a severe hemodynamic complication of TAVR and discuss a diagnostic and management approach for patients who present with hemodynamic instability after TAVR.
- To learn the management principles of dynamic LVOTO based on its unique pathophysiology.
- To understand the roles of VA-ECMO and IABP in the management of patients with dynamic LVOTO.

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ABBREVIATIONS AND ACRONYMS

AS = aortic stenosis

BP = blood pressure

IABP = intra-aortic balloon pump

LV = left ventricular

LVEF = left ventricular ejection fraction

LVOTO = left ventricular outflow tract obstruction

SAVR = surgical aortic valve replacement

TAVR = transcatheter aortic valve replacement

VA-ECMO = venoarterial extracorporeal membrane oxygenation

area = 0.54 cm^2) with no midventricular gradient (**Figure 2**, Video 1). The patient was diagnosed with acute decompensated heart failure attributed to severe AS resulting in afterload mismatch and preload reserve exhaustion. The patient was deemed high risk for surgical aortic valve replacement (SAVR) by the heart team and was referred for transcatheter aortic valve replacement (TAVR). Coronary angiography revealed nonobstructive coronary artery disease. Computed tomography showed an effective annulus diameter of 22 × 26 mm, an annulus area of 447 mm^2, and an annulus perimeter of 76 mm. The patient underwent TAVR (26-mm SAPIEN3 valve, Edwards Lifesciences) via the left femoral artery. After successful valve deployment (**Figure 3**), the patient's BP severely dropped, and he developed hemodynamic collapse.

PAST MEDICAL HISTORY

The patient had a history of long-standing hypertension and chronic kidney disease stage 5 (baseline creatinine of ~5 mg/dL). He had a recent outpatient visit for dialysis access creation. The patient was on furosemide 80 mg daily in addition to antihypertensive medications. He is independent for activities of daily living, although recently his exercise tolerance worsened (NYHA functional class II to III).

DIFFERENTIAL DIAGNOSIS

The most common complications of TAVR resulting in immediate hemodynamic collapse are acute coronary obstruction, aortic dissection (or annular rupture) with pericardial tamponade, valve malpositioning with severe aortic regurgitation, and retroperitoneal hemorrhage. Less commonly, the development of dynamic left ventricular outflow obstruction (LVOTO) after afterload relief can lead to unexplained severe hypotension and shock.

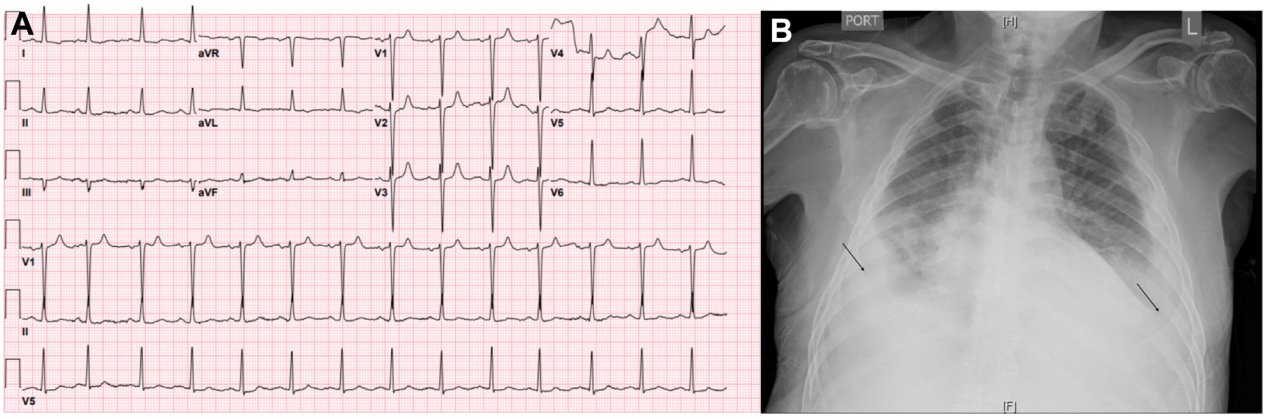
INVESTIGATIONS

The laboratory data on presentation included troponin I of 0.04 ng/mL, peaking at 0.05 ng/mL (normal <0.03 ng/mL). B-type natriuretic peptide was elevated at 2,728 pg/mL (normal <250 pg/mL). Creatinine and white blood count were not significantly elevated compared to baseline. Once the hemodynamic collapse occurred, angiography ruled out severe aortic regurgitation, annular rupture, and coronary obstruction (Videos 2 and 3). Emergency esophageal echocardiography showed a normal bioprosthetic valve position and no pericardial effusion but did show a small LV chamber with obliteration, dynamic LVOTO, and a low stroke volume (Video 4).

MANAGEMENT

Despite the use of both norepinephrine and epinephrine, the patient remained hypotensive

FIGURE 1 Initial Imaging



(A) Electrocardiography and (B) chest x-ray on presentation showing pulmonary edema with moderate bilateral pleural effusions (arrows).

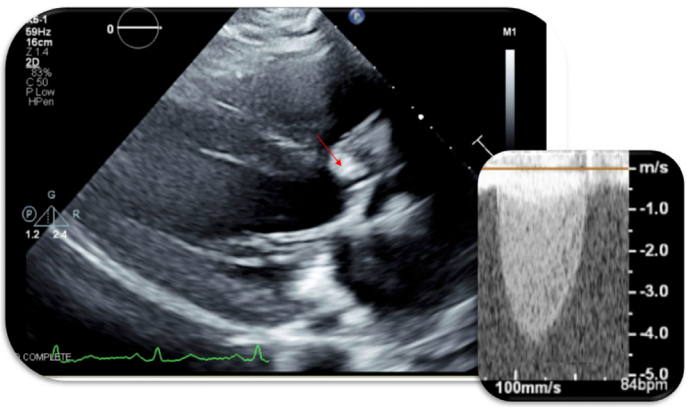
(BP = 50/30 mm Hg, heart rate = 90 beats/min), ultimately progressing to bradycardia and arrest. Advanced cardiovascular life support was initiated, and the patient was intubated. The LUCAS device (Physio-Control Inc/Jolife AB, Lund, Sweden) and further epinephrine boluses led to the return of spontaneous circulation (Video 2). The patient became severely hypertensive (BP = 280/140 mm Hg) followed by a second arrest. Because reinitiation of advanced cardiovascular life support led to no response, the patient was placed on venoarterial-extracorporeal membrane oxygenation (VA-ECMO), which provided rapid hemodynamic stabilization (Video 5), allowing subsequent decannulation on the table. An intra-aortic balloon pump (IABP) was placed, and the patient was transferred to the cardiac intensive care unit.

Unfortunately, the patient's hemodynamic condition did not improve over the next 48 hours despite the IABP and pressors. Echocardiography (contrast) showed a hyperkinetic left ventricle (Video 6). Interestingly, once the IABP was put on standby to reassess the patient's hemodynamics, the BP rose immediately (**Table 1**). The IABP was removed, leading to a dramatic hemodynamic improvement and allowing the complete removal of the pharmacologic support and extubation. Twelve days later, he was downgraded to cardiology telemetry floors and eventually discharged to subacute rehabilitation.

DISCUSSION

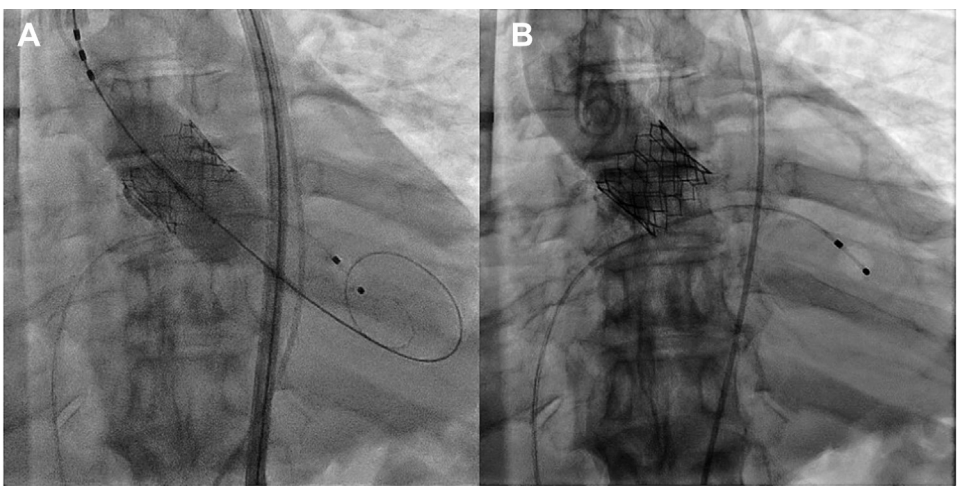
Since the first publication of a case of dynamic LVOTO after TAVR[1] and the exponential use of TAVR for the treatment of severe AS,[2] this phenomenon has been increasingly observed, and its unique pathophysiology fascinated the cardiology community.[3] Although it was first described for the right ventricle after successful pulmonary valvuloplasty in the pediatric population,[4] the pathophysiology is similar and triggered in all cases by an acute afterload reduction in a susceptible ventricle. Similarly, this complication is also observed after balloon aortic valvuloplasty or SAVR, to the point that a prophylactic myectomy is considered in selected patients undergoing surgery.[5,6] Patients with dynamic LVOTO physiology demonstrate different degrees of

FIGURE 2 Transthoracic Echocardiography



Echocardiography showing fibrocalcific disease of the aortic valve with severe aortic stenosis (arrow).

FIGURE 3 Fluoroscopy During Transcatheter Aortic Valve Replacement



(A) Valve deployment. (B) An aortogram showed no aortic annular rupture, aortic dissection, and severe aortic regurgitation.

TABLE 1 Effect of TAVR and IABP on Patient's Hemodynamics

	Pre-TAVR	Post-TAVR (Suicide LV)	IABP 1:1[a]	IABP Standby[a,b]
SBP, mm Hg	165	–	82	130
DBP, mm Hg	64	–	50	62
MBP, mm Hg	101	30	65	82
Augmentation BP, mm Hg	NA	NA	118	NA
HR, beats/min	62	92	106	100

[a]Invasive hemodynamic assessment in relation with the IABP was obtained under the same pharmacologic support (epinephrine 0.03 μg/kg/min and norepinephrine 5 μg/min intravenously). [b]Hemodynamic readings were obtained after 2 to 3 minutes of standby mode.

BP = blood pressure; DBP = diastolic blood pressure; HR = heart rate; IABP = intra-aortic balloon pump; LV = left ventricle; MBP = mean blood pressure; NA = not applicable; SBP = systolic blood pressure; TAVR = transcatheter aortic valve replacement.

unmasked LV obstruction following afterload reduction and, as a result, variable degrees of hemodynamic response.[7] Dynamic LVOTO can present with or without systolic anterior movement and mitral regurgitation. Although the development of unmasked intraventricular gradients after aortic valve replacement is not uncommon (~13.3%),[8] the incidence of true LVOTO leading to hemodynamic collapse after TAVR remains unknown. The overall published number of cases of this complication is still scarce[1,7]; thus, a lack of a specific management protocol is anticipated.

The approach to these patients should start by identifying those who are at risk of developing this complication. Left ventricles that develop dynamic LVOTO are mostly small and hypercontractile with intraventricular accelerated flow velocities, concentric hypertrophy, or sigmoid ventricular septum (depending on the dominant obstruction type, midventricular vs LVOTO) and extreme valvular stenotic features.[8] In this case, the patient had some (ie, small LV cavity) but not all (ie, LVEF of ~45%) LV risk factors for dynamic LVOTO. Although the latter is atypical for dynamic LVOTO, the patient was treated with diuretic agents and hemodialysis, possibly leading to unfavorable LV loading conditions before the procedure. In fact, dynamic LVOTO after TAVR is often triggered/exacerbated when a second variable ("unfavorable condition/inciting event") is present or introduced and the preload, afterload, or contractility is further modified.[1,7] This is why excessive volume reduction, stopping beta-blockers, or using pure inotropes or vasodilators should also be avoided in the perioperative care. Likewise, case reports have documented the worsening effect of IABP on the recovery phase of SAVR/TAVR in patients with dynamic LVOTO physiology.[9,10] In the present case, the deleterious effect of the IABP prevented the patient's recovery. Conversely, VA-ECMO was able to improve the patient's hemodynamics. The differential effects of VA-ECMO and IABP on LVOTO rely on the opposite effects on LV afterload and preload. A successful experience of VA-ECMO in a patient with refractory LVOTO after SAVR was also reported.[5] Afterload restoration, preload optimization, and contractility modulation are the foundation of dynamic LVOTO treatment.

FOLLOW-UP

At the 1-month follow-up, the patient was discharged from subacute rehabilitation. Follow-up echocardiography showed a normal LVEF, normal aortic bioprosthetic function (mean gradient = 6.4 mm Hg with no evidence of aortic regurgitation or paravalvular leak), and no subvalvular gradient. He remained on stable antihypertensive medications and hemodialysis.

CONCLUSIONS

Dynamic LVOTO is a rare but potentially fatal complication of TAVR. A complete understanding of the predisposing factors, pathophysiology, and treatment principles is key in managing these cases. Major causes of hemodynamic collapse should be ruled out first because their treatment differs completely. Afterload restoration, preload optimization, and contractility modulation are cornerstones in the initial treatment approach for these patients. Although an IABP should be avoided for patients with dynamic LVOTO, VA-ECMO represents an attractive strategy for selected patients with refractory hemodynamic collapse, providing patients with a real possibility of survival.

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REFERENCES

1. Suh WM, Witzke CF, Palacios IF. Suicide left ventricle following transcatheter aortic valve implantation. *Catheter Cardiovasc Interv*. 2010;76:616–620.

2. Mori M, Gupta A, Wang Y, et al. Trends in transcatheter and surgical aortic valve replacement among older adults in the United States. *J Am Coll Cardiol*. 2021;78:2161–2172.

3. Leya F, Tuchek JM, Coats W. Abnormal distortion of aortic corevalve bioprosthesis with suicide left ventricle, aortic insufficiency, and severe mitral regurgitation during transcatheter aortic valve replacement. *Catheter Cardiovasc Interv*. 2016;88:1181–1187.

4. Ben-Shachar G, Cohen MH, Sivakoff MC, et al. Development of infundibular obstruction after percutaneous pulmonary balloon valvuloplasty. *J Am Coll Cardiol*. 1985;5:754–756.

5. Lioufas PA, Kelly DN, Brooks KS, et al. Unexpected suicide left ventricle post-surgical aortic valve replacement requiring veno-arterial extracorporeal membrane oxygenation support despite gold-standard therapy: a case report. *Eur Heart J Case Rep*. 2022;6:ytac020.

6. Desai MY, Alashi A, Popovic ZB, et al. Outcomes in patients with obstructive hypertrophic cardiomyopathy and concomitant aortic stenosis undergoing surgical myectomy and aortic valve replacement. *J Am Heart Assoc*. 2021;10:e018435.

7. Endo N, Otsuki H, Domoto S, et al. Haemodynamic collapse immediately after transcatheter aortic valve implantation due to dynamic intraventricular gradient: a case report and review of the literature. *Eur Heart J Case Rep*. 2021;5:ytaa565.

8. Tsuruta H, Hayashida K, Yashima F, et al. Incidence, predictors, and midterm clinical outcomes of left ventricular obstruction after transcatheter aortic valve implantation. *Catheter Cardiovasc Interv*. 2018;92:E288–E298.

9. Morewood GH, Weiss SJ. Intra-aortic balloon pump associated with dynamic left ventricular outflow tract obstruction after valve replacement for aortic stenosis. *J Am Soc Echocardiogr*. 2000;13:229–231.

10. Fabbro M, Goldhammer J, Augoustides JG, et al. CASE 1-2016 problem-solving in transcatheter aortic valve replacement: cardiovascular collapse, myocardial stunning, and mitral regurgitation. *J Cardiothorac Vasc Anesth*. 2016;30:229–236.

KEY WORDS **dynamic left ventricular outflow obstruction, intra-aortic balloon pump, transcatheter aortic valve replacement, venoarterial extracorporeal membrane oxygenation**

APPENDIX **For supplemental videos, please see the online version of this paper.**