

## MINI-FOCUS ISSUE: TRANSCATHETER INTERVENTIONS

ADVANCED

## CASE REPORT: CLINICAL CASE

# Dynamic Left Ventricular Outflow Tract Obstruction Post-Transcatheter Aortic Valve Replacement



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## ABSTRACT

We describe the first case of successful management of left ventricular outflow tract obstruction developing late after transcatheter aortic valve replacement with right ventricular apical pacing. The possible mechanisms of obstruction resolution are described. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:871-4) © 2021 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 81-year-old man with diabetes was referred to our service with dyspnea (New York Heart Association functional class III). Physical examination revealed features of severe aortic valve stenosis, confirmed at echocardiography: concentric left ventricular hypertrophy (of 11 mm) with a sigmoid septum, normal systolic function, and a severely calcified aortic valve. The aortic valve area was 0.7 cm<sup>2</sup>, with a mean transvalvular gradient of 42 mm Hg. No evidence of left ventricular outflow tract obstruction (LVOTO),

systolic anterior motion of the mitral valve (SAM), intraventricular gradient, or asymmetric septal hypertrophy was identified.

## PAST MEDICAL HISTORY

The patient was deemed at intermediate risk for conventional surgery by a cardiothoracic surgeon (Society of Thoracic Surgery predicted risk of mortality 2.8%) and accepted for transfemoral transcatheter aortic valve replacement (TAVR). Under local anesthesia, an Edwards SAPIEN XT (Edwards Lifesciences, Irvine, California) size 29 was positioned after pre-dilatation of the native valve and deployed under rapid ventricular stimulation. There was superior displacement of the valve that embolized into the aorta. The guidewire position was maintained to secure orientation of the embolized valve, and a second similar valve was deployed ([Video 1](#)). The first valve was then snared and stented into position in the descending aorta. Post-operative mobile echocardiogram revealed a well-functioning prosthesis

## LEARNING OBJECTIVES

- To have a differential diagnosis for a patient presenting with increased symptoms after TAVR.
- To be able to diagnose LVOTO and differentiate it from valvular obstruction post-TAVR.
- To have an approach to different treatment modalities for this complication.

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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](#).

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## ABBREVIATIONS AND ACRONYMS

**HCM** = hypertrophic cardiomyopathy

**HOcm** = hypertrophic obstructive cardiomyopathy

**LVH** = left ventricular hypertrophy

**LVOT** = left ventricular outflow tract

**LVOTO** = left ventricular outflow tract obstruction

**SAM** = systolic anterior motion of the mitral valve

**SAVR** = surgical aortic valve replacement

**TAVR** = transcatheter aortic valve replacement

with a mean gradient of 14 mm Hg and trivial paravalvular leak. At 6 weeks follow-up, the patient reported dyspnea worse than before the TAVR.

## DIFFERENTIAL DIAGNOSIS

LVOTO due to undiagnosed subaortic ridge, hypertrophic cardiomyopathy with obstruction (HOcm), or SAM were considered, as well as valve dysfunction due to paravalvular leak, valve thrombosis, infective endocarditis, or patient prosthesis mismatch. Finally, late heart block had to be excluded.

## INVESTIGATIONS

Repeat echocardiogram revealed a normal functioning aortic prosthesis with no evidence of early valve thrombosis and no aortic regurgitation due to cusp rupture or paravalvular leak. A “dagger-shaped” continuous-wave Doppler tracing, typical of dynamic obstruction of the LVOT was noted with peak gradient of 150 mm Hg (Figure 1A). This was accompanied by SAM (Videos 2 and 3; transesophageal echo images of LVOT with and without color Doppler, demonstrating SAM). There were no other features of hypertrophic cardiomyopathy (HCM). The idealized indexed valve area based on manufacturer-supplied prosthesis area was 1.2 cm<sup>2</sup>/m<sup>2</sup>, making patient prosthesis mismatch unlikely in this case.

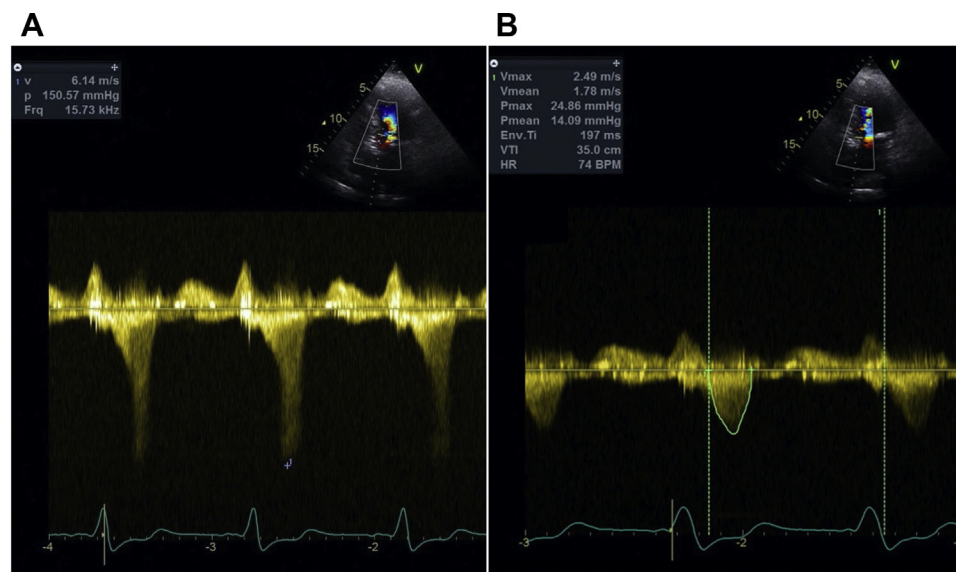
## MANAGEMENT

Importantly, although the patient’s LVOT physiology behaved like HOcm, there were no features of HCM, and tailored therapy was required. A literature search revealed only case reports of late LVOTO in patients known with HOcm or severe LVH before aortic valve implantation (1-3). There are isolated cases of the development of LVOTO at the time of TAVR that improved with fluid boluses and intravenous beta-blockade, as well as emergent alcohol septal ablation (4,5).

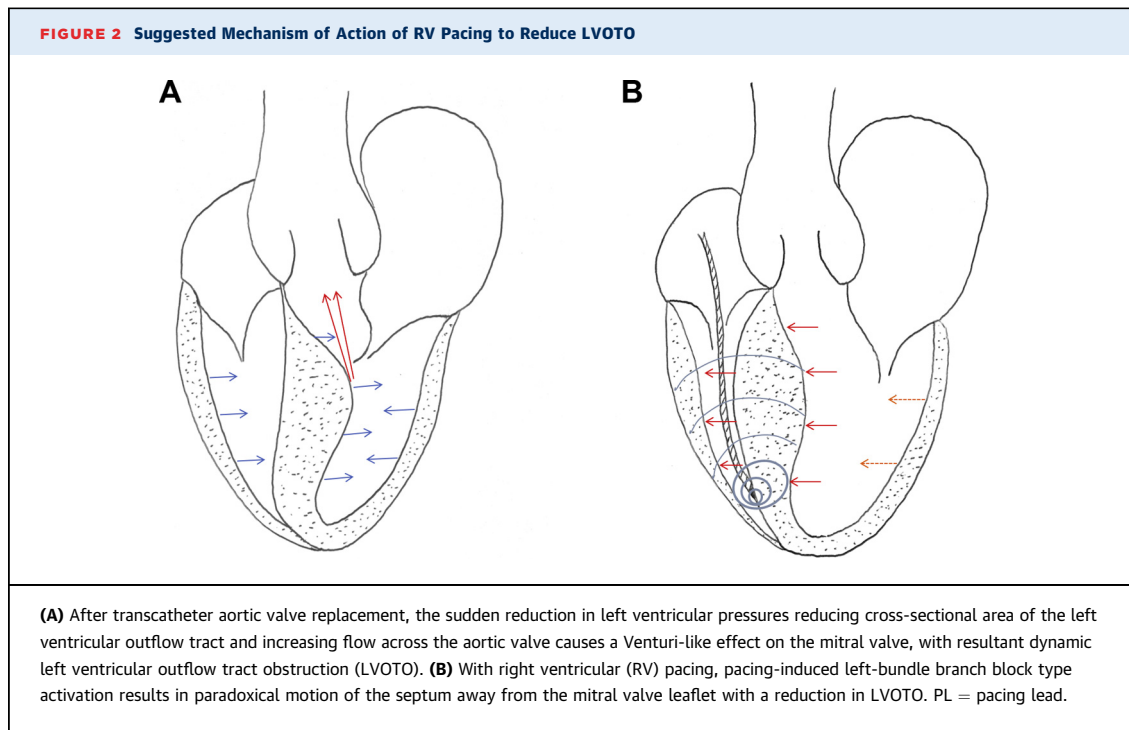
Adequate beta-blockade did not alter the patient’s clinical condition. Because the patient was elderly, we opted not to add verapamil. Disopyramide was an option but is not available in our center. Finally, surgical myectomy could be considered. In our patient, his advanced age was believed to be prohibitive. Alcohol septal ablation would be an alternative but again not without risk.

Although we do not view the evidence for pacing in the HOcm population with LVOTO to be conclusive (6), we deemed this the safest and least invasive next step in this patient. He was scheduled for biventricular pacemaker insertion, as there is some evidence that this approach is superior to right ventricular pacing alone (7). We failed to achieve an adequate position for the coronary sinus lead, and a dual-chamber system was implanted instead. This pacemaker was then optimized, using

**FIGURE 1** Doppler Tracing Through Left Ventricular Outflow Tract



**(A)** Apical 5-chamber continuous-wave Doppler tracing through the left ventricular outflow tract demonstrating the typical dagger-shaped pattern of dynamic left ventricular outflow tract obstruction. **(B)** After pacing, the gradient is significantly reduced.



echocardiography, to the shortest atrioventricular delay possible (8). There was complete abolition of the SAM with improvement of both the peak LVOT gradient, from 150 mm Hg to 24 mm Hg (Figure 1B), and the patient's symptoms.

## DISCUSSION

The development of LVOTO in the post-surgical aortic valve replacement (SAVR) group is well documented. To our knowledge, however, there are no reported cases describing the late development of LVOTO post-TAVR in patients without HCM or severe LVH before valve implant (2,3,8).

The mechanism behind SAM and LVOTO after TAVR is poorly understood. It is suggested that chronic aortic stenosis results in an increase in intra-ventricular pressures, which splints the septum and prevents inward motion. This prevents both mid-ventricular as well as LVOTO. The sudden reduction of this LV pressure after AVR results in a reduced LVOT area. In addition, the increased flow across the LVOT results in a Venturi effect, drawing the anterior mitral valve leaflet into the LVOT with dynamic obstruction (4).

In the SAVR group, factors that may suggest dynamic LVOTO post-valve implant include a sigmoid-shaped septum, asymmetric septal hypertrophy (septal to ventricular free wall ratio >1.4), increased basal septal thickness (>15 mm), and narrow LVOT

(<18 mm), as well as a ratio of anterior to posterior mitral valve leaflet length <1.3 (8). Whether these can be extrapolated to the TAVR group is not known. None of the other predictors of LVOT obstruction were identified in our patient. Although sigmoid septum is not an established indication for SAVR over TAVR, TAVR has been associated with increased risk for pacemaker requirement, and if significant LVOTO can be shown pre-operatively, SAVR can potentially be combined with a myectomy (9).

The management of dynamic LVOTO post-AVR is complex. In the acute setting, factors such as increasing LV preload with fluid boluses and reduction in LV contractility by weaning of inotropes and increasing beta-blockade may reduce obstruction. The use of dual-chamber pacing in the setting of patients with severe shock has also been suggested (8).

TAVR candidates are older and higher risk than SAVR and HCM patients, and management strategies therefore have to be individualized. Our approach was to start with conservative treatment and escalate cautiously.

The proposed mechanism by which ventricular pacing works is by changing the activation pattern of myocardial depolarization by initiating ventricular depolarization at the right ventricular apex. The short PR interval and pacing-induced left bundle branch block cause paradoxical movement of the septum away from the anterior mitral valve leaflet with

reduction in the dynamic outflow obstruction (Figure 2). Decreased LV inotropy may also contribute to the reduction in obstruction (6). Optimization of the atrioventricular delay using a combination of echocardiography and the surface electrocardiogram (to ensure that there is no fusion between intrinsic and paced QRS complexes and therefore full pre-excitation from the apical pacing site) is crucial to ensure an optimal result (8,10).

#### FOLLOW-UP

At the 3-month follow-up, the patient remained without a significant gradient and symptomatically improved.

#### CONCLUSIONS

Dual-chamber pacing may have a role in the management of an elderly patient with LVOTO post-TAVR.

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**KEY WORDS** aortic valve, cardiomyopathy, pacemaker, valve replacement

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