

CASE REPORT

ADVANCED

CLINICAL CASE SERIES: PROCEDURAL COMPLICATIONS

A Word of Caution Before Treating Aortic Stenosis in Patients With Concomitant LVOT Obstruction



Itzik Ben-Dor, MD,^a Toby Rogers, MD, PhD,^{a,b} Giorgio A. Medranda, MD,^a Diego Medvedofsky, MD,^c Gaby Weissman, MD,^c Brian C. Case, MD,^a Lowell F. Satler, MD,^a Ron Waksman, MD^a

ABSTRACT

Patients with aortic stenosis and concomitant left ventricular outflow tract obstruction undergoing transcatheter aortic valve replacement are at risk of hemodynamic collapse after the procedure due to worsening left ventricular outflow tract obstruction. We present 3 cases highlighting the important interplay between these 2 disease states and associated diagnostic and treatment challenges. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2022;4:1162-1168) © 2022 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/>).

Patients with aortic stenosis (AS) and concomitant left ventricular outflow tract (LVOT) obstruction are at risk of developing a “suicide left ventricle” with hemodynamic collapse after transcatheter aortic valve replacement (TAVR). We

present 3 cases of TAVR using the CoreValve Evolut PRO+ (Medtronic) transcatheter heart valve (THV) that illustrate the complex hemodynamic interplay between severe AS and concomitant LVOT obstruction. These cases emphasize the importance of comprehensive invasive hemodynamic assessment and multimodality imaging, including computed tomography (CT) imaging, magnetic resonance imaging (MRI), and transesophageal echocardiogram (TEE), when determining which pathology to treat first. All patients consented for their cases to be reported.

LEARNING OBJECTIVES

- To make a differential diagnosis between hypertrophic obstructive cardiomyopathy and aortic stenosis, understanding that treatment of aortic stenosis can exacerbate left ventricular outflow tract obstruction.
- To identify the risk factors, mechanisms, and treatment options before and after transcatheter aortic valve replacement in patients with hypertrophic obstructive cardiomyopathy.
- To know treatment options in case of hemodynamic collapse.

CASE 1

An 84-year-old woman presented with severe symptomatic AS (aortic valve area: 0.5 cm²; mean gradient: 60 mm Hg). Echocardiography showed a left ventricular ejection fraction of 65% with a prominent septal bulge (**Figure 1A, Video 1**). Continuous-wave Doppler revealed typical AS and the classic

From the ^aSection of Interventional Cardiology, MedStar Washington Hospital Center, Washington, DC, USA; ^bCardiovascular Branch, Division of Intramural Research, National Heart, Lung and Blood Institute, National Institutes of Health, Bethesda, Maryland, USA; and the ^cDivision of Cardiology, MedStar Washington Hospital Center, Washington, DC, USA.

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“sawtooth” pattern indicative of LVOT obstruction (Figure 1B). Coronary angiography revealed non-obstructive coronary artery disease. The aortic valve had calcification with restricted leaflet opening on CT imaging (Figure 1C, Video 2). Invasive hemodynamic assessment showed a mean gradient of 70 mm Hg with a spike-and-dome pattern after premature ventricular contraction (Figure 1D). The patient was deemed to have high surgical risk and referred for transfemoral TAVR. A 26-mm self-expanding THV was deployed with good position and no paravalvular leak (Video 3).

After deployment of the THV, the patient developed profound hypotension and hypoxia requiring intubation and pressor support. Hemodynamic assessment revealed an LVOT gradient of 40 mm Hg, provokable to 80 mm Hg with ectopy. Emergent TEE revealed a

prominent septal bulge with systolic anterior motion (SAM) of the mitral leaflet and severe mitral regurgitation (MR) (Figure 2A, Video 4). Despite aggressive medical therapy with fluid and an intravenous alpha-agonist, the patient’s hypoxia and shock persisted. Alcohol septal ablation (ASA) to debulk the LVOT was pursued (Figure 2B). Contrast agent was injected through the lumen of an over-the-wire balloon, inflated to occlude the septal, to confirm it subtended the target myocardium (Figures 2C and 2D). This was followed by injection of 3 mL of ethanol (Video 5). SAM resolved, and the patient’s MR improved to mild (Figure 2E, Video 6). The LVOT gradient improved (Figure 2F). The patient was successfully extubated 6 hours post-

**ABBREVIATIONS
 AND ACRONYMS**

- AS** = aortic stenosis
- ASA** = alcohol septal ablation
- CT** = computed tomography
- LVOT** = left ventricular outflow tract
- MR** = mitral regurgitation
- MRI** = magnetic resonance imaging
- SAM** = systolic anterior motion
- TAVR** = transcatheter aortic valve replacement
- TEE** = transesophageal echocardiogram
- THV** = transcatheter heart valve

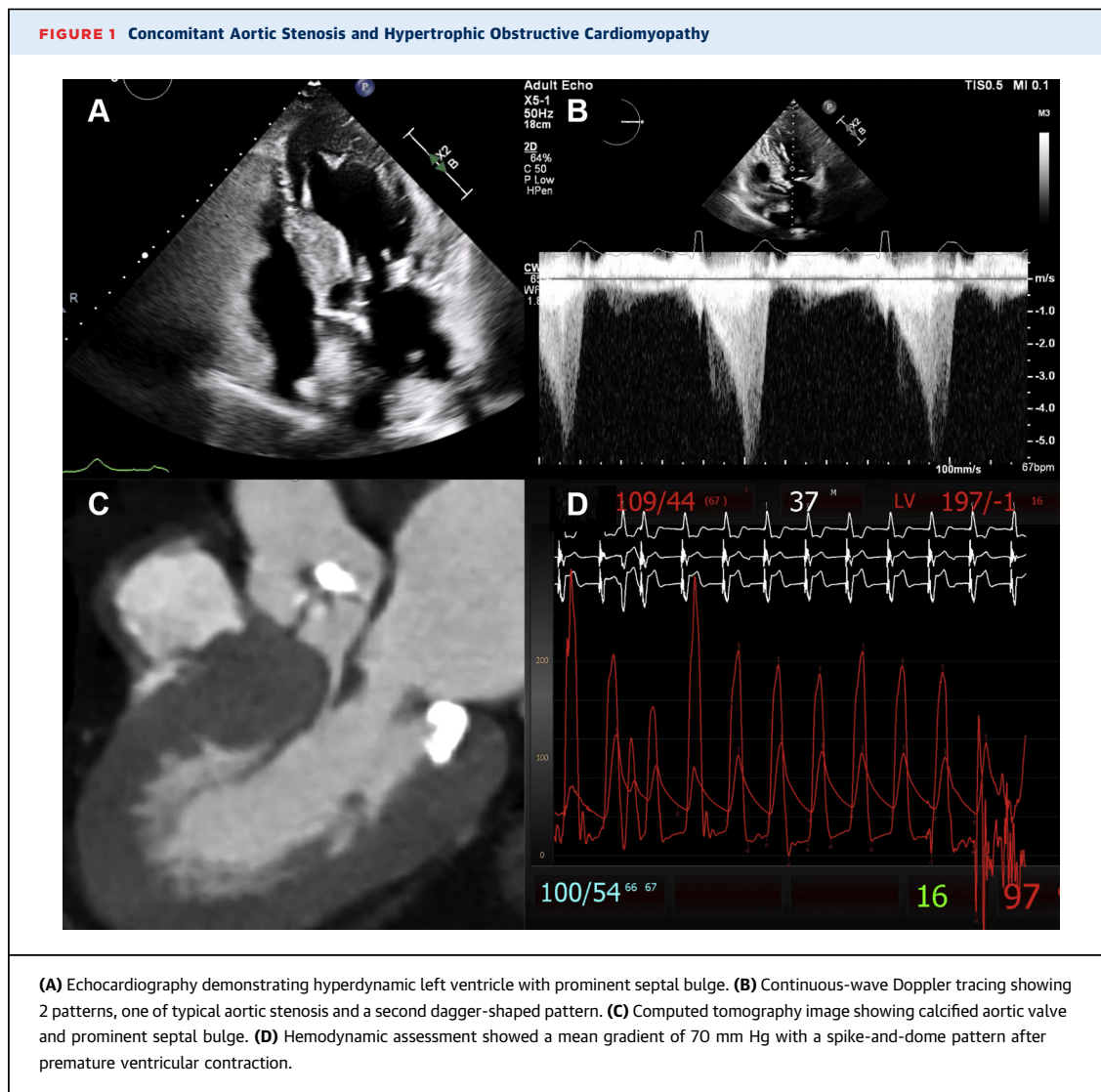
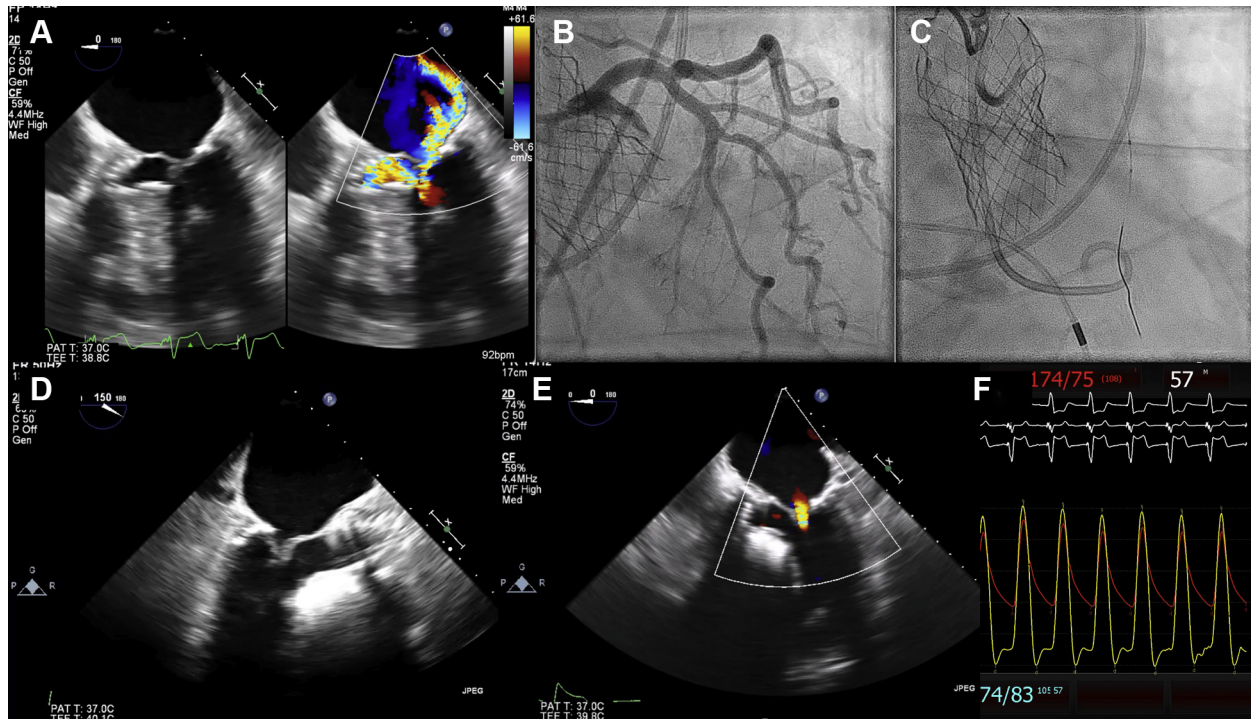


FIGURE 2 Transcatheter Aortic Valve Replacement in Patient With Hypertrophic Obstructive Cardiomyopathy Leading to Hemodynamic Collapse Treated With Alcohol Septal Ablation

(A) Transesophageal echocardiogram revealed prominent septal bulge with systolic anterior motion of the mitral leaflet and severe mitral regurgitation. (B) The left main coronary angiography engaged through the self-expanding frame. Contrast agent was injected through a balloon inflated in the first septal artery (C) lighting up the target septal myocardium on transesophageal echocardiogram (D). (E) Systolic anterior motion resolved and mitral regurgitation decreased. (F) Left ventricular outflow tract gradient fell to 10 mm Hg.

TAVR and discharged home 5 days later. Upon reflection, it is probably better to address the hypertrophic obstructive cardiomyopathy before AS, as shown in the following case.

CASE 2

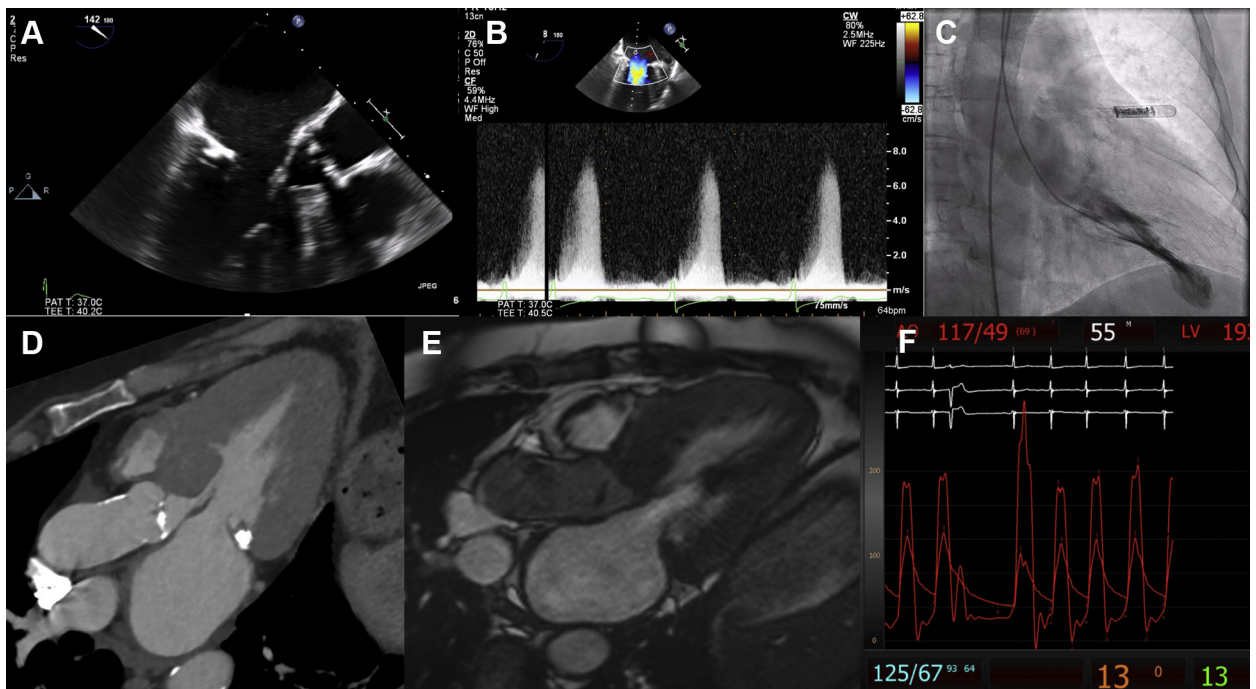
A 76-year-old woman presented with heart failure with recurrent gastrointestinal bleeding and was found to have severe AS (aortic valve area: 0.9 cm²; mean gradient: 80 mm Hg) with Heyde syndrome. Echocardiography revealed hyperdynamic left ventricular function and a prominent septal bulge (Figure 3A). Continuous-wave Doppler revealed typical AS and the classic “sawtooth” pattern indicative of LVOT obstruction (Figure 3B). Coronary angiography revealed nonobstructive coronary artery disease and a hyperdynamic left ventricle with cavity obliteration (Figure 3C, Video 7). CT scanning showed SAM of the mitral valve and severe aortic valve calcification (Figure 3D, Video 8). MRI confirmed these findings (Figure 3E, Video 9). Invasive

hemodynamic assessment showed a mean gradient of 50 mm Hg with a positive Brockenbrough-Braunwald-Morrow sign after premature ventricular contraction (Figure 3F). The patient was deemed to have high surgical risk and referred for ASA before transfemoral TAVR. The septal target was identified by using a contrast agent through an over-the-wire balloon and TEE guidance (Figures 4A and 4B, Video 10). This was followed by injecting 2 mL of ethanol occluding the artery. SAM resolved and the patient’s MR improved to mild. The LVOT gradient improved (Figure 4C). Two months later, a 29-mm self-expanding THV was deployed with good position and no paravalvular leak (Figure 4D, Video 3).

CASE 3

A 90-year-old man presented with exertional dyspnea and was found to have severe AS (mean gradient: 50 mm Hg). Echocardiography revealed a left ventricular ejection fraction of 60% with a septal thickness of 1.2 cm (Figures 5A and 5B). Coronary

FIGURE 3 Multimodality Imaging of Patient With Aortic Stenosis and Hypertrophic Obstructive Cardiomyopathy



(A) Transesophageal echocardiogram showed hyperdynamic left ventricle with prominent septal bulge. (B) Continuous-wave Doppler tracing exhibiting a typical dagger-shaped pattern. (C) Left ventriculogram showed hyperdynamic ventricle with cavity obliteration. (D) Computed tomography imaging revealed asymmetric septal hypertrophy and systolic anterior motion of the mitral leaflet. (E) Magnetic resonance imaging showing thickened basal septum with systolic anterior motion of the mitral leaflet. (F) Aortic pulse pressure decreased after premature ventricular construction provocation with an increase in the combined gradient to 110 mm Hg.

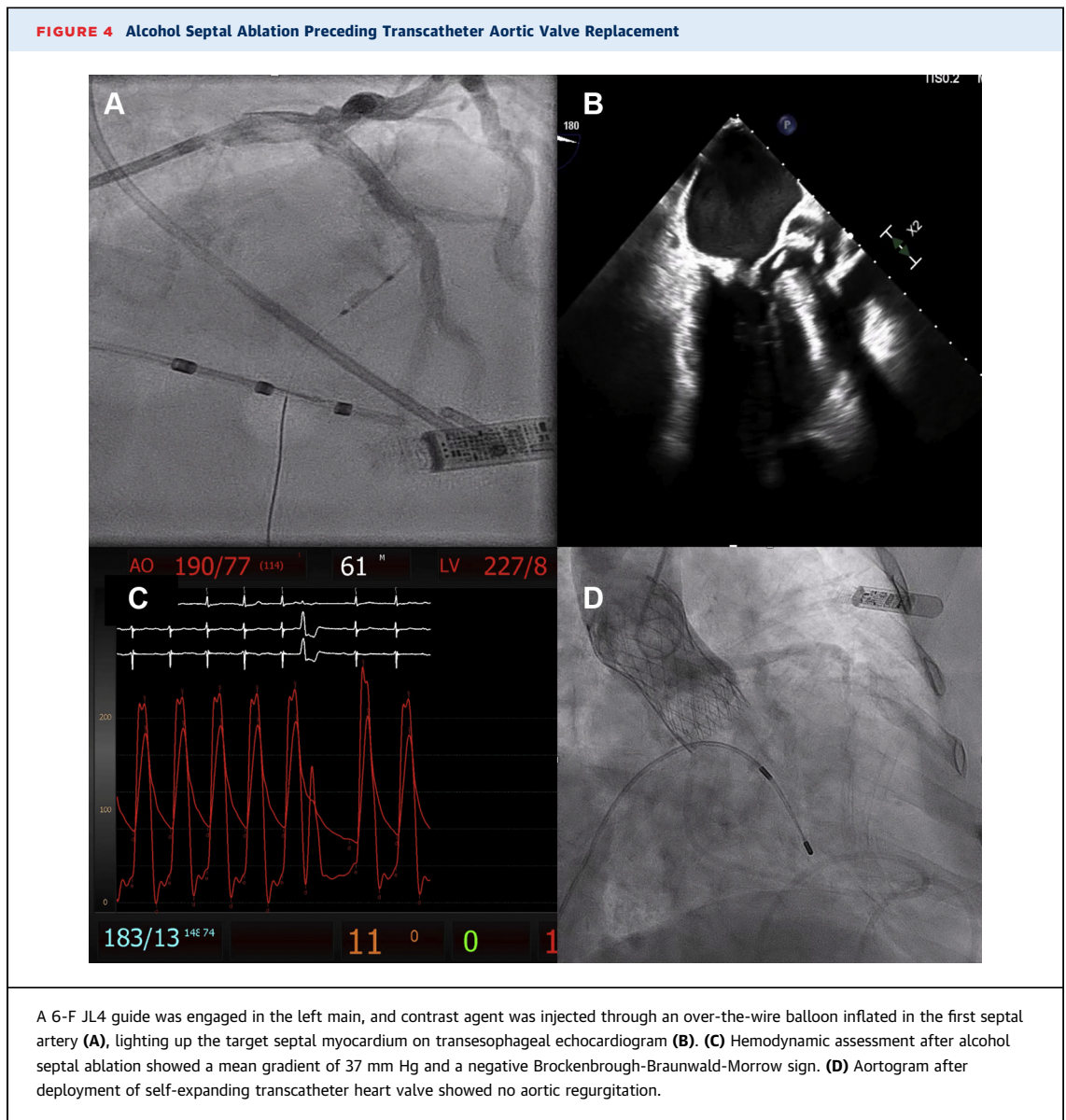
angiography showed nonobstructive coronary artery disease. CT imaging revealed no septal hypertrophy (Figure 5C). The patient was deemed to have high surgical risk and referred for transfemoral TAVR. A 26-mm self-expanding THV was deployed with good position, with no paravalvular leak and no gradient (Figures 5D and 5E). On follow-up, the patient's symptoms resolved. Post-THV deployment, there was no gradient across the valve. Post-TAVR echocardiography revealed a small hyperdynamic left ventricular cavity with almost complete cavity obliteration during systole and an intracavitary gradient of 120 mm Hg with SAM (Figure 5F). The patient was given 200 mg of oral metoprolol succinate with resolution of the intracavitary gradient on 3-month follow-up echocardiogram.

DISCUSSION

Immediate relief of aortic valve obstruction in patients with significant LVOT obstruction and concomitant severe AS can result in an immediate

decrease in afterload, acutely worsening LVOT obstruction and leading to severe hemodynamic instability. Deciding which patients can tolerate TAVR and which should undergo staged septal reduction before TAVR remains a challenge (Figure 6).

For patients with severe AS and concomitant LVOT obstruction undergoing surgery, myectomy is often performed in conjunction with surgical aortic valve replacement.¹ For patients referred for TAVR, however, the decision to pre-emptively treat their LVOT obstruction remains difficult. A large, nationally representative study found that the presence of LVOT obstruction in patients undergoing TAVR was associated with markedly increased in-hospital renal failure, cardiogenic shock, and mortality.² In patients with severe LVOT obstruction undergoing TAVR, ASA has been described as an effective means to reduce LVOT gradients before TAVR, thereby reducing the likelihood of post-TAVR hemodynamic collapse.³ The ideal time frame remains unclear but is likely to be at least 4 to 6 weeks before TAVR to allow acute edema to resolve and myocardial debulking to occur, as



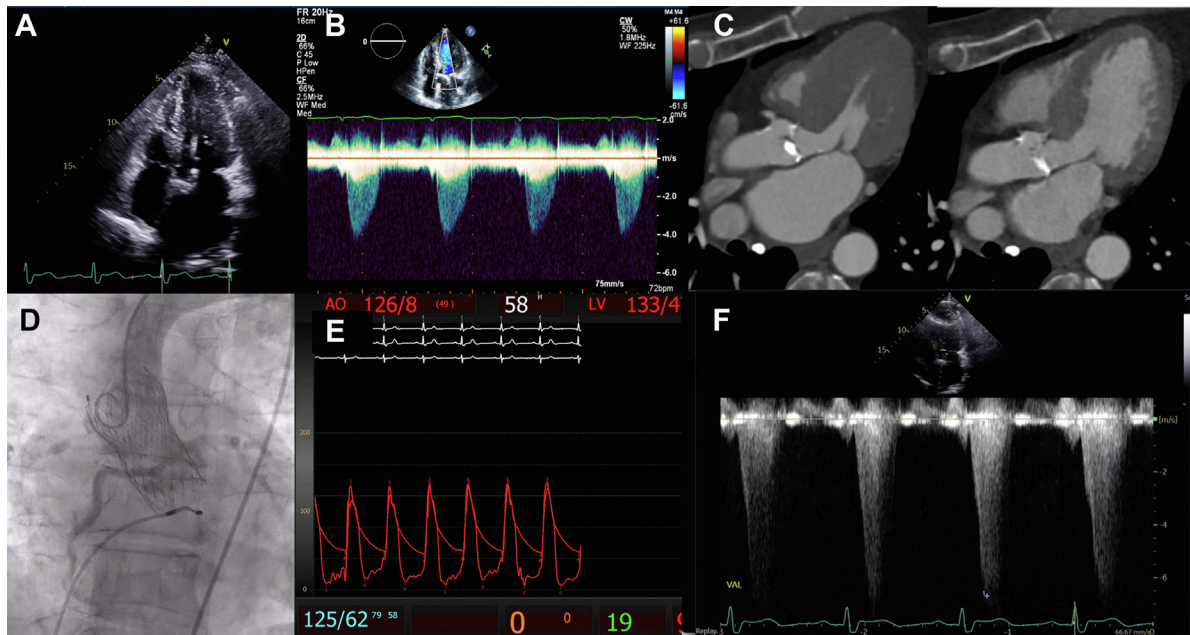
shown in our second case. Transcatheter edge-to-edge repair using the MitraClip (Abbott) has been described as an alternative to ASA for nonsurgical management of symptomatic SAM.⁴ This is potentially a useful strategy in patients with normal septal thickness (<15 mm), absent suitable septal perforator artery, or a target septal artery that collateralizes a chronically occluded right coronary artery and, therefore, cannot be safely occluded.⁵

In cases of post-TAVR acute hemodynamic collapse due to LVOT obstruction, the first step is fluid resuscitation, then discontinuation of inotropes (eg, norepinephrine, epinephrine, milrinone, dobutamine) and initiation of α_1 -agonists (eg,

phenylephrine) to increase afterload and beta-blockers to slow the heart rate. Temporary pacing from the right ventricular apex may improve LVOT obstruction in some patients.

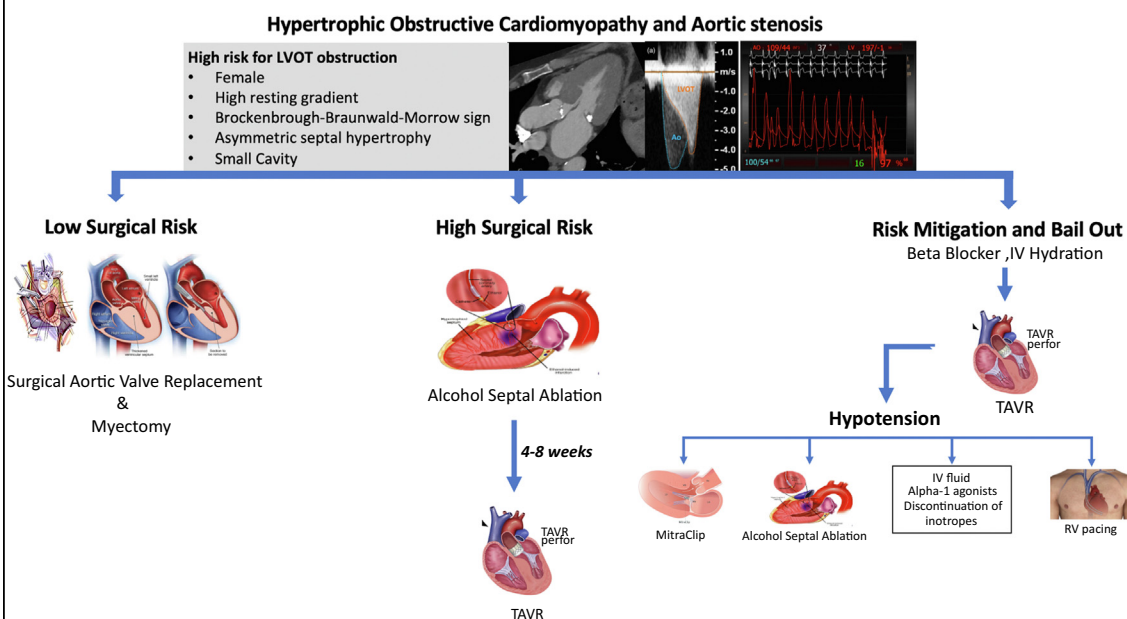
There are no data to support one valve over the other for patients with AS and hypertrophic obstructive cardiomyopathy. Patients receiving a balloon-expandable THV may be at an increased risk for THV embolization during deployment and interaction with the septum. Certain self-expanding THVs allow for repositioning deeper into the LVOT, over the septal bulge. However, coronary re-access might be more difficult when ASA is needed through a self-expanding THV.

FIGURE 5 Transcatheter Aortic Valve Replacement Patient With Hypertrophic Obstructive Cardiomyopathy Aggravating Left Ventricular Outflow Tract Gradient Treated Medically



(A) Transthoracic echocardiogram showing concentric hypertrophy. (B) Continuous-wave Doppler revealed a mean gradient of 50 mm Hg with typical aortic stenosis pattern. (C) Computed tomography scan demonstrating concentric hypertrophy and mild septal bulge but no systolic anterior motion. (D) Aortogram deployment of self-expanding transcatheter heart valve (29 mm) showed no aortic regurgitation. (E) Hemodynamic assessment showed no gradient across the valve. (F) Continuous-wave Doppler tracing demonstrating typical dagger-shaped pattern with gradient increased to 120 mm Hg.

FIGURE 6 Algorithm for Treating Patients With Severe Aortic Stenosis and Hypertrophic Obstructive Cardiomyopathy at High Risk for LVOT Obstruction



IV = intravenous; LVOT = left ventricular outflow tract; RV = right ventricular; TAVR = transcatheter aortic valve replacement.

CONCLUSIONS

We present 3 cases that illustrate the complex hemodynamic interplay between severe AS and concomitant LVOT obstruction with a wide range of presentations and challenges in treatment. Further studies are needed to define more definitive criteria when determining which patients should undergo pre-emptive septal reduction before TAVR.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Rogers has served as proctor and consultant for Medtronic and Edwards Lifesciences; served on the Advisory Board for Medtronic; has equity interest in Transmural Systems; and is a co-inventor on patents for dedicated transcatheter electrosurgery devices, assigned to the National Institutes of Health. Dr Weissman is director of an academic

cardiac computed tomography core laboratory with institutional contracts with Ancora Heart and LivaNova. Dr Waksman has served on the advisory boards of Abbott Vascular, Boston Scientific, Medtronic, Philips IGT, and Pi-Cardia Ltd; has served as a consultant for Abbott Vascular, Biotronik, Boston Scientific, Cordis, Medtronic, Philips IGT, Pi-Cardia Ltd, Swiss Interventional Systems/SIS Medical AG, Transmural Systems Inc, and Venous MedTech; has received grant support from AstraZeneca, Biotronik, Boston Scientific, Chiesi, Medtronic, and Philips IGT; served on the Speakers Bureau for AstraZeneca; and is an investor in MedAlliance and Transmural Systems Inc. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Ron Waksman, MedStar Washington Hospital Center, 110 Irving Street, NW, Suite 4B-1, Washington, DC 20010, USA.
E-mail: ron.waksman@medstar.net. Twitter: [@ron_waksman](https://twitter.com/ron_waksman).

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KEY WORDS aortic stenosis, left ventricular outflow tract obstruction, transcatheter aortic valve replacement

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