Transcatheter Aortic Valve Replacement in High-Risk Surgical Patient with Severe Aortic Insufficiency

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Abstract

Background: Transcatheter aortic valve replacement (TAVR) is approved by the FDA for severe aortic stenosis (AS) in patients of all surgical risk categories but has yet to be studied for its utility in aortic insufficiency (AI), despite the need for a safe alternative to surgery for prohibitive surgical risk patients.

Case Report: We describe a case of a female patient who presented with acute decompensated congestive heart failure (CHF) with New York Heart Association (NYHA) Class IV symptoms. She was found to have severe AI leading to acute decompensation. Two years prior to this, she had aortic valve endocarditis that had potentially resulted in severe AI. Considering her underling comorbidities including diabetes mellitus, hypertension, morbid obesity and multiple myeloma on active chemotherapy at the time of evaluation, the patient was a high-risk surgical candidate for surgical aortic valve repair (SAVR) in view of elevated risk of mortality, infection, and poor wound healing. After critical and comprehensive assessment, transcatheter aortic valve intervention was considered to be an appropriate choice of treatment. TAVR was successfully performed that resulted in immediate improvement of aortic valve function. On subsequent follow-ups, she demonstrated markedly improved symptoms and reduced status to NYHA Class II HF symptoms. Conclusion: TAVR is a potential treatment modality for patients with severe AI who are poor surgical candidates for SAVR. We hope our case contributes to the growing pool of studies investigating the utility of TAVR procedure in patients with severe AI.

Keywords

TAVR, Aortic Insufficiency, Aortic Sclerosis, Congestive Heart Failure, Infective Endocarditis
1. Background

Transcatheter aortic valve replacement (TAVR) has evolved since its introduction in 2002 to become an effective alternative to surgical aortic valve replacement (SAVR) for severe aortic stenosis (AS). The latest U.S. Food and Drug Administration approval in 2019 now permits TAVR use in patients with severe AS of all surgical risk categories based on the evidence from the PARTNER trials [1]. The outcome reported in these studies included all-cause mortality, stroke, major vascular events, and 1-year rehospitalization. In non-surgical patients, TAVR was superior to the available alternative therapy of balloon valvuloplasty, with reduced all-cause mortality and 1-year rehospitalization [2]. In high-risk and intermediate-risk patients, TAVR was found to be non-inferior to SAVR [3] [4]. In low-risk patients, TAVR was found to be superior to SAVR with reductions in all-cause mortality, stroke, and rehospitalizations [5].

Despite its effectiveness in severe AS, the utility of TAVR has yet to be thoroughly investigated in patients with isolated aortic insufficiency (AI). It is noteworthy that up to 75% of AS patients may have unreported aortic regurgitation [6]. Currently, SAVR is a Class I recommendation for patients with symptomatic severe AI [7]. However, there are no published guideline recommendations for patients with prohibitive surgical risk. In the recent large retrospective study (n = 14,720) by Anas Alharbi et al. (2021) examining outcomes in patients with pure AI, 6.2% underwent TAVR while the rest were treated with SAVR [8]. This study found no difference between the two groups for in-hospital mortality but showed that patients who received TAVR had lower incidence of acute kidney injury, cardiogenic shock, postoperative respiratory complications, and lower length of hospital stay.

Pursuant to the available evidence for TAVR in AI, we report the case of our patient with severe symptomatic AI treated successfully with transcatheter approach. We describe pertinent imaging and valve sizing considerations.

2. Case Report

A 70-year-old female presented to our hospital for fatigue, worsening exertional dyspnea, and lower extremity edema present for more than a week. She had New York Heart Association (NYHA) Class IV symptoms. She did not have fever, cough, chest pain, palpitations, or syncopal episodes. Her medical history included aortic valve endocarditis with vancomycin-resistant enterococci (VRE) treated with antibiotics a year and a half ago, insulin-dependent diabetes, hypertension, hyperlipidemia, hypothyroidism, morbid obesity (BMI 47), obstructive sleep apnea (OSA), and bilateral deep venous thrombosis subsequently requiring inferior vena cava filter. She was also being treated with chemotherapy for multiple myeloma. She had never smoked, used alcohol, or used illicit drugs. Her family history included arrhythmia in her father, and heart attack and carotid endarterectomy in her brother.

On arrival, she was afebrile, hypertensive with a widened pulse pressure of
147/51 from a baseline blood pressure of 128/50, tachypneic and in moderate respiratory distress with oxygen saturation of 92% on ambient room air. On physical examination, she had crackles over the lungs, regular heart rate and rhythm with a grade III/VI diastolic murmur, 3+ pitting edema of her lower extremities, and chronic venous stasis-related skin discoloration without erythema or tenderness. There was no jugular venous distention or hepatomegaly.

Laboratory studies as demonstrated in Table 1 showed macrocytic anemia and pancytopenia, normal electrolytes, liver and renal functions. Her troponin was within normal range and N-terminal proBNP was elevated. Respiratory panel, urine culture, and blood cultures were all normal. Chest x-ray showed an enlarged cardiac contour with mild bibasilar disease (Figure 1). Electrocardiogram showed normal sinus rhythm. Transthoracic echocardiogram (TTE) revealed a preserved left ventricular ejection fraction (LVEF) of 50%, trileaflet AV with calcified leaflets and moderate to severe aortic regurgitation (AR), and moderately dilated left atrium (LA) (Figure 2). Transesophageal echocardiogram (TEE) revealed mildly dilated ascending aorta of 3.4 cm, severe AR with jet width > 65% of left ventricular outflow tract (LVOT), vena contracta (VC) 0.75 cm, AI P1/t2 252.6 msec, pressure half time (PHT) 361 ms and holodiastolic reversal as observed in descending aorta, Figure 3.

Table 1. Pertinent laboratory results with reference ranges.

<table>
<thead>
<tr>
<th>Lab</th>
<th>Results</th>
<th>Ref. Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin 1</td>
<td>&lt;0.012</td>
<td>&lt;0.034</td>
</tr>
<tr>
<td>N-Terminal proBNP</td>
<td>1960</td>
<td>&lt;300 pg/ml</td>
</tr>
<tr>
<td>BUN</td>
<td>22</td>
<td>7 - 25 mg/dL</td>
</tr>
<tr>
<td>Cr</td>
<td>0.9</td>
<td>0.5 - 1.5 mg/dL</td>
</tr>
<tr>
<td>WBC</td>
<td>2.5</td>
<td>4.0 - 10.5 10^3/μL</td>
</tr>
<tr>
<td>Hgb</td>
<td>11.1</td>
<td>12.0 - 17.0 g/dL</td>
</tr>
</tbody>
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Figure 1. 12-lead EKG shows normal sinus rhythm.
Figure 2. (A) Transthoracic echocardiography (TTE) parasternal long axis of aortic valve with color doppler showing severe aortic regurgitation. (B) TTE parasternal short axis of aortic valve showing aortic regurgitation. (C) Continuous Wave (CW) doppler showing severe aortic regurgitation based upon a rapid diastolic deceleration rate. The peak velocity across the aortic valve is normal and there is no aortic stenosis. LA: Left atrium. LV: Left ventricle.

Figure 3. Transesophageal echocardiography with color doppler (long and short axis views of the aortic valve) showing severe aortic regurgitation. LA: Left atrium.
She was admitted to the cardiac intensive care unit for acutely decompensated congestive heart failure (CHF) due to severe AI. After appropriate diuresis and clinical stabilization, patient underwent cardiac catheterization that revealed minimal atherosclerotic disease of the coronary arteries; however, left-sided filling pressures were found to be elevated along with severe pulmonary hypertension with a normal transpulmonary pressure gradient. She was a poor surgical candidate due to underlying comorbidities hence a transcatheter aortic valve replacement was considered. Patient was discharged home with the plan to explore compassionate use of TAVR for severe AI.

For valve sizing and anatomical orientation, CT TAVR angiogram was performed as shown in Figure 4. Two weeks later, patient underwent TAVR with a #26 SAPIEN-3 valve via left transfemoral access (Figure 5). Intraprocedural temporary pacemaker (TPM) was inserted. Incomplete left bundle branch block was

**Figure 4.** (A) CT aortic annulus plane. (B) CT aortic sinuses of Valsalva showing minor calcium deposits on the left coronary leaflet. (C) CT co-planar view of aortic root. RC: Right coronary leaflet. LC: Left coronary leaflet.

**Figure 5.** Aortic angiogram of TAVR valve post deployment (red arrow). (A) Safari tm x-small wire in the LV. (B) 5Fr balloon tipped pacemaker wire in the RV. (C) 5Fr pigtail in the aortic root. (D) Edwards Sapien-3 deployment system in the descending aorta.
noted on post TAVR EKG; however, patient continuously remained in sinus rhythm without a block thus TPM was discontinued. Post-procedure TTE showed a normal functioning TAVR valve with a normal gradient and without paravalvular leak or pericardial effusion (Figure 6). She was discharged the next day on dual antiplatelet therapy. On follow-up within a week, patient reported significant improved symptoms now NYHA Class II CHF. She was continued on dual antiplatelet therapy for 3 months with subsequent follow-ups in the outpatient setting.

3. Discussion

We present a case of acutely decompensated CHF due to severe aortic insufficiency in a patient with a history of resolved VRE aortic valve endocarditis. Due to her underlying immunocompromised status secondary to ongoing chemotherapy for multiple myeloma, diabetes mellitus, COPD, morbid obesity and OSA, she was deemed prohibitive surgical risk by our institutional interdiscipli

![Figure 6](image-url). Post TAVR Transthoracic echocardiography parasternal long (A) and short (B) axis of aortic valve with color doppler showing no paravalvular or central prosthetic leak. LA: Left atrium. LV: Left ventricle.

Considering TAVR has shown significant success in the treatment of calcific aortic valve stenosis, the potential utility of TAVR for pure AI is compelling. Prevalence of AI outnumbers that of AS up until the sixth decade of life [7]. The 2020 ACC/AHA guidelines place a Class I recommendation for SAVR for the treatment of severe AI [8]. There are, therefore, limited treatment options for severe AI patients who cannot undergo surgery. In this high-risk surgical group of patients with severe AI, alternative less invasive treatment modality such as TAVR approach can be considered.

Acute AI is most often due to endocarditis or aortic dissection [8] [9] [10]. Our case is unique in that severe AI occurred due to valve degeneration related to prior treated endocarditis rather than active infection. Established guidelines recommend early surgery for infective endocarditis caused by highly resistant
organisms, such as VRE [10]. However, in the case of our patient, she was successfully treated for endocarditis as shown by the absence of AV vegetation on echocardiography, negative blood cultures and clinical improvement. A more frequent surveillance echocardiography studies to interrogate AV post endocarditis treatment might have potentially led to early detection of valvular deterioration.

We propose that patients undergoing TAVR for treatment of pure AI should undergo the standard institutional protocol for TAVR CT scan imaging. Particular attention should be placed on the amount of AV leaflet calcification, as this may aid in valve positioning and skirt seal. The presence of mild leaflet calcification in our patient was thought to lend to the successful valve deployment. The prosthetic valve would more likely adhere to the calcifications on the valve leaflets. If no significant LVOT or sinotubular junction (STJ) calcium is present, we propose that valve sizing with the SAPIEN-3 platform should be at least 20% above CT derived aortic annulus area. This will help ensure proper valve positioning, seal and limit degree of paravalvular leak. Careful CT-guided procedure planning should be performed. It is often difficult to visualize the co-planar view during valve deployment due to lack of calcium and presence of severe AI. Valve deployment should be approximately “60/40” (60% aortic) in order to ensure sufficient contact of the sealing skirt with the LVOT, and prevent valve embolization.

Although our report has significant inherent limitations by design, we ponder that the utility of TAVR as a treatment modality for patients with severe AI with prohibitive surgical risk remains an unexplored area. We reason that our experience will add to the slowly growing literature on this subject.

4. Conclusion

This case demonstrates the successful use of TAVR in a patient with severe AI who is a poor surgical candidate. This can be further investigated in large prospective studies and randomized-controlled trials for its efficacy and safety.

Informed Consent

The case was reviewed by the Institutional Review Board and informed consent was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References


