Transcatheter Aortic Valve Replacement

Alcohol Septal Ablation as a Bail-Out Procedure for Suicide Left Ventricle After Transcatheter Aortic Valve Implantation

Ulrich Gerckens, MD, Luciano Pizzulli, MD, Konstantinos Raisakis, MD

ABSTRACT: With the advent of transcatheter aortic valve implantation (TAVI), many AS patients, formerly considered inoperable, can receive effective treatment. The relief of the left ventricular pressure overload could lead, in some cases, to the occurrence of dynamic intracavity pressure gradients (DIG) with deleterious clinical impact. This phenomenon resembles the physiology seen in hypertrophic obstructive cardiomyopathy. We report a case in which alcohol septal ablation was used as a bail-out therapy for the acutely developed intracavity obstruction after TAVI. Potential dynamic intracavity gradients should always be excluded in the acutely deteriorated patient postoperatively. Alcohol septal ablation can be considered as a salvage therapeutic tool when other therapies are ineffective to treat subvalvular obstruction.

Key words: aortic stenosis, transcatheter aortic valve implantation, alcohol septal ablation, hypertrophic obstructive cardiomyopathy, suicide left ventricle

Although aortic valve replacement (AVR) is currently the treatment of choice for patients with symptomatic severe aortic stenosis (AS), such an operation can be withheld in elderly patients with high operative risk.1 With the advent of transcatheter aortic valve implantation (TAVI), many AS patients, formerly considered inoperable, can receive effective treatment. This less invasive alternative technique is associated with favorable results in mortality and quality of life when compared with standard medical therapy and its outcomes may be comparable to those achieved with conventional surgical therapy.2

The presence of asymmetric septal left ventricular hypertrophy (LVH) is not uncommon in the setting of AS, which in some cases could lead to dynamic intraventricular gradients (DIG), acutely after the valvular obstruction is removed. This phenomenon resembles what is seen in hypertrophic obstructive cardiomyopathy (HOCM), which contributes significantly to the development of debilitating symptoms in a subset of patients. Alcohol septal ablation (ASA) has emerged as an alternative to septal myectomy, and is a less invasive method for the treatment of the drug-refractory symptoms caused by HOCM.

In this report, we describe a patient in whom DIG with hemodynamic collapse was unmasked shortly after TAVI was performed. Since medical therapy failed to improve his critical condition, ASA was successfully selected as a bail-out therapeutic choice.

Case Presentation

Clinical evaluation. The patient was an 88-year-old woman referred to our institution for consideration for TAVI. She was admitted to the hospital two months prior due to acute chest pain and electrocardiographic changes (negative T-waves in V5 and V6 leads). Acute coronary syndrome was excluded and she was diagnosed with severe degenerative aortic stenosis. She complained of a 6-month history of stable angina, as well as progressively worsening dyspnea (New York Heart Association functional class III congestive heart failure).

Her cardiovascular history included arterial hypertension and dyslipidemia. Her past medical history was comprised of mediastinitis due to perforated Zenker’s diverticulum, sigmoid diverticulitis, chronic pancreatitis, cholecystectomy, and hysterectomy. On physical examination, her blood pressure was 126/55 mm Hg and her heart rate was 78 bpm. Lung examination was unremarkable. There was an ejection systolic murmur best audible on the right second intercostal space radiating to the carotids, with markedly diminished aortic component.

A transesophageal echocardiogram was performed, which demonstrated thickened and calcified aortic valve cusps with reduced mobility. The mitral valve was mildly thickened and calcified with mild subvalvular thickening. The left ventricle was normal in dimensions, with hypertrophied walls more pronounced in the interventricular septum (19 mm). Global left ventricular systolic function was normal, with no regional wall motion abnormalities (ejection fraction, 60%). Peak aortic velocity by using continuous wave Doppler across the aortic valve measured 5.7 m/s, with peak pressure gradient of 115 mm Hg and mean pressure gradient of 92 mm Hg. Effective aortic orifice area estimated with continuity was 0.6 cm². Estimated mitral valve orifice area was 1.1 cm² by using pressure half-time across the mitral valve. Mild aortic and mitral regurgitation were identified. Coronary angiogram showed no significant coronary artery stenosis.

Aortic valve replacement was indicated based on the described symptoms and echocardiogram findings. The patient was referred to the cardiac surgeon team. Despite the relatively low STS operative mortality risk score of 7.06%, she was declined for an open aortic valve replacement due to chronic mediastinitis from the previous Zenker’s diverticulum resection. The patient was reviewed by the TAVI team and allocated for percutaneous valve implantation after she was found to meet the appropriate criteria for the intervention.
Pre-TAVI multislice computer tomography scan measured the aortic annulus at 21 mm. Moderate aortic valve calcification was present. No significant aorto-iliac disease was identified. Transesophageal echocardiogram was not performed due to the patient's refusal.

**Transcatheter aortic valve implantation.**

An active pacemaker lead was fixed in the right ventricular apex via right jugular access the day before the procedure, while the pacemaker device remained extracorporeal. Right transfemoral access was obtained using a 16 Fr expandable sheath under local anesthesia and mild sedation. The mean pressure gradient across the aortic valve by using a pigtail catheter measured 114 mm Hg. Due to the presence of moderate valve calcification, the preimplantation balloononing strategy was preferred. Balloon aortic valvuloplasty was performed with rapid ventricular pacing, using a 20 x 45 mm Loma Vista balloon (Loma Vista Medical) over an Amplatz Extra-Stiff wire (Cook Medical). A 23 mm Edwards SAPIEN aortic valve prosthesis (Edwards Lifesciences) was then advanced and successfully implanted with rapid pacing of 190 bpm. The hemodynamic profile of the patient throughout and immediately after the procedure was stable. Supravalvular aortography demonstrated only mild perivalvular leak. Due to the relatively deep, vulnerable to dislocation positioning of the valve, and given that the hemodynamic parameters of the patient remained unchanged, no re-crossing of the Edwards valve with a pigtail catheter was attempted. The patient was alert and feeling well, and was admitted to the Cardiac Care Unit.

Twelve hours later, the patient complained of shortness of breath while her blood pressure decreased to 90/60 mm Hg. On examination, crackles on both lung bases and a harsh systolic murmur were heard. Inotropic support with intravenous administration of doputamine (initially 5 µg/kg/min, which then titrated to 5 µg/kg/min) led to paradoxical response, with further dropping of the blood pressure and worsening of patient's clinical status. A new echocardiogram was performed, which demonstrated a small left ventricle with hyperdynamic systolic function. Midcavity obstruction during systole was identified. New moderate mitral regurgitation related to systolic anterior motion (SAM) of the anterior leaflet of the mitral valve was present, consistent with HOCM physiology. The Edwards valve was in situ and mild paravalvular regurgitation was documented. No pericardial fluid was identified. Peak aortic velocity by using continuous-wave Doppler across the aortic valve and the left ventricular cavity measured

---

**Figure 1.** Transthoracic echo 12 hours after TAVI. (A) Apical four-chamber view at end diastole. Hypertrophy of the left ventricle more pronounced in the basal septal and lateral walls. (B) Apical four-chamber view at end systole. Left ventricle mid-cavity obstruction. (C) Continuous-wave Doppler across the aortic valve-LVOT shows a peak velocity of 5.2 m/s with peak pressure gradient of 108 mm Hg. (D) Color Doppler on apical four-chamber view showing moderate mitral regurgitation.

**Figure 2.** Invasive estimation of pressure gradient. (A) The pigtail catheter is positioned at the subvalvular area. (B) No significant pressure recorded across the prosthetic valve. (C) The pigtail catheter is positioned at the apex. (D) A mean pressure gradient of 79 mm Hg was documented.
5.2 m/s with peak pressure gradient of 108 mmHg (Figure 1). As soon as HOCM physiology with mid-left ventricular cavity obstruction was identified, intravenous dobutamine administration was discontinued. Conservative measures aiming to increase left ventricular filling pressures were applied. Prompt intravenous administration of normal saline (500 mL over 1 hour), and decrease of heart rate (pacemaker adjusted to 55 bpm, intravenous beta-blockers) failed to improve the patient's condition. Emergent invasive estimation of the valve function was decided. The patient was taken to the cardiac catheterization laboratory, where obstructive coronary disease and the presence of intracoronary thrombus were excluded. The Edwards prosthetic valve was crossed with a pigtail catheter and pressure gradients were measured both across the valve and in the left ventricular cavity. With the catheter positioned in the subvalvular area, the mean pressure gradient measured was 12 mm Hg, while the mean pressure gradient measured with the catheter placed in the ventricular apex was 79 mm Hg (Figure 2). On left ventriculography, there was obliteration of the mid-ventricular cavity in systole with significant mitral regurgitation identified (Figure 3). Given the above findings, the patient's deterioration was attributed to HOCM physiology developed after the removal of aortic pressure overload. Since conservative measures didn't improve the patient's status, ASA was performed as a bail-out strategy.

The active pacemaker lead used for the TAVI procedure was still in situ. Due to a large septal area involved to the intracavity obstruction, we decided on a strategy of 3 septal perforator ablation. The Edwards 3.5 coronary angioplasty catheter was positioned at the ostium of the left coronary artery. A Whisper 0.014˝ angioplasty wire (Abbott Laboratories) was advanced into the first large septal branch followed by the passage of a 2.0 x 15 mm over-the-wire balloon. The angioplasty balloon was inflated and a small quantity of contrast was injected through the central lumen of the balloon, in order for the involved septal obstructive area to be verified. An alcohol injection of 1.5 mL was then slowly administered over 5 minutes. The balloon was deflated and then removed 3 minutes after the injection. The same procedure was performed to the second and third septal branch with administration of 1.25 mL of alcohol to each vessel. The final coronary angiogram verified septal branch occlusion and the hemodynamic control with the use of the pigtail catheter showed an acute decrease to intracavity pressure gradient of 21 mm Hg (Figure 4). The patient developed third-degree atrioventricular block and the transient pacemaker adjusted to 70 bpm. She was transferred to the coronary care unit with improved symptoms and stable hemodynamic profile. A new echocardiogram was performed the day after the procedure, and showed normal global left ventricular systolic function with septal akinesia. Peak pressure gradient using continuous-wave Doppler across the aortic valve and the left ventricular cavity measured 17 mm Hg. Mild mitral regurgitation unrelated to the SAM phenomenon was identified. The patient remained asymptomatic and uncomplicated for the following 7 days. A permanent two-chamber pacemaker was implanted and the patient was discharged from the hospital 2 days later.

**Discussion**

Persistent high transvalvular gradient after TAVI procedure can be attributed to different causes. Thrombus formation as well as “frozen” valve leaflet could limit valve outflow, leading to acute deterioration of the patient's clinical condition. Dynamic subvalvular obstruction due to hypertrophy has been equally recognized as a potential cause that could be interpreted cautiously.

Long-standing, slowly progressive aortic stenosis induces substantial functional and morphological changes in the LV. Chronic pressure overload results in LVH, which is the main
compensatory mechanism in order for the stroke volume and normal ejection fraction to be maintained. Although LVH secondary to AS is usually concentric, in approximately 10% of patients, according to the existing data, a non-uniform septal pattern of hypertrophy is identified. Whether this asymmetric LVH is caused by concomitant primary HOCM or is only secondary to the LV adaptation mechanisms during the course of AS is not easily clarified on echo beforehand. Moreover, echocardiographic diagnose of subaortic stenosis and the degree of its contribution to the overall pressure gradient across the valve-LVOT system is challenging in the context of severe aortic stenosis. Independently of the cause, acute relief of the high-pressure overload status by either AVR or TAVI, in the presence of asymmetric LVH and hyperdynamic left ventricular systolic function, could lead to DIG and hemodynamic collapse.

Prophylactic septal myectomy at the time of AVR has been recommended when DIG caused by residual subaortic obstruction is anticipated. Predictive echocardiographic factors have been reported and comprise small LV diameters, good overall contractility, discrete asymmetric hypertrophy, high transvalvular gradients, and small LV outflow tracts. Surgical myectomy doesn’t seem to increase the operative risk and contributes to a grater LV mass regression postoperatively.

In the TAVI era, high operative risk patients with symptomatic aortic stenosis receive less invasive, alternative treatment. Although regression of LVH has been documented within the first 6 months, these patients are also susceptible to postprocedural DIG, similar to the surgical patients. Suh, et al described a case where HOCM physiology and immediate clinical deterioration developed soon after TAVI was performed. They termed the condition “suicide left ventricle,” resembling the anatomical and clinical characteristics sometimes seen in the right ventricle after valvular pressure afterload relief. In their case, clinical improvement was accomplished after conservative measures were taken. Filling pressure increase by intravenous fluid administration as well as decrease of inotropy by giving beta-blockers were sufficient to improve the patient’s condition. In case of conservative treatment failure, surgical myectomy is not an option for this group of patients, since they have already been declined for an open surgery due to the existence of compelling contraindications.

Alcohol septal ablation is a well-established therapy for the treatment of eligible patients with HOCM physiology. Candidates for the method are patients with severe drug-refractory symptoms, septal hypertrophy, and dynamic LVOT gradient when septal perforator artery anatomy is favorable. This method could be an alternative solution to medical refractory suicide left ventricle after TAVI. Sorajja, et al reported on a case that was successfully treated with ASA 6 months after TAVI. The patient’s recurrent symptoms were attributed to dynamic LVOT obstruction and worsened despite optimal medical therapy. The patient was free of symptoms at 3-month follow-up.

To the best of our knowledge, our case is the first description of using ASA as a bail-out therapy for DIG after TAVI. Conservative measures were taken first, but failed to improve the patient’s condition. Given that the patient was not a surgical candidate, no other therapeutic options were available for relieving the acutely developed hemodynamic collapse. Thus far, there are no well-established prophylactic measures, similar to the combined AVR-myectomy operation, that could be taken during the TAVI procedure in order for an acute or long-term LVOT obstruction to be avoided. Deeper valve implantation covering part of the hypertrophied LVOT has been shown to be helpful in particular cases, especially when longer types of valve are used. Yet, the possible favorable result should always be balanced with the higher risk of postprocedural development of significant aortic regurgitation. Moreover, this strategy could not be applied to cases where septal hypertrophy causes mid-cavity obstruction. Hence, care for maintaining normal LV filling pressures and decreasing heart rate and inotropy should be taken during the immediate post-TAVI period when DIG is anticipated. Administration of inotropes could lead to deterioration of the patient’s hemodynamic parameters and should be avoided. In those cases where conservative therapy fails to provide symptomatic relief, ASA, although not yet indicated as a routine additional prophylactic procedure, should be considered as an emergency solution.

Conclusion
TAVI is an efficient, less invasive alternative therapy for high-risk patients with severe aortic stenosis, with outcomes comparable to those achieved with conventional surgical valve replacement. Although valvular obstruction removal is accomplished, the method fails to confront potential subvalvular obstruction, which could result in hemodynamic deterioration shortly after the procedure. Although ASA could not be applied, thus far, as a preventative method when asymmetrical LVH is documented in the pre-TAVI screening, this case demonstrates that ASA should be considered as an efficient adjunctive to TAVI for urgent therapy in the subgroup of patients developing suicide left ventricle.

References