Subacute Aortic Root and Valve Thrombosis following Transcatheter Aortic Valve Replacement in a Left Ventricular Assist Device Patient: From One Problem to the Next

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INTRODUCTION

Up to 30% of patients with continuous-flow left ventricular assist devices (LVADs) develop moderate to severe aortic regurgitation (AR).1 It has been associated with an increased risk of rehospitalization and mortality, prompting either surgical or more recently percutaneous intervention.1 We present a case of an LVAD patient with ongoing symptoms of heart failure in the setting of severe AR. He underwent transcatheter aortic valve replacement (TAVR) complicated by thrombosis, leading to heart transplantation.

CASE PRESENTATION

A 71-year-old black male with a history of chronic atrial fibrillation, a cerebrovascular accident without residual deficits, chronic kidney disease, and heart failure with reduced ejection fraction secondary to a nonischemic cardiomyopathy complicated by ventricular tachycardia (VT) underwent placement of a Heartmate 3 LVAD for treatment of his end-stage cardiomyopathy with a concomitant central apoposition suture (Park stitch) of his aortic valve to correct a degenerative valve with nodular thickening and central AR. At the time, he was deemed not to be a candidate for cardiac transplantation due to age and frailty.

Following LVAD implantation, he continued to experience a severe reduction in his exercise capacity. Invasive hemodynamics with an LVAD RAMP study on postoperative day (POD) 57 (Table 1) showed improvement in filling pressures and CO despite serial increases in LVAD speed up to 6,100 RPM and aggressive medical management. Transthoracic echocardiogram showed moderate to severe AR, previously noted as only mild on his initial postoperative transthoracic echocardiogram on POD 35.

He was rehospitalized on POD 119 with heart failure symptoms and a transesophageal echocardiogram (TEE) showed severe eccentric AR (Figure 1, Video 1) due to what appeared an eccentrically located ruptured stitch of the central opposition suture performed at the time of implant. Doppler analyses of the LVAD inflow cannula confirmed normal flow through the device (Figure 2). Options for management of the AR were the use of an Amplatzer atrial septal defect occluder device, TAVR, or surgery. After extensive discussions, the heart team decided to proceed with TAVR, and on POD 165 TAVR was successfully performed under cardiopulmonary bypass with an oversized 29 mm Sapien 3 valve (Edwards Lifesciences, Irvine, CA), using an additional 5 mL in-valve balloon followed by balloon postdilatation with an additional 4 mL. Transesophageal echocardiogram showed stable Sapien 3 position with trivial paravalvular AR (Figure 3). He clinically improved, with normalization of hemodynamics. Seven days after TAVR implant, he had an episode of sustained VT requiring defibrillation and was placed on amiodarone. He was discharged 9 days post-TAVR.

Table 1 Invasive hemodynamic RAMP study

|               | Baseline | RAMP 1 | RAMP 2 |
|---------------|----------|--------|--------|
| Speed, RPM    | 5,500    | 5,900  | 6,100  |
| Device flow, L/minute* | 4.3      | 5.1    | 5.4    |
| Right atrium, mm Hg | 9        |        |        |
| Right ventricle, mm Hg | 53/9     |        |        |
| PA, mm Hg     | 53/24 (34) | 54/26 (35) | 55/25 (35) |
| PA O2 sat, %  | 59       | 62     | 60     |
| PCWP, mm Hg   | 24 (v 35) | 22 (v 30) | 20 (v 30) |
| Aorta, mm Hg  | 98/78 (85) | 104/82 (89) | 136/94 (108) |
| Aorta O2 sat, % | 98       | 98     | 98     |
| CO, L/minute Fick | 3.5      | 4.2    | 4      |
| Cardiac index, L/minute/M² | 1.6      | 1.9    | 1.8    |
| PVR, Wood units | 2.9      | 3.1    | 3.8    |
| Heart rate, bpm | 90       | 90     | 90     |

PA, Pulmonary artery; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; Sat, saturation. Values in parenthesis indicate mean values.

*Estimated LVAD flow.
Ten days later, he presented with more implantable cardioverter-defibrillator discharges due to sustained monomorphic VT. Computed tomography angiography was ordered to evaluate the LVAD, cannulas, and left ventricular size. It revealed complete thrombosis of the Sapien 3 valve and adjacent sinuses of Valsalva without coronary artery occlusion (Figure 4). The peak CK-MB was 5.7 (0.6-6.3 ng/mL), and the troponin I was 0.89 (0-0.03 ng/mL). A probable cause for his presentation at the time was thought to be coronary embolism. There was no occlusive coronary lesion seen on the computed tomography angiography performed, but it was not protocolled for coronary evaluation. A coronary angiogram was not performed as it would carry extreme risk given the presence of aortic root thrombus. The VT morphology was suggestive of a focus in the posterior part of the left ventricle. The patient continued to have VT, which was eventually controlled on lidocaine and dofetilide. His case was presented at the heart transplant selection meeting, and he was listed for transplantation as the risk of continued device therapy was felt to be very high due to his electrical instability and valve thrombosis. He underwent successful cardiac transplantation on the

![Figure 1](image-url)  
**Figure 1** Transesophageal image of the aortic valve in the transgastric view. Biplane imaging is utilized to visualize the aortic valve (blue asterisk) and mitral valve (red asterisk) in end diastole. The bottom panels demonstrate color Doppler of the same biplane view with significant AR noted.
same admission. He was doing well at his 5-month follow-up visit. Intraoperatively, the extracted Sapien 3 valve was found to be completely thrombosed on the aortic side (Figure 5).

**DISCUSSION**

Aortic regurgitation is being seen more commonly with prolonged continuous-flow LVAD support and is related to a number of factors including commissural fusion, which has been associated with infrequent valve opening, elevated retrograde aortic pressures leading to root dilatation, and myxoid leaflet degeneration. Data are limited and stem mainly from case series and reports. It is described as a progressive process, making serial echocardiography an important tool in recognizing worsening AR. Comorbidities such as pulmonary hypertension, peripheral vascular disease, and chronic kidney disease have been associated with worsening AR over time. In addition, women, elderly patients, and patients with a body surface area <2 m² are at higher risk. For this reason, when there is evidence of AR at the time of LVAD implant, aortic valve leaflets are commonly stitched together. However, the literature reports limitations associated with the Park stitch method as its durability is in question.

Aortic valve closure with (off-label) use of Amplatzer atrial septal defect closure devices has been reported; concerns of superimposed thrombosis reaching out to the level of the coronary ostia do exist. The collective experience of TAVR to address severe AR in LVAD patients is growing, with favorable reported outcomes. However, complications can be encountered due to the lack of leaflet calcification to anchor the implanted valve and due to LVAD suction forces, resulting in device migration as well as paravalvular regurgitation. This complication can be prevented as in this case by oversizing the dilation balloon and turning down the LVAD speed during the implantation; with our
patient, the latter was enabled by the use of a cardiopulmonary bypass circuit.

Interestingly, in our case the significant thrombus burden was found 21 days after TAVR and was associated with increased episodes of VT. The frequency of aortic valve opening may have contributed to thrombus formation. Following TAVR, several attempts were made to increase the low frequency of the aortic valve opening by way of speed adjustments; however, it would not open even at lower speeds and we did not want to compromise LVAD flow to obtain this.

CONCLUSION

Patients are living longer on LVAD support, but this has led to an increase in some adverse events, including the development of significant AR. Percutaneous treatments will be considered more frequently in the future due to improving device technology along with operator and institutional expertise. As AR presents late in the course of LVAD therapy, we expect this to occur more so in an older patient population, further making TAVR the intervention of choice to salvage AR, as surgical options would carry higher risk in this population.

Our case highlights the need for future research on the optimal management of these patients. It is essential to investigate proper LVAD and valve management strategies including appropriate speed settings as it relates to frequency of aortic valve opening, suitable antithrombotic strategies, and ideal valve type and LVAD combinations. This will result in further reduction in the risk associated with treatment of hemodynamically significant AR, a serious and relatively common LVAD complication.

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2020.12.005.

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Figure 4  (A) Postprocedural computed tomography (CT) shows the ostium and proximal portion of the left main coronary artery (red asterisk) and significant thrombus within the implanted transcatheter aortic valve (orange dagger).  (B) Postprocedural CT shows the ostium and proximal portion of the right coronary artery (yellow asterisk).

Figure 5  Thrombosed transcatheter aortic valve seen at the time of cardiac transplantation.