



Original Research

Management of Aortic Insufficiency in Patients with Left Ventricular Assist Device – a Retrospective Analysis

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Abstract

Background

Aortic insufficiency is increasingly recognized as a complication of left ventricular assist device (LVAD) support and may lead to clinical decompensation requiring correction. This article describes experiences in managing patients presenting with concomitant aortic insufficiency and with de novo aortic insufficiency following left ventricular assist device implantations.

Methods

All patients undergoing LVAD implantation between 2012 and 2014 were included in this retrospective analysis if aortic valve insufficiency was present on implantation or newly developed (de novo) after implantation. Moderate to severe aortic valve insufficiency was corrected at implantation.

Results

The data of 39 patients were included. At the time of LVAD implantation, moderate to severe aortic valve insufficiency was present in 3 patients and was corrected by bioprosthetic valve replacement (2 patients) and by bioprosthetic valve replacement associated with ascending aorta with hemi arch replacement with a graft due to ascending aortic aneurysm (one patient). Four patients developed moderate to severe aortic insufficiency after LVAD surgery. Treatment with



conservative medical management was successful in 3 patients. One patient underwent transcatheter aortic valve occlusion using an Amplatzer closure device after failure of medical management.

Conclusions

Concomitant aortic valve replacement with LVAD implantation is a safe and viable option in managing aortic valve insufficiency. De novo aortic insufficiency may lead to recurrent heart failure and presents a clinical treatment challenge following successful LVAD support; the most appropriate and effective treatment option awaits definition.

Keywords: aortic insufficiency, left ventricular assist device, recurrent heart failure

Introduction

The number of advanced heart failure patients treated with mechanical ventricular assist devices for either bridge-to-transplant therapy or for permanent destination therapy is growing.¹ This expansion led to an increase in the number of patients with concomitant valvular heart disease,² and clinicians are now faced with adapting treatment plans to multi-diagnosis conditions. Whereas moderate to severe mitral valve regurgitation is common in advanced heart failure and can be successfully treated with left ventricular assist device (LVAD) implantation,³ aortic valve pathology, specifically moderate to severe aortic insufficiency (AI), is uncommon at the time of LVAD implantation. Pal and colleagues reported moderate to severe aortic insufficiency in only 4% of a series of 281 heart failure patients.⁴ Several studies have described the development of AI as well as the progression from mild AI resulting in increasing recirculation of blood volume (closed-loop mechanism with blood flowing from the LV through the LVAD into the aorta and back into the LV).^{5,6} This altered circulation path may be etiological in the recurrence of heart failure symptoms despite LVAD implantation. This article describes the treatment and interventions selected for heart failure patients presenting with concomitant aortic valve insufficiency and with de novo aortic valve insufficiency following LVAD implantations.

Methods

Medical records of all patients who underwent LVAD implantation between October 2012 and December 2014 were extracted from the Heart Failure Data Registry of Florida Hospital Orlando, USA, and were reviewed retrospectively. The analysis included data of all patients with moderate to severe aortic valve insufficiency at implantation time as well as data of all patients with normal aortic valve function who developed aortic valve insufficiency after LVAD implantation (de novo). Moderate or more severe AI at LVAD implantation was corrected as recommended in the literature.⁵ Post-operative management included pump speed optimization to allow for adequate aortic valve opening. Progressive and de novo aortic regurgitation post LVAD implantation was addressed with medical management as first-line therapy.



Patients underwent standard pre- and intraoperative evaluation with transthoracic and transesophageal echocardiography (TTE, TEE), left and right heart catheterization, chest computer tomography, and pulmonary artery catheter evaluation. Patients with postoperative new onset AI and complications were assessed by TTE, right heart catheterization and LVAD interrogation for medical management optimization with VAD flow adjustment, diuresis, and hypertension control.

Due to the retrospective nature of the study ethical approval was waived by Florida Hospital ethics committee prior to the analysis. All patients signed an informed consent form and agreed that their data will be archived anonymously at hospital and national data registries (e.g. INTERMACS)

Results

Data of 39 patients (21-76 years of age, 13% female, 23% bridge-to-transplant, 77% destination therapy) were included in this retrospective analysis. Overall, patient 1-year survival was 89% with bridge-to-transplant VADs and 75% with destination VADs.

Intraoperative TEE assessments revealed moderate to 2+ AI in three patients with a history of dilated cardiomyopathy (patients 1-3, Table 1). Two patients were considered for destination therapy and one patient was listed and bridged to heart transplantation. The destination therapy patients (patients 1 and 2) underwent bioprosthetic aortic valve replacement (21 and 23 mm standard Carpentier-Edwards bioprosthetic valves [Edwards Lifesciences Corporation, Irvine, CA, USA]) combined with HeartMate II implantation (HM II; Thoratec Corporation, Pleasanton, CA, USA). They were connected to cardiopulmonary bypass through standard distal ascending aorta and right atrial cannulation and cooled down to 34° C. After LVAD implantation, a short period of antegrade hypothermic crystalloid arrest after aorta cross-clamping allowed standard aortic valve replacement through a hockey stick-like aortotomy 1.5 cm distal to the origin of the right coronary artery. After release of the cross-clamp and reperfusion of the heart, the outflow graft was trimmed to its appropriate length and sutured end-to-side to the ascending aorta using a partial occlusion clamp. The third patient presented with moderate AI and severe ascending aortic dilatation to 52 mm with a very thin aortic wall (patient 3). He underwent a relatively complex procedure consisting of aortic bioprosthetic valve replacement (Carpentier-Edwards bioprosthetic valve) with ascending and hemi aortic arch replacement using circulatory arrest together with LVAD implantation. The surgical strategy was similar as described for the two destination therapy patients followed by hemi arch replacement using deep hypothermic circulatory arrest and 30 mm tube graft prosthesis with a single sidearm. The LVAD outflow graft was sutured end-to-end to the sidearm of the vascular prosthesis. All three patients made an uneventful recovery and were monitored monthly with TTE in the program affiliated clinics. There were no complications related to the prosthetic aortic valve such as thrombosis, stroke, or valve degeneration. All three patients survived the first year following LVAD implantation.



Table 1. Patients with end-stage cardiomyopathy treated using aortic valve replacement and left ventricular assist devices

Patient	Gender	Age (years)	Cardio-myopathy	Procedure/d evice	Type of therapy	Diagnosis of aortic insufficiency	Medical managemen t of aortic insufficienc y
1	Male	70	Dilative	AVR/HM II	DT	Time of implant	No
2	Male	68	Dilative	AVR/HM II	DT	Time of implant	No
3	Male	56	Dilative	Complex AVR/HVAD	BTT	Time of implant	No
4	Female	64	Dilative	HM II	DT	8 months postoperative	Yes
5	Male	70	Ischemic	HM II	DT	8 months postoperative	Yes
6	Male	58	Dilative	HVAD	BTT	8 months postoperative	Yes + Heart transplant
7	Male	74	Ischemic	HM II	DT	8 months postoperative	Yes + Amplatzer device

AVR, aortic valve replacement; BTT, bridge-to-transplant; DT, destination therapy; HM, Heartmate; HVAD, HeartWare

Eight months following LVAD implantation, four patients (patients 4-7, Table 1) presented with heart failure and AI onset (3 patients on HM II, one patient on HeartWare HVAD [HeartWare, Framingham, MA, USA]). Heart failure was recurrent and was attributable to the newly developed AI. The initial management was conservative with medical treatment including diuretics and vasodilators but also TTE-controlled LVAD interrogation, LVAD flow reduction through a decrease in pump speed until frequent aortic valve opening was documented. These conservative measures were successful in two of the four patients (patients 4 and 5). The condition of the third patient (patient 6) deteriorated 14 months after an initial positive response to medical treatment and LVAD flow adjustments; he underwent orthotopic heart transplantation which was successful. In the fourth patient (patient 7), left-sided symptoms persisted and worsening shortness of breath was observed. Eleven months after HM II implantation as third-time



sternotomy for ischemic cardiomyopathy he presented with severe right and left heart failure, multiorgan dysfunction, and hepatorenal failure. De novo AI and clinical decompensation were diagnosed. A decrease of HM II flow through reducing RPM from 8800 RPM to 8200 RPM markedly attenuated the AI but medical optimization management was not successful. The patient required inotropic medications and mechanical ventilatory support for worsening right heart failure and pulmonary edema. Immediate pre-procedure hemodynamics were right arterial pressure 17 mmHg, pulmonary artery pressure 54/32 mmHg, pulmonary artery occlusion pressure 22 mmHg, left ventricular pressure 90/50 mmHg, left ventricular end-diastolic pressure 20 mmHg, and cardiac output 2.4 L/min. Ultimately, the patient underwent percutaneous transcatheter closure of the aortic valve with a multi fenestrated 30 mm Amplatzer Cribriform (AGA Medical Corp, Plymouth, MN, USA) closure device (reported as a case report⁷). Immediate post-procedure hemodynamics were right arterial pressure 3 mmHg, pulmonary artery pressure 32/14 mmHg, pulmonary artery occlusion pressure 16 mmHg. Fluoroscopy and TTE confirmed the successful aortic valve closure. Under echocardiography, the LVAD flow was increased to 9400 RPM. Two weeks after the procedure, the patient remained stable with improved symptoms and functional status and without evidence of further AI or device migration. The patient was discharged after 34 days of rehabilitation and nutritional support and was monitored closely by the heart failure team. He did not achieve the same exercise tolerance level as observed before aortic valve closure. The patient expired 14 months after LVAD implantation in a hospice following a fall and head trauma associated with intracranial bleeding.

Discussion

Aortic stenosis or insufficiency is uncommon in patients with advanced heart failure considered for LVAD implantation.⁴ The main concern lies in the initiation of LVAD support which can exacerbate pre-existing AI.^{8,9} It was suggested that LVAD implantations create a continuous transvalvular pressure gradient (aortic pressure over LV pressure) across the aortic valve leading to rare aortic valve openings throughout the cardiac cycle. This may lead to fibrous tissue deposits along the commissures of the leaflets, resulting in leaflet adherence, fusion, and retraction of the leaflet tips and generation of a central orifice.¹⁰ A closed aortic valve and limited antegrade blood flow can lead to stasis on the ventricular surface of the aortic valve generating thrombus formation and organization which further aggravates leaflet fusion.^{8,10} The transvalvular pressure gradient described above can result in regurgitant flow from aorta to LV through the central orifice. Pathophysiologically, this recirculating blood volume may result in the recurrence of heart failure symptoms. Given the potential for AI to progress after LVAD insertion, all moderate or greater degrees of AI at the time of implantation were corrected. Three patients successfully underwent concomitant bioprosthetic aortic valve replacement at the time of LVAD implantation. We are not in favor of LV outflow tract closure techniques such as coaptation stitches of the leaflet tips, buttressing stitches of the commissures, or a sutured circular patch or membrane plug technique. Although these simple repair techniques require a shorter period of cardioplegic arrest, they are limited, because they complicate cardiovascular support measures in case of device malfunction and pump failure. Furthermore, a subset of advanced heart failure patients unloaded by LVAD may experience a



return of cardiac function and become candidates for device weaning and explantation.¹¹ We favor bioprosthetic aortic valve replacement as the treatment of choice for moderate or more severe AI given its low complication risks and reliable results and agree with the view that aortic valve provides a “bail-out” mechanism should acute LVAD malfunction occur.⁵ A subgroup analysis revealed a markedly lower long-term survival of patients undergoing LVAD and isolated aortic valve procedures, however, these patients were significantly sicker, older, and with increased right ventricular dysfunction.¹² After multivariate adjustments, surgical aortic valve closure was associated with increased mortality when compared with aortic valve replacement in patient with AI who underwent LVAD insertion.¹³

The development of aortic insufficiency was noted in 25% to 52% of patients by 1 year of LVAD support.⁵ Thirty-six of 166 patients supported by LVAD for a median 461 days developed moderate AI at a median 273 days after LVAD implantation.⁶ AI development was more pronounced in continuous-flow compared with pulsatile-flow LVAD.¹⁴ Newly developed AI after LVAD implantation affects device performance, leads to recurrent heart failure symptomatology, and impacts patient outcomes. In our series of heart failure patients supported by LVAD, de novo AI occurred in four patients in close follow-up ambulatory studies over an average of 8 months. Serial echocardiography revealed intermittent aortic valve opening and pulsatile flow. First-line treatment included diuretics and oral vasodilators to control hypertension and to reduce gradients driving aortic valve insufficiency. Additionally, the selected pump speed setting was adjusted by echocardiography guidance to ensure sufficient circulatory support at rest while allowing intermittent aortic valve opening and maintaining normal LV dimensions. Three of the four patients showed a mild degree of LV myocardial recovery on echocardiography and associated increased ejection and pulsatility allowing a reduction in pump speed.

Clinically significant de novo AI refractory to medical management and LVAD flow optimization is best treated with surgical AI correction or heart transplantation. In cases where conventional aortic valve replacement was of very high risk, percutaneous transcatheter aortic valve closure has been previously described in 5 LVAD patients with de novo AI.¹⁵ The authors concluded that percutaneous transcatheter closure (if successfully employed) appears to be a safe and effective method to treat AI in patients with LVAD, a population often too sick to undergo reoperation for treatment. Although the results are encouraging, the technique needs to be studied in a larger cohort for long-term follow-up. The transcatheter aortic valve replacement might be another good option for these patients with de novo AI; it is, however, not yet approved by the Food and Drug Administration for AI.

In summary, concomitant aortic valve surgery with LVAD implantation is safe and associated with a good outcome. De novo aortic insufficiency and regurgitation may lead to recurrent heart failure and remains a clinical treatment challenge following successful LVAD implantation and support. Most of the patient with de novo AI will respond to optimized medical therapy; the most appropriate and effective treatment option awaits definition.



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