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Case Report

Left Ventricular Recovery with Explantation of Continuous-Flow Left Ventricular Assist Device after 5 Years of Support

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Mechanical circulatory support may result in sufficient myocardial recovery to allow for explantation of the left ventricular assist device (LVAD). The duration of support associated with left ventricular recovery has generally been 6–12 months. In this report, we present a patient in whom the left ventricle recovered after 5 years of support with a LVAD. Our report demonstrates that long-term monitoring for left ventricular recovery is prudent and may allow for late device explantation.

Keywords: circulatory support devices, heart failure, myocardial remodeling

Introduction

Left ventricular assist devices (LVADs) are effective as a bridge-to-transplant strategy or as a destination device for patients with decompensated congestive heart failure. However, the risks associated with LVAD therapy are serious and include infection, stroke, and device malfunction. The other treatment strategy for these patients is cardiac transplantation, which carries its own set of drawbacks including the requirement for life-long immunosuppression and is limited by the lifespan that can be expected of a donor graft.

Prolonged periods of mechanical circulatory support may result in sufficient myocardial recovery to allow explantation of the LVAD.[1)] We have previously reported on patients who were successfully bridged to a return to medical therapy through a process of device weaning that allowed for pump explantation after ventricular function improved.[2)] However, pump explantation after prolonged support by an LVAD is very rare.

Here, we report a case of a patient who was sustained on LVAD therapy for more than 5 years before being deemed eligible for LVAD explantation. Our report demonstrates that long-term monitoring of patients may be useful in identifying patients who have undergone reverse remodeling and regained adequate left ventricular function.

Case Report

A 25-year-old man presented with acute heart failure and fever and leukocytosis suggestive of viral myocarditis. He had a left ventricular ejection fraction <20% and severe pulmonary hypertension, with a pulmonary artery systolic pressure >50 mmHg. He underwent placement of a TandemHeart percutaneous ventricular assist device and intubation for acute respiratory deterioration and cardiogenic shock. His preoperative hospital course was further complicated by acute kidney injury, infection, and pancreatitis.

The patient was treated with intravenous immunoglobulin and methylprednisolone. He was extubated, but

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Table 1 Echocardiography readings

Time	LVAD speed (rpm)	LVIDd (cm)	LVEF (%)	MR	AR	TR
Pre-implant	-	5.3	<20	1	0	1
Post-TandemHeart	-	-	<20	1	0	0
Immediately post-implant	9000	4.0	25–29	0	Not open	0.5
1 month	8600	4.8	-	1	Opens	1
3 months	8000	6.2	-	1	Opens	0.5
6 months	8000	5.8	-	1.5	Not open	1.5
9 months	9000	4.9	-	0	Not open	0
12 months	9000	5.5	-	1	Not open	1
24 months	8400	5.9	25–29	1	Opens	1
36 months	6000	5.9	25–29	0.5	Opens	1
48 months	6000	5.7	30–34	0	Opens	0.5
60 months	8200	5.6	-	0.5	Opens	1
Post-explant	-	6.7	30–34	1	0	0.5

LVAD: left ventricular assist device; LVIDd: left ventricular internal diastolic diameter; LVEF: left ventricular ejection fraction; MR: mitral valve regurgitation; AR: aortic valve regurgitation; TR: tricuspid valve regurgitation

his cardiac function did not recover after 12 days of TandemHeart support. A HeartMate II LVAD was implanted in a standard fashion, via median sternotomy with device placement in a pre-peritoneal position. He had no postoperative complications and was discharged home on postoperative day 19 on aspirin (81 mg daily), carvedilol (6.25 mg twice daily), lisinopril (5 mg daily), and warfarin (7.5 mg daily); he was temporarily kept on sildenafil (20 mg daily) and dipyridamole (75 mg 3 times daily) until 1-month follow-up. He was not listed for heart transplantation at this time due to lack of funding and family support but was maintained on destination therapy with the possibility of converting to bridge-to-recovery therapy after careful monitoring.

Histopathologic examination of heart tissue specimens taken at the time of LVAD implantation was consistent with multifocal interstitial fibrosis with chronic inflammation with some hemosiderin deposition, suggestive of old multifocal ischemia. The overall findings supported the diagnosis of viral myocarditis.

The patient was seen regularly in the LVAD clinic for follow-up examinations to reassess optimal LVAD speed and perform stress tests. The echocardiography data from these examinations showed a steadily increasing left ventricular ejection fraction and no progression of mitral and tricuspid regurgitation (**Table 1**). After 24 months, the patient's aortic valve was consistently opening on every heartbeat. The patient had chronic driveline infections that required frequent antibiotic therapy. After 5 years and 4 months, his echocardiographic examination demonstrated significant improvement in left ventricular systolic function, and a weaning protocol was initiated. The pump speed of 8200 rpms was reduced daily by 400 rpms while his heart function was closely monitored on echocardiogram. Once the pump speed reached 7000 rpms, a bicycle exercise stress test showed normal left ventricular systolic function with exercise. The LVAD was subsequently turned off, and the driveline was cut at the skin level. During this time, the patient was maintained on warfarin (10 mg daily) with a target international normalized ratio of 2–3. All LVAD hardware was successfully explanted 17 months later due to abdominal pain from driveline irritation of the surrounding tissue. He remains in good condition with stable cardiac function 5 years after discontinuing LVAD support.

Discussion and Conclusion

With effective LVAD support, a decompensated heart has the potential to be reconditioned, which allows for the possibility of LVAD removal and a return to medical management.[3] The peak time for reverse remodeling to occur is reportedly within 40–120 days of LVAD implantation.[4] Furthermore, studies suggest that prolonged LVAD therapy may actually reduce myocardial performance and make explant less likely once the window of time for peak improvement has passed.[5] Our case, however, demonstrates that remodeling can occur much later, and patients may still be successfully weaned, even after 5 years of LVAD support. Thus, long-term monitoring of patients should be encouraged.

Long-term follow-up studies of patients who underwent LVAD explant show good survival and low complication

rates; however, the rate of recovery in LVAD patients may be as low as 2%, and patients still face the risk of recurring heart failure.[6] The durability of recovery is significantly higher when combined with pharmacologic therapy, especially when considering the rates only in those with dilated cardiomyopathy, thus demonstrating the potential of reverse remodeling.[2–4] In our experience, we have found that patients with the highest potential of myocardial recovery are those with acute-onset, nonischemic cardiomyopathy, such as viral myocarditis (as seen in this case), postpartum cardiomyopathy, or tachycardia-induced cardiomyopathy.[1] This scenario is particularly true in younger patients who are able to maintain a high degree of physical activity while on continuous-flow LVAD support.

While the frequency of reverse remodeling has been studied, researchers are now trying to better understand the process of reverse remodeling. Samples of myocardial tissue obtained during implantation and explantation allow for comparison of the myocardium before and after mechanical unloading, and this comparison can provide insight into the mechanisms involved in reverse remodeling. Studies have shown that reverse remodeling due to LVAD unloading resulted in decreases in cardiomyocyte size, increases in calcium channels and β-adrenergic receptors, reduced TNF-α expression, and many other transcriptional changes that are currently being examined.[5]

In addition to the molecular changes seen in reverse remodeling, researchers are studying the fluid dynamics that incite these changes. Pulsatile LVADs have previously been reported to offer an increased capacity for reverse remodeling.[6] In our patient, left ventricular function was recovered enough so that the aortic valve naturally began opening although we maintained the pump at a constant speed leading to increased pulsatility and parallel flow. Similar processes that occur during early ventricular recovery may have manifested in our patient; however, further study is necessary to understand the mechanisms underlying these processes.

The recurrence of heart failure is a risk that comes with device explantation in patients who are deemed bridge to recovery. In that scenario, LVAD reimplantation may be necessary, potentially subjecting the patient to additional sternotomies. Thus, our institution uses a conservative approach in timing device explantation; the patient should demonstrate sufficient support with the device on the lowest pump setting of 6000 rpms, which simulates neutral net flow with a total pump flow of 1 L/min.[7] Furthermore, to avoid an additional procedure to explant the device, we ligate the outflow cannula and cut the driveline, which we have previously shown as safe in these cases.[8]

Although more studies are required to fully identify the mechanism of late reverse remodeling, we believe it is prudent to continue to assess LVAD recipients for potential left ventricular recovery, even when the duration of support extends beyond the traditional 6- to 12-month window that is generally associated with recovery. Moreover, developing an institutional protocol for late myocardial recovery may increase the likelihood of identifying patients whose left ventricular function has improved sufficiently to allow for LVAD explantation.

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