Srce i krvni sudovi 2025; 44(2) : 31-33

Case report

Explantation of a Left Ventricular Assist Device Following Reverse Remodeling in Non-Ischemic Cardiomyopathy

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Abstract Left ventricular assist devices (LVAD) improve clinical outcomes in patients with advanced heart failure (HF), including those with acute refractory HF. In carefully selected patients, depending on HF etiology, duration of HF, and degree of end-organ dysfunction, LVAD support may induce profound reverse structural remodeling and even functional myocardial recovery, ultimately permitting LVAD explantation. This "bridge to recovery" strategy, although conceptually appealing and clinically transformative, remains underutilized.

Here, we present a case demonstrating that this strategy offers a valuable alternative to transplantation and highlights the importance of standardized protocols in identifying and managing LVAD recovery candidates.

Key-words Heart failure, LVAD, Recovery

Introduction

Left ventricular assist devices (LVAD) improve clinical outcomes in patients with advanced heart failure (HF), including those with acute refractory HF. In carefully selected patients, depending on HF aetiology, duration of HF, and degree of end-organ dysfunction, LVAD support may induce profound reverse structural remodelling and even functional myocardial recovery, ultimately permitting LVAD explantation. This "bridge to recovery" strategy, although conceptually appealing and clinically transformative, remains underutilized. Explantation rates in contemporary cohorts from the U.S. and Europe are consistently low, typically below 5%[1–3]. This is primarily due to challenges in identifying patients with true recovery potential, lack of standardized recovery monitoring protocols, and the irreversible nature of myocardial damage in many patients by the time LVAD support is initiated.

Case report

We present a case of a 52-year old male patient diagnosed with an advanced heart failure due to myocarditis. Despite receiving the guideline-directed medical therapy (GDMT), he exhibited left ventricle (LV) dilatation with severely reduced ejection fraction of 23%, moderate diastolic dysfunction and severe functional mitral regurgitation. Levosimendan infusion failed to improve LV function and the patient remained inotrope-dependent. The ischemic etiology of HF was excluded and right heart catheterization revealed mild postcapillary pulmonary hypertension. Due to progressive worsening the patient was worked-up per our institutional heart transplant protocol. As no contraindications for heart transplantation were found, the patient was initially put on international high urgency list. Due to progressive worsening despite inotropic support, we opted for long-term mechanical circulatory support and LVAD HearMate II (HM II) was implanted as a bridge to transplant. Postoperative course was unremarkable and the patient was put on optimal heart failure medical therapy as per contemporary ESC guidelines, including angiotensin convertase inhibitor (ACEI), beta blocker and mineralocorticoid receptor antagonist (MRA). During the three years after implantation, echocardiography demonstrated progressive cardiac reverse remodelling, with normalization of LV and right ventricle dimensions, improved ejection fraction on ramp test (defined as LVEF > 40%), and reduction of mitral regurgitation. However, ventricular arrythmias emerged due to small LV cavity, which were likely caused by mechanical pressure of the inflow cannula on the LV septum. Furthermore, the patient developed a persistent driveline infection with Staphylococcus aureus, which was initially resolved with antimicrobial therapy, but reoccurred within five months. At that time driveline infection was managed with VAC, surgical driveline transposition and chronic antimicrobial therapy, but eventually progressed to chronic driveline infection.

Given sustained cardiac recovery, recurrent ventricular arrythmias and the unresolved driveline infection, we attempted weaning the patient off LVAD support. A turn-down hemodynamic assay was done which showed persistently improved LV and RV function also in the conditions of "zero flow" across the LVAD. We thus opted for discontinuation of LVAD support: the pump was left in situ, the driveline was cut above the infectious region

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and buried in the subcutaneous tissue and the outflow graft was surgically closed thorough right mini thoracotomy. The pump remained *in situ*, requiring continued anticoagulant therapy.
At 5-year follow-up, the patient remains clinically stable on GDMT, with a preserved LV systolic function, mild diastolic dysfunction and non-hemodynamical significant valvular disease.

Discussion

Optimal LVAD management, in conjunction with guideline-directed medical therapy (GDMT) for heart failure (HF), facilitates left ventricular (LV) unloading and promotes reverse cardiac remodelling[4]. Although myocardial recovery under LVAD support is generally considered infrequent, it is strongly influenced by HF etiology. The highest recovery rates are observed in younger patients with acute-onset non-ischemic HF, such as myocarditis (7.7%), as was the case in our patient, peripartum cardiomyopathy (4.4%), and chemotherapy-induced cardiomyopathy (4.1%)[5,6].
The RESTAGE-HF trial [7] demonstrated significant improvement in LV function within six weeks of pump speed optimization, with continued functional gains over an 18-month follow-up period. Notably, reverse remodelling can extend beyond two years of device support and occurs along a spectrum—ranging from partial to complete responders [8–10]. The international VAD Wean Registry defines three responder categories: RESTAGE-HF responders (ReHF), meeting at least three criteria (LVEDD < 60 mm, LVESD < 50 mm, LVEF > 45%, PCWP ≤ 15 mmHg, and CI > 2.4 L/min/m²); Utah-Inova responders (UI-R), with LVEF ≤ 40% and LVEDD ≤ 60 mm but not fulfilling ReHF criteria; and partial responders (PR), demonstrating LVEF improvement without meeting ReHF or UI-R definitions[11]. In a cohort of 402 patients from the VAD Wean Registry, freedom from the composite endpoint (death, LVAD reimplantation, or heart transplant) was 84% at 1 year, 67% at 5 years, and 50% at 10 years, with comparable outcomes across responder groups[11].
Reported rates of LVAD explantation vary widely. While some institutional studies have reported explantation rates as high as 73%[7,12–17], large registry-based analyses from EUROMACS, INTERMACS, and UNOS consistently report rates below 4%[5,18,19]. The discrepancy may reflect differences in patient selection, experience with myocardial recovery programs, and—critically—a lack of standardized protocols for identifying and managing recovery candidates[4,19]. Furthermore, many patients are referred for LVAD support only at advanced stages of disease, when irreversible myocardial damage has occurred, and recovery potential is minimal.
Reverse remodelling is a multifactorial process dependent on both mechanical unloading and neurohumoral modulation, the latter driven by the use of HF GDMT[4]. According to INTERMACS registry data[20], patients treated with at least one class of HF GDMT while on LVAD support had significantly better quality of life and higher 4-year survival rates (56% vs. 44%) compared to those not receiving any HF GDMT. Patients on triple GDMT (recommended at that time) achieved the highest 4-year survival at 66%. The most recent HFSA Expert Consensus Statement[21] recommends a stepwise approach: first achieving adequate decongestion and LV unloading via device speed optimization and diuretic therapy, followed by optimization of HFrEF GDMT. Initiation and uptitration of ARNI or ACEI/ARB and beta-blockers should be followed by MRA therapy, especially in hypervolemic or hypokalemic patients. As per current HF GDMT, SGLT2i are also recommended. The use of ivabradine or guanylate cyclase stimulators may be considered, although robust data in LVAD patients are lacking[22].
Serial echocardiographic monitoring is critical for identifying reverse remodelling and emerging myocardial recovery[21]. Delayed recognition of recovery can increase the risk of pump thrombosis, as improved LV contractility and reduced LV chamber size may reduce flow through the device, and predispose patients to ventricular arrhythmias, promoting blood stasis in the pump[4,23].
Currently, no universal protocols exist for LVAD explantation. Historically, LVAD explantation involved full median sternotomy with removal of the pump and outflow graft, which carries the risk of myocardial injury and HF recurrence[24]. In such patients, at least three months of anticoagulation with a vitamin K antagonist—with or without adjunctive antiplatelet therapy—is recommended post-explant[25–27]. Recently, less invasive techniques such as driveline excision and vascular plug deployment (as employed in our case) have emerged as viable and safer alternatives[24].
Evidence regarding post-weaning management is limited. In one study, discontinuation of HF pharmacotherapy after apparent recovery led to HF relapse in 44% of patients, compared to none in the group that continued GDMT[28-30]. Thus, even after complete reverse remodelling, long-term maintenance on HF GDMT is advised to sustain myocardial recovery[7]. Importantly, promoting myocardial recovery and successful LVAD weaning an opportunity of avoiding heart transplantation altogether.

Conclusion

Although myocardial recovery on LVAD support remains uncommon, it is achievable in selected patients through optimized unloading, guideline-directed medical therapy, and structured follow-up. Despite low explantation rates in registries, dedicated recovery programs demonstrate that sustained recovery and LVAD weaning are possible. This strategy offers a valuable alternative to transplantation and highlights the importance of standardized protocols in identifying and managing recovery candidates.

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Sažetak

Eksplantacija LVAD-a kao posledica reverznog remodelovanja kod pacijenta sa ne-ishemijskom kardiomiopatijom

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Uređaji za potporu leve komore (LVAD) poboljšavaju kliničke ishode kod pacijenata sa uznapredovalom srčanom insuficijencijom (HF), uključujući i one sa akutnom refraktornom HF. Kod pažljivo odabranih pacijenata, u zavisnosti od etiologije HF, trajanja HF i stepena disfunkcije krajnjeg organa, podrška LVAD-a može izazvati značajno reverzno strukturno remodeliranje, pa čak i funkcionalni oporavak miokarda, što na kraju omogućava eksplantaciju LVAD-a. Ova strategija „mosta do oporavka", iako konceptualno privlačna i klinički transformativna, ostaje nedovoljno iskorišćena.

Sada predstavljamo slučaj koji pokazuje da ova strategija nudi vrednu alternativu transplantaciji i ističe važnost standardizovanih protokola u identifikaciji i lečenju kandidata za oporavak LVAD-a.

Ključne reči: *Srčana insuficijencija, LVAD, oporavak*