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▶ To cite this version:

Federica Valente, Matteo Pozzi, Laurent Sebbag, Guillaume Baudry. Left Ventricular Assist Device as a Bridge-to-Recovery Strategy in Alcoholic Cardiomyopathy: A Case Series. Canadian Journal of Cardiology, 2024, Online ahead of print. 10.1016/j.cjca.2024.08.278. hal-04694540

HAL Id: hal-04694540 https://hal.univ-lorraine.fr/hal-04694540v1

Submitted on 11 Sep 2024 $\,$

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Canadian Journal of Cardiology = (2024) 1-4

Case Report

Left Ventricular Assist Device as a Bridge-to-Recovery Strategy in Alcoholic Cardiomyopathy: A Case Series

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Alcoholic cardiomyopathy (ACM) is a prevalent cause of nonischemic cardiomyopathy, with one-third of patients recovering systolic function through alcohol abstinence and heart failure (HF) treatment. However, in advanced ACM leading to cardiogenic shock (CS), where hemodynamic instability hinders the initiation of HF treatment, long-term mechanical circulatory support emerges as a viable strategy for bridge to recovery. This report details 3 cases of successful use of the Abbott HeartMate 3 as bridge to recovery in ACM-induced CS, highlighting the positive long-term outcomes after left ventricular assist device (LVAD) removal.

Case 1

A 36-year-old man (Supplemental Table S1), with no previous history excepted 15 years of heavy alcohol and tobacco consumption, presented to the emergency department with stage C CS. The clinical presentation was suggestive of ACM. Further diagnostic work-up, including thoracic computed tomography and coronary angiography, corroborated this hypothesis.

Despite inotropic intravenous treatment, the patient's condition deteriorated with necessity of veno-arterial

Received for publication March 3, 2024. Accepted August 21, 2024.

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extracorporeal membrane oxygenation (VA-ECMO) support. After 9 days, an LVAD was implanted as a bridge-to-recovery strategy because ECMO weaning was impossible.

Case 2

A 38-year-old man (Supplemental Table S1) with a history of heavy alcohol consumption was admitted to the intensive care unit (ICU) because of respiratory failure and CS. After 12 hours of lack of response to intravenous medical treatment, the heart team rapidly considered the patient to be in stage D requiring use of VA-ECMO.

Coronary angiography and assessment to rule out ischemic cardiomyopathy were normal, indicating ACM as the likely cause. Following an unsuccessful attempt to wean from VA-ECMO, an LVAD was implanted as bridge to recovery or candidacy. The ICU stay was prolonged to 36 days, marked by severe acute right HF that required temporary mechanical right ventricular support.

Case 3

A 44-year-old man (Supplemental Table S1) with a history of heavy alcohol consumption presented with stage C cardiogenic shock. Further evaluation revealed atrial tachycardia and severe left ventricular dysfunction consistent with nonischemic cardiomyopathy, likely stemming from ACM. Attempts to manage supraventricular arrhythmia with external electrical cardioversion resulted in hemodynamic failure, requiring inotropic support and femoro-femoral VA-ECMO. Despite unsuccessful weaning from VA-ECMO after 10 days, the patient underwent implantation of an LVAD.

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Perioperative Management After LVAD Implantation

We performed short-term right ventricular assist device implantation in the first 2 patients owing to uncertainty regarding associated right ventricular dysfunction. Indeed, we use a liberal implantation strategy when precise and reliable assessment of the right ventricle is not feasible due to VA-ECMO support. Histologic analysis of the apical fragment retrieved during LVAD implantation was performed for all patients, revealing nonspecific findings suggestive of cardiomyopathy, and ruling out myocarditis. These findings included collagenous fibrous septa dividing muscle bundles and myocytes displaying vacuolisation and hyalinisation (Supplemental Figs. S1 and S2). Genetic testing was performed on all patients. A variant of uncertain significance was identified in the *TTN* gene (titin gene) only in patient 1.

Joint Management After LVAD Implantation

All patients underwent cardiac rehabilitation, achieving successful alcohol withdrawal, and received guideline-directed medical therapy.

Two patients were on triple therapy with beta-blockers, angiotensin-converting enzyme inhibitors, and mineralocorticoid receptor antagonists, and the third received quadruple therapy, including an sodium-glucose cotransporter 2 inhibitor. All patients reached New York Heart Association functional class I status, with normalised cardiac parameters, as detailed in Supplemental Table S2.

Recovery assessment and weaning decision making were based on transthoracic echocardiography (TTE) and right heart catheterisation (RHC) data obtained during a ramp test, as outlined in the protocol provided in Supplemental Appendix S1. Successful weaning tests led to LVAD deactivation in all patients after a median of 13 months. The deactivation procedure involved percutaneous occlusion of the outflow graft, followed by subcutaneous transection of the driveline. For patients 1, 2, and 3, respectively, percutaneous occlusion of the outflow graft was performed with a Watchman FLX 27 device (Boston), an Amplatzer Amulet 20 mm plug (Abbott), and a Watchman 21 device (Boston), as shown in Video 1

Maintenance of anticoagulation therapy, HF treatments, and alcohol abstinence ensured patient stability during the 2year postdeactivation period. Computed tomography confirmed complete thrombosis of the outflow graft, as dhown in Figure 2. Absence of complications such as infections, thromboembolic events, and arrhythmias validated the safety and efficacy of the weaning technique approach. No clinical events were recorded during the follow-up periods, which extended up to 24 months for patient 1, 30 months for patient 2, and 36 months for patient 3 (Supplemental Table S3).

Discussion

ACM is a myocardial disease characterised by ventricular dilation and impaired contraction, often linked to prolonged alcohol abuse.¹ This report discusses 3 cases of refractory CS due to ACM, where ECMO weaning failed, and successful treatment was achieved through LVAD intervention. LVAD

provides significant volume and pressure unloading, reversing stress-related compensatory responses and abolishing neuro-hormonal and inflammatory activation, alongside pharmacologic optimisation.^{2,3}

Although myocardial recovery allowing successful LVAD deactivation is rare, recent studies, such as RESTAGE-HF (Remission From Stage D Heart Failure),³ have shown promising results. Our patients, who were young and had nonischemic cardiomyopathy with a shorter duration of HF, underwent LVAD implantation with a bridge-to-recovery strategy.

A recent large INTERMACS series⁴ demonstrates that alcohol consumption at the time of implantation was not associated with increased mortality, but was significantly associated with increased device malfunction, pump thrombosis, device-related infection, and all-cause hospitalisation. This indicates that LVAD implantation is a reasonable survival strategy, and early deactivation could reduce long-term complications. This encourages us in our current strategy of favouring short-term support to promote recovery. Despite the lack of standardised weaning protocols for HeartMate 3, recovery assessment and weaning decision making were based on TTE and RHC data during a ramp test under optimal anticoagulation.

Current experience with LVAD deactivation is limited, with only a few reports documenting the use of a transcatheter approach. Previously published case reports and series detailing percutaneous LVAD decommissioning are summarised in Supplemental Table S4.

Although there are inherent risks associated with this technique, such as thromboembolic events, ventricular



Figure 1. Fluoroscopy shows the implantation of the Watchman device (Boston Scientific Corp, Marlborough, MA) (arrow) in the distal outflow graft (OG; dashed lines) proximal to the aortic anastomosis.

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Figure 2. Computed tomography (A: sagittal view; B: axial view) performed at 1-year follow-up shows complete thrombosis of the outflow graft (clear arrowhead) after occlusion by a Watchman device (solid arrowheads)

arrhythmias, and device-related infections, a meta-analysis conducted by Choi et al.⁵ suggests that decommissioning presents a feasible alternative to LVAD explantation, yielding similar clinical outcomes. Factors influencing this decision included the aim to avoid sternotomy, circumvent cardiopulmonary bypass, prevent alterations to left ventricle geometry, and reduce perioperative infectious risks. Percutaneous occlusion of the outflow graft with the use of a left atrial appendage occlusion device was successfully performed in our patients, offering a safe and effective alternative to surgical pump explantation.

This strategy could be applicable to all instances of *de novo* nonischemic cardiomyopathy that present with CS necessitating mechanical circulatory support, aiming to circumvent heart transplantation and potentially facilitate recovery. Nonetheless, the effectiveness and safety of this approach require confirmation through prospective studies.

After LVAD deactivation for myocardial recovery, lifelong follow-up is essential to mitigate the risk of HF recurrence. In conclusion, LVAD serves as an effective bridge-to-recovery therapy for refractory CS in ACM patients, with percutaneous deactivation offering safe and favourable long-term outcomes.

Acknowledgements

We thank Dr Salim Si-Mohamed for the contribution of the computed tomography images and Dr Lara Chalabreysse for providing the histology images.

Data Availability

All data generated or analysed during this study are included in this published article.

Ethics Statement

Ethical approval was deemed not required as this is a retrospective analysis of anonymized data, in which patient identity is fully protected and no new interventions or changes to treatment protocols were introduced.

Patient Consent

The authors confirm that written consents for submission and publication of this case series including images and associated text have been obtained from the patients in line with COPE guidelines.

Funding Sources

The authors have no funding sources to declare.

Disclosures

The authors have no conflicts of interest to disclose.

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Supplementary Material

To access the supplementary material accompanying this article, visit the online version of the *Canadian Journal of Cardiology* at www.onlinecjc.ca and at https://doi.org/10. 1016/j.cjca.2024.08.278.