Combined mechanical circulatory support for ventricular fibrillation in left ventricular assist device patient

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Abstract

Ventricular fibrillation, a life-threatening ventricular arrhythmia, may result in pulselessness, loss of consciousness and sudden cardiac death. In this case report, we describe our experience in managing a 54-year-old man with HeartMate3 left ventricular assist device (LVAD) as a bridge to transplantation due to dilated non-ischemic cardiomyopathy, presenting with incessant ventricular arrhythmia for 35 days despite multiple attempts to restore normal rhythm with external direct current cardioversion and anti-arrhythmic medications. The patient remained stable in ventricular arrhythmia with no progression to asystole, but hemodynamic collapse due to right heart failure occurred in the third week. Combined use of two mechanical circulatory support devices (LVAD with VA ECMO) was needed to achieve haemodynamic and metabolic stability, eventually leading to successful heart transplantation in the index admission. The patient was discharged home 2 weeks after transplantation in good clinical condition.

Keywords

Combined ECMO and LVAD; Case report; Heart transplantation post ECMO and LVAD; Ventricular arrhythmia in LVAD

Received: 31 October 2021; Revised: 12 March 2022; Accepted: 8 May 2022
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Learning points

Ventricular arrhythmia is not uncommon scenario in LVAD patients; a prompt intervention is mandatory before irreversible end-organ damage and haemodynamic instability develops. Stable patients should be treated with ventricular tachycardia ablation or cardiac sympathetic denervation. In haemodynamically unstable patients with severe right ventricular failure, the case is more complex but combined support with veno-arterial ECMO seems to be a promising option as a bridge to emergent heart transplantation.

Introduction

Sustained ventricular tachycardia (VT) and ventricular fibrillation (VF) are life-threatening arrhythmias and are highly prevalent in patients with advanced heart failure. Ventricular arrhythmias may impair the hemodynamic support provided by a left ventricular assist device (LVAD) and lead to more frequent hospitalizations and need for anti-arrhythmic therapies, external defibrillations and emergent heart transplantation. Persistent ventricular arrhythmia in LVAD patients may lead to right ventricular (RV) failure and hemodynamic compromise. A prompt intervention to support the failing RV is mandatory to prevent irreversible end-organ damage.
We report an unusual case of persistent VF lasting for almost 3 weeks in a patient receiving LVAD support with worsening RV failure. Management consisted of combined support with extracorporeal membrane oxygenation (ECMO) until heart transplantation in the same admission.

Case report

The patient was a 54-year-old man with dilated non-ischaemic cardiomyopathy who had been receiving LVAD support (HeartMate3, Abbott, Chicago, IL, USA) for 3 years as a bridge to transplantation. An implantable cardioverter defibrillator (ICD) was inserted 6 years before admission for primary prevention of sudden cardiac death.

During the second year on LVAD support, RV failure developed with severe tricuspid regurgitation and deteriorating functional capacity. The dose of furosemide was increased to 120 mg per day, resulting in alleviation of the edema but at the price of hospitalizations for recurrent episodes of VT related to electrolyte abnormalities, mainly hypokalemia. (No preventive measures for hypokalemia were taken. Magnesium was within normal limits.) Arrhythmic episodes were successfully treated with ICD cardioversion and potassium supplements.

Following an episode of persistent VT requiring five ICD shocks and prolonged hospitalization, mexiletine was added to the already prescribed amiodarone, with good response. During all of the ventricular arrhythmic events, the patient remained hemodynamically stable. Therefore, considering that the patient presented clinically with only mild weakness, for alleviating his severe anxiety caused by the unpredicted ICD shocks, the defibrillation mode in the ICD was turned off.

Recurrent VT without ICD discharges persisted, leading to brief hospitalizations for external cardioversion under sedation. Ablation was ruled out because of the estimated excessively high procedural risk. Owing to the worsening RV failure and continuing ventricular arrhythmias on LVAD support, the patient was listed for high-urgency heart transplantation.

On 2 April 2021, the patient presented with severe weakness and signs of worsening RV failure for the last 2 weeks. Interrogation of the ICD revealed a prolonged arrhythmic event: 3 days of VF preceded by 2 weeks of VT. At admission, the patient was fully alert but weak. Mean blood pressure (measured by the Doppler technique) was 45 mmHg at LVAD pump speed of 5500 rpm, and the estimated flow was 3.0 L/min. Kidney function was preserved (creatinine 1.1 mg/dL). Hemoglobin measured 12.0 gr/dL, potassium 5.0 mEq/L and magnesium 2.4 mg/dL. Transthoracic echocardiography showed a severely dilated left ventricle (end-diastolic diameter 84 mm) with severely reduced function manifested by an estimated left ventricular ejection fraction of 15%, severe RV dysfunction and moderate to severe tricuspid regurgitation. The aortic valve remained closed throughout the entire study.

Successful direct current cardioversion lasted for only 24 h. VF recurred and persisted despite recurrent external defibrillations and administration of a variety of anti-arrhythmic drugs including amiodarone, mexiletine, quinidine and procainamide.

During the next hours, the patient’s clinical condition deteriorated, with low blood pressure, reduced urine output and evidence of multi-organ failure. The impaired organ perfusion was attributed to the severely reduced cardiac output: worsening renal function, abnormal liver enzymes and increasing lactate levels.

The patient was sedated and placed on a ventilator. Transthoracic echocardiography showed severely dilated and extremely reduced RV function with wide-open tricuspid regurgitation. More invasive hemodynamic evaluation with right heart study was not performed because of the estimated high risk that outweighed the minimal therapeutic relevance.

On 4 April 2021, with the indication of bridge to decision and need for prompt maximal hemodynamic support, the patient was placed on veno-arterial (VA) ECMO. The LVAD rotational speed was reduced to the lowest needed to achieve proper unloading of the left ventricle. Nevertheless, electrical cardioversion failed. A trial of cardioplegia using 3 gr of intravenous potassium chloride resulted in a return to sinus rhythm but for less than a minute, with rapid deterioration back to VF.

Following the ECMO support, the patient’s clinical condition improved. The next day, the patient was extubated, and awakened, but VF persisted. The hemodynamics were typical of ECMO circulation. Normalization of the patient’s metabolic state was indicated by the return to normal range of lactate and creatinine levels and improvement in liver function. The patient was listed at the highest urgency status for heart transplantation.

By the end of the third week, on 20 April 2021, while receiving combined ECMO and LVAD support, the patient underwent successful orthotopic heart transplantation. In the immediate post-operative period, primary graft failure developed, manifested by RV dilation and dysfunction. The patient recovered completely following 2 days of ECMO. He was discharged home 2 weeks thereafter (Figure 1).

Discussion

Ventricular arrhythmias are not uncommon in patients supported with an LVAD. Some investigators suggested that the arrhythmogenic effect of LVADs is explained by the introduction of new areas of scar or alterations in gene expression of ion channels possibly involved in arrhythmogenesis.4,5 Pa-
Patients may tolerate life-threatening ventricular arrhythmias for a certain period of time, as the device supports their native cardiac function. By unloading the left ventricle, pulmonary resistance decreases with improvement in the pulmonary circulation. During that time, the right ventricle may act more as a conduit than a pump.

The natural history of VF is thought to include inevitable progression to asystole, but as this and other reports illustrate, it may be sustained if cardiac and systemic perfusion is secured. The approach to patients with an LVAD in whom ventricular arrhythmia results in worsening RV failure is very challenging.

Triggers for the development of arrhythmia, such as electrolyte abnormalities, acute ischemia, fever or intercurrent illness, suction events or ventricular irritation from contact with the inflow cannula, should be defined and managed straightaway. The first therapeutic intervention should be medical (anti-arrhythmic drugs or cardioversion). If the ventricular arrhythmia persists despite optimal medical therapy, catheter ablation should be considered in the relatively stable patients. When catheter ablation is unfeasible (or considered excessively risky), or RV dysfunction or hemodynamic collapse occurs, hemodynamic support should be promptly applied to prevent irreversible end-organ damage. The mechanical options for immediate hemodynamic support are ECMO or a right ventricular-assist device (RVAD), either durable or temporary.

In our case, several concerns led us to opt for VA-ECMO over a temporary or durable RVAD. They included the difficult adjustment of simultaneous LVAD and RVAD function; the unacceptably high risk of implanting a durable RVAD (such as another HeartMate3 in the RV position) in a hemodynamically unstable patient and the only partial hemodynamic support achievable with percutaneous RVAD (Protek Duo). VA-ECMO can be established promptly, even bedside, and may supply full hemodynamic support. In cases of left ventricular failure, it results in increased left ventricular afterload requiring concomitant unloading of the left ventricle. This can be accomplished by adding an intra-aortic balloon pump or the Impella (ECPella) device or by the LVAD itself. In our case, the LVAD rotational speed was modulated to achieve proper unloading of the left ventricle, thereby avoiding hemodynamic competition between the two devices.

Our patient was maintained on the LVAD and ECMO support for 18 days due to persistent VF. Overall, he was in life-threatening ventricular arrhythmia for 35 days: 14 days stable at home in VT, 3 more days, still at home, in VF and an additional 18 days in hospital with persistent VF, including 16 days on concomitant LVAD and ECMO support.

In 2014, Fux and colleagues described a similar case of a younger patient (45 years old) with dilated cardiomyopathy supported with the second-generation LVAD, HeartMate2, as a bridge to transplantation. The patient was stable for 23 days with persistent VF and RV failure under treatment with VA-ECMO in parallel to the LVAD until heart transplantation.

Future treatment approaches and strategies for ventricular arrhythmias should include cardiac sympathetic denervation. Using this method, Vlismas et al. recently reported promising results in LVAD-supported patients with ventricular arrhythmias who were unresponsive to pharmacological agents, catheter ablation or ICD therapy.

Figure 1 Case report timeline.
In conclusion, ventricular arrhythmia is not uncommon in patients with an LVAD, and it can be well tolerated unless RV function deteriorates. The trigger for the arrhythmia should be identified and treated accordingly, and ablation and cardiac sympathetic denervation should be considered in stable patients. If RV dysfunction and hemodynamic compromise occur, full support with VA-ECMO in parallel with the LVAD is highly recommended as a bridge to heart transplantation, taking the unique hemodynamics of this combination into consideration.

Conflict of interest
None declared.

Funding
None.

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