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GRAND ROUND
Heart Failure

Case report: incessant ventricular fibrillation in a conscious left ventricular assist device patient

Aiste Monika Jakstaite [1], Peter Luedike[1], Reza Wakili[1], Simon Kochhäuser[1], Arjang Ruhparwar [2], Tienush Rassaf [1], and Maria Papathanasiou[1]*

[1]Department of Cardiology and Vascular Medicine, West German Heart and Vascular Center, University Hospital Essen, Hufelandstrasse 55, 45147 Essen, Germany; and
[2]Department of Thoracic and Cardiovascular Surgery, West German Heart and Vascular Center, University Hospital Essen, Hufelandstrasse 55, 45147 Essen, Germany

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Background Ventricular arrhythmia in left ventricular assist device (LVAD) recipients represents a challenging clinical scenario and the optimal treatment strategy in this unique patient population still needs to be defined.

Case summary We report on a 61-year-old LVAD patient with incessant ventricular fibrillation (VF) despite multiple unsuccessful attempts to restore normal rhythm with external defibrillation and antiarrhythmic medication. He remained initially stable as an outpatient and subsequently developed secondary organ failure.

Discussion This case demonstrates that under LVAD support long-term haemodynamic stability is possible even in case of VF, a situation that resembles Fontan circulation. However, ventricular arrhythmias are associated with a high risk of secondary organ damage due to right heart failure if left untreated. In case of refractory ventricular tachycardia or electrical storm listing for heart transplantation with high priority status should be pursued when possible. Alternatively, catheter ablation may be considered in selected cases and be performed in experienced centres in close collaboration with all involved specialists.

Keywords Heart failure • Left ventricular assist device • Ventricular arrhythmias • Ventricular fibrillation • Electrical storm • Case report

Learning points

- Left ventricular assist device (LVAD) can provide long-term haemodynamic stability even in the context of ventricular fibrillation (VF); VF may remain asymptomatic until severe congestion or end-organ dysfunction occur.
- Refractory ventricular arrhythmias may have devastating consequences if untreated.
- Eligibility for heart transplantation in LVAD patients with refractory ventricular arrhythmias should be considered early; catheter ablation is an alternative option.

Introduction

The burden of ventricular arrhythmias in left ventricular assist device (LVAD) patients is known to be high. In this setting, ventricular arrhythmias can be well-tolerated but if persistent, they may lead to haemodynamic compromise, right ventricular (RV) failure, and secondary organ dysfunction.[1] The management of ventricular arrhythmias in LVAD patients remains challenging and requires close

* Corresponding author. Tel: +49(0)201 723 82350, Fax: +49(0)201 723 5488, Email: Maria.Papathanasiou@uk-essen.de
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collaboration of heart failure cardiologists, arrhythmiologists, and heart transplant surgeons. We report here on a patient under long-term LVAD support who presented with long-lasting ventricular fibrillation (VF).

Timeline

Day 1	Admission for progressive dyspnoea NYHA class IV and anasarca, electrocardiogram demonstrates ventricular fibrillation (VF) while patient remains fully conscious, internal cardioverter defibrillator interrogation reveals ongoing VF for >2 months prior to admission. History of type 2 amiodarone-induced thyrotoxicosis diagnosed 4 months prior to current admission with subsequent discontinuation of amiodarone and initiation of thiamazole therapy with now normal thyroid function tests.
Day 21	Trans-oesophageal echocardiogram without evidence of intracardiac thrombi, three unsuccessful external defibrillations, re-initiation of oral amiodarone 800 mg/day as a bail-out option.
Day 28	Three unsuccessful external defibrillations, discontinuation of amiodarone.
Day 42	Acute kidney injury with anuria, admission to the intensive care unit, and initiation of continuous veno-venous haemodialysis.
Day 43	Exacerbation of chronic driveline infection firstly diagnosed 10 months ago (6 years of post-left ventricular assist device implantation), further clinical deterioration, shared decision for best supportive care.
Day 46	Death due to septic shock with multi-organ failure.

Case presentation

A 61-year-old male patient with advanced heart failure under LVAD support presented to the emergency department with worsening dyspnoea, NYHA Class IV, and massive lower extremity and scrotal oedema. A continuous-flow LVAD had been implanted 7 years ago due to dilated cardiomyopathy with heart failure refractory to medical therapy. During LVAD support, the patient developed morbid obesity (body mass index 41 kg/m^2) and chronic driveline infection. At presentation, arterial pulse was absent with normal capillary refill time. The core temperature was normal. The neurologic exam was unremarkable. Electrocardiogram demonstrated VF (*Figure 1*). Interrogation of the implanted cardioverter defibrillator revealed VF while antitachycardic therapy had been previously deactivated due to RV lead malfunction. Left ventricular assist device interrogation showed no acute deviations of pump flow and power readings from the patient's baseline (pump flow 6.0 L/min, power 5.0 W). Laboratory testing showed elevated serum creatinine (2.3 mg/dL, reference, 0.9–1.3 mg/dL) and N-terminal prohormone of brain natriuretic peptide (39 330 pg/mL, reference, <125 pg/mL). Serum lactate level in venous blood was 3.5 mmol/L (reference, 0.5–1.6 mmol/L). Trans-oesophageal echocardiography was then performed that depicted global akinesia of all cardiac chambers with profound dilation of the right ventricle and faint movements of the tricuspid valve leaflets, a situation resembling Fontan circulation (*Figure 2* and *Videos 1 and 2*). We initiated intravenous furosemide to relieve congestion and performed external defibrillation after excluding intracardiac thrombosis with trans-oesophageal echocardiography. After multiple unsuccessful attempts to restore normal rhythm with external defibrillation, oral amiodarone was initiated followed again by multiple unsuccessful defibrillations. Due to morbid obesity despite multiple dietary consultations and the patient's wish not to proceed to bariatric surgery due to the high procedural risk, listing for heart transplantation was not possible. It was a shared decision to refrain from performing catheter ablation in the setting of severe comorbidities including morbid obesity, severe renal failure, chronic driveline infection, and incessant VF. The patient eventually developed end-stage renal failure and died due to complications of sepsis.

Discussion

The burden of ventricular arrhythmias in LVAD recipients is known to be high[1] and VF has been reported to affect >10% of patients after LVAD implantation.[2] In the ASSIST-ICD study, 494 LVAD patients, of whom 75% had an internal cardioverter defibrillator (ICD) in place were followed up in 19 tertiary French centres.[3] Late ventricular arrhythmias (>30 days post-implantation) affected 27% of patients after a median follow-up of 19 months and electrical storm occurred in 9% of patients. Ventricular fibrillation or combined VT/VF was observed in 39% of patients with electrical storm.[4] As the number of implants and the duration of support continue to rise it is anticipated that ventricular arrhythmias including VF will further evolve to a challenging clinical problem.

Ventricular arrhythmias under LVAD support are usually well-tolerated but if persistent may lead to collapse of the left ventricle, haemodynamic instability, or RV failure.[1] In order to avoid painful shocks in conscious state and considering the unclear survival benefit with ICD in this population, shock therapy is often deactivated and elective replacement of the generator or any malfunctioning leads may be deferred according to the individual patient needs.[5,6] This case demonstrates that long-term haemodynamic stability is possible even in the context of VF in LVAD supported circulation and may even remain unrecognized until severe congestion or end-organ dysfunction occur. However, VF is an extreme clinical scenario with atrial and ventricular near-standstill and unpredictable clinical outcome. It may also promote intracardiac thrombosis and embolic events due to blood

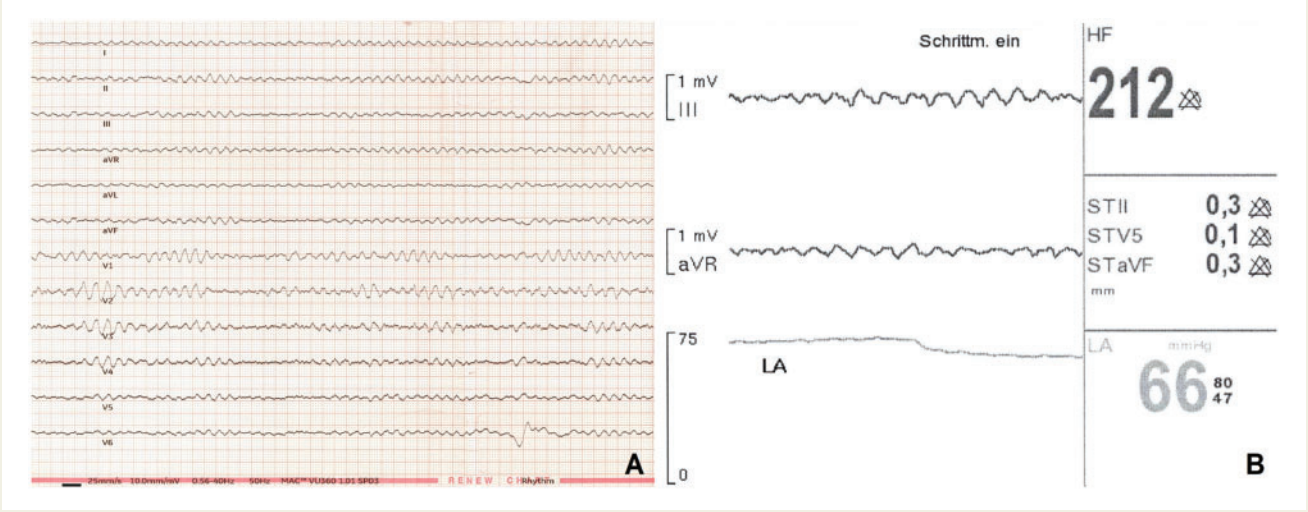


Figure 1 (*A*) Electrocardiogram at admission demonstrating ventricular fibrillation. (*B*) Arterial pressure waveform recording demonstrating the absence of arterial pulse and a mean arterial pressure of 66 mmHg during ongoing ventricular fibrillation. LA, left radial artery.

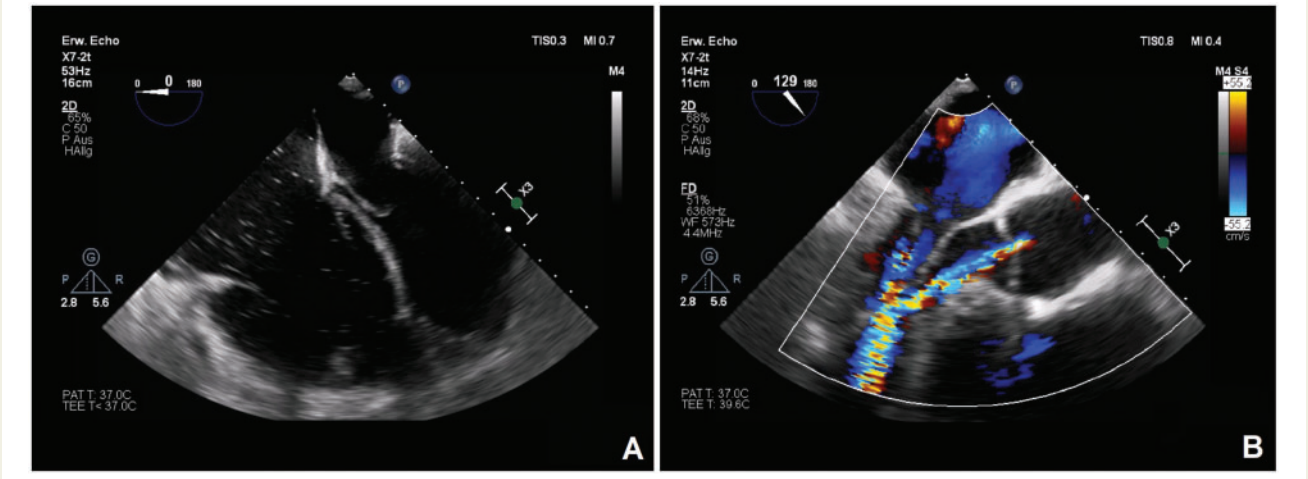
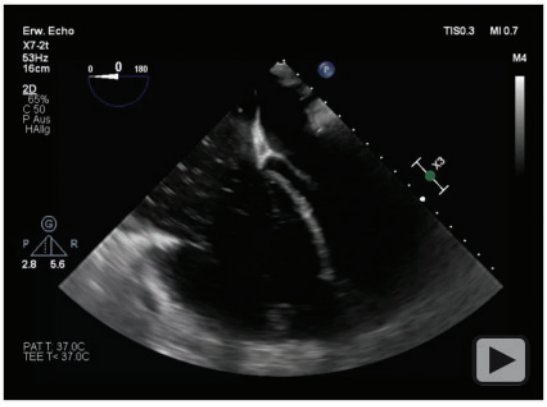


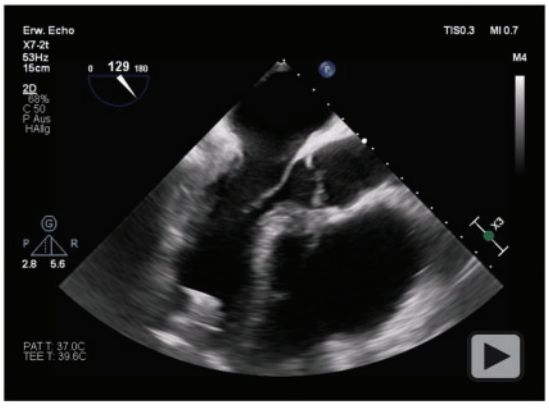
Figure 2 Still images of (*A*) mid-oesophageal four-chamber view and (*B*) mid-oesophageal long-axis view during ventricular fibrillation.

stasis or slow flow. Right ventricular volume and pressure overload may lead to secondary organ failure if left untreated for prolonged period of time. Presumably, normal pulmonary vascular resistance is a prerequisite for maintaining long-term stability in this context and thus, pulmonary hypertension may be a therapeutic target in such patients but should be carefully balanced against potential side effects.[7,8]

In LVAD, patients with recurrent ventricular tachycardias despite antiarrhythmic therapy catheter ablation should be considered as an alternative treatment option. This strategy requires highly experienced operators, familiarity with the LVAD physiology, and close collaboration between heart failure cardiologists and arrhythmiologists.[9] Although catheter ablation is often the only alternative in cases of sustained ventricular tachycardia, there are nearly no data in the context of incessant VF, which is completely refractory to defibrillation. Recent studies on catheter-free, non-invasive cardiac radioablation using stereotactic body radiation therapy for ventricular tachycardias have shown promising results that could be of interest for the LVAD population.[10] Cardiac sympathetic denervation could also be a promising new therapeutic option for the management of recurrent ventricular arrhythmias.[11] Heart transplantation represents the optimal therapy for patients who experience refractory ventricular arrhythmias or VF during LVAD support. In this case, listing with a high



Video 1 Mid-oesophageal four-chamber view during ventricular fibrillation. Profound dilation of right heart cavities with akinetic tricuspid valve leaflets whereas mitral valve opening and closure is preserved under left ventricular assist device support. LVAD, left ventricular assist device.



Video 2 Mid-oesophageal long-axis view during ventricular fibrillation. Visualization of the left ventricular assist device inflow cannula in the cardiac apex. Dilated right ventricle and decreased internal diameter of the left ventricle under concurrent left ventricular assist device unloading. LVAD, left ventricular assist device.

priority status has to be considered timely if no other contraindications exist to avoid secondary organ damage.

Lead author biography

Ms. Aiste Monika Jakstaite is a resident in internal medicine and cardiology at the West German Heart and Vascular Center in Essen, Germany. After graduating from medical school in Vilnius, Lithuania, she moved to Germany and began her doctoral thesis on the role of phosphodiesterase 5 inhibitor in mechanical circulatory support. Her research interest focuses on advanced heart failure and cardiac amyloidosis. Since 2020, she serves as junior editor in *EHJ Case Reports*.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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