

Permanent cardiac arrest in a patient with a left ventricular assist device support

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The left ventricular assist device (LVAD) support significantly reduces mortality in end-stage heart failure patients and potentially restores eligibility for heart transplantation in patients with initially significant pulmonary hypertension [1, 2]. Ventricular fibrillation and asystole lead to a decrease in right ventricular output and left ventricular preload, which significantly diminishes efficiency of LVAD support [3–5]. In this clinical vignette, we present a patient with an implanted continuous-flow LVAD (CF-LVAD) who developed permanent asystole as a consequence of an electric storm.

A 38-year-old male patient with an end-stage heart failure due to non-ischemic, post-inflammatory cardiomyopathy, qualified for heart transplantation in February 2018 and then implanted with a CF-LVAD HeartMate 3 (HM3, Abbott Inc, Chicago, IL, US) in May 2018 as a bridge-to-transplant due to recurrent episodes of circulatory decompensations, was referred to our institution in February 2019 with P-wave asystole (Figure 1B). In February 2014, the patient was implanted with a cardiac resynchronization therapy defibrillator (CRT-D) for primary prevention of sudden cardiac death. On admission, the patient was conscious and presented low blood pressure (mean arterial pressure [MAP], 53 mm Hg) and diminished pump flow of 2.4 l/min, which was accompanied by low-flow LVAD alarms. Other pump parameters, such as power consumption, speed, and pulsatility index (PI) were within the normal range (3.7 Watt, 5500 rpm, 3.0 PI, respectively). An echocardiography revealed an image of aki-

netic myocardium of both ventricles filled with enhanced echogenic blood (four-chamber view measurements: right ventricle 50 mm, left ventricle 60 mm, intraventricular septum in the middle position) with permanently closed aortic valve and constantly opened tricuspid and mitral valve. On CRT-D interrogation we found numerous, adequate high voltage interventions for a ventricular storm (Supplementary material, Figure S1) a day before admission and subsequent permanent asystole. Laboratory tests showed features of severe multiorgan failure (Supplementary material, Table S1). Intravenous fluid therapy under strict control of the fluid balance allowed for a rapid improvement of pump flow and achievement of satisfactory MAP (70–80 mm Hg) while systemic heparinization with unfractionated heparin prevented pump thrombosis. The patient remained conscious and hemodynamically stable with gradual end-organ function improvement. He was consulted by a psychiatrist due to the results of laboratory tests that revealed the presence of metabolites of psychoactive substances. The consultant emphasized the elevated risk of noncompliance but did not state absolute contraindications to orthotopic heart transplantation (OHT) and after several psychotherapy sessions, the patient declared full commitment to abstinence and further good compliance. Due to the estimated high risk of hemodynamic deterioration, the patient was listed for an urgent OHT, which was performed 11 days later. Pathomorphological examination of the patient's native heart

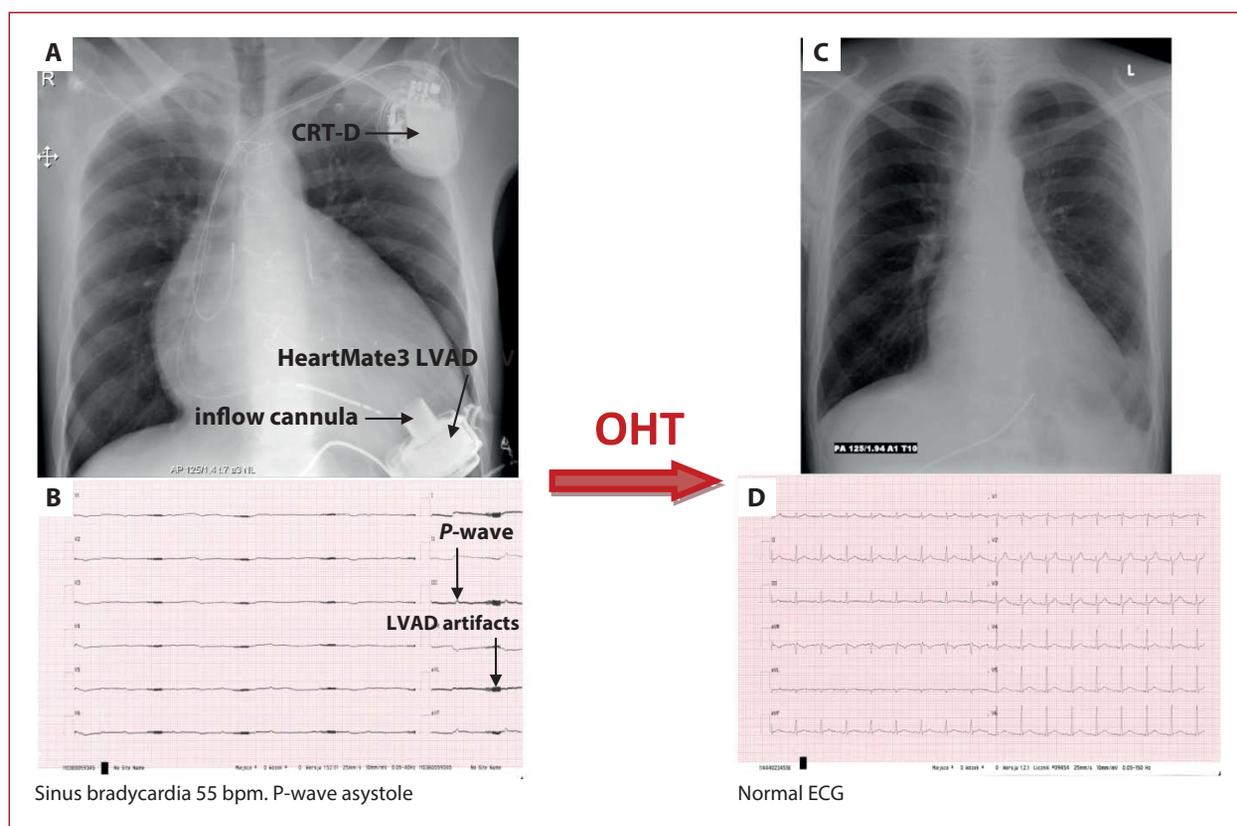


Figure 1. Chest X-ray and ECG before and after OHT

Abbreviations: CRT-D, cardiac resynchronization therapy defibrillator; ECG, electrocardiogram; OHT, orthotopic heart transplantation; LVAD, left ventricular assist device

revealed numerous foci of necrosis. The post-transplantation short- and long-term course was uneventful. The patient was treated according to the routine immunosuppressive protocol and presented stable levels of immunosuppressive drugs. All the follow-up echocardiographic examinations showed normal graft function, and there were no significant cellular and humoral rejections in the consecutive protocol biopsies. A chest X-ray and electrocardiogram before and after OHT are presented in **Figure 1**. He stayed in good condition and did not present any signs of drug and alcohol abuse at check-up visits. Unfortunately, he died due to a post-traumatic intracerebral hemorrhage 27 months after OHT.

Article information

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