Case Report

Left Ventricular Assist Device Vegetation: "Cure" Without Device Explantation

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Infection following the implantation of a left ventricular assist device (LVAD) is a life-threatening complication with mortality rates ranging from 15% to 44%. *Staphylococcus aureus* and *Staphylococcus epidermidis* are the most frequently identified pathogens and are responsible for 60% of LVAD-related infections, local as well as systemic. In this report we describe the successful therapeutic management of a patient who received a Heart Mate II as "bridging-to-recovery", which was complicated by device infection that was managed without device explantation.

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Device infection remains the Achilles' heel of left ventricular assist device (LVAD) implantation, occurring both early postoperatively, and in the long term after device implantation, with sepsis being the leading cause of mortality and morbidity in this patient population.[1] Infection of the LVAD manifests as local infections, affecting either the LVAD cutaneous exit site (Figure 1A) or the pump pocket site, or as bloodstream infections due to infection of the internal lining of the device, resulting in LVAD endocarditis (Figure 1B).[2,3]

Case presentation

A 42-year-old male patient (weight 110 kg, height 185 cm) implanted with a Heart Mate II (Thoratec, Pleasanton, CA, USA) as "bridging-to-recovery", presented in the outpatient clinic with intermittent fever and weakness. His medical history consisted of non-compaction cardiomyopathy diagnosed ten years previously, and he was in New York Heart Association (NYHA) class III and heart failure stage III before intervention. After device implantation, the patient experienced a dramatic improvement in clinical status as well as in his quality of life (NYHA class I), requiring only routine follow-up visits for clinical and laboratory examinations. At the last visit, clinical examination revealed a body temperature of 37.8° C, blood pressure of 80/60 mm Hg despite the continued mechanical support, a heart rate of 80 beats/min, and a moist drive line exit site (percutaneous lead) with seropurulent material discharged (Figure 1A). Blood cultures revealed infection with methicillin-resistant *Staphylococcus aureus*, while the seropurulent material was positive for *Klebsiella pneumoniae*, sensitive to garamycin.

Assessment with two-dimensional transthoracic echocardiography revealed a 3 × 2 mm mobile echogenic vegetation, protruding from the inflow cannula of the device

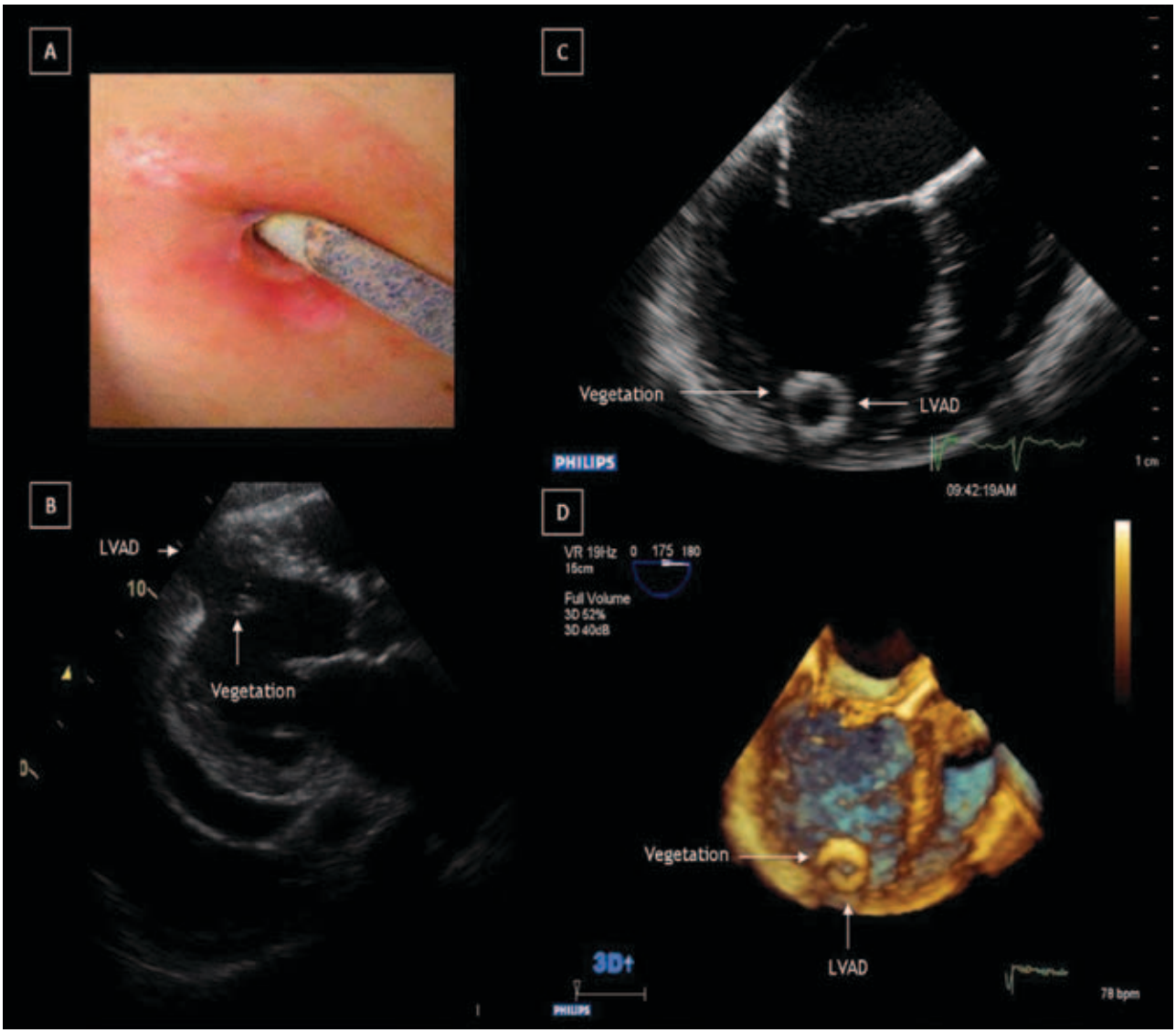

Figure 1. A. Left ventricular assist device (LVAD) exit site infection. B. Two-dimensional long-axis view reveals an echogenic material protruding from the inflow cannula of the device to the left ventricle. Moderate pericardial effusion and enlargement of the coronary sinus are also visible. C. Two-dimensional transoesophageal echocardiography revealed a thickened echogenic material (cross-section of the inflow cannula). D. Real-time three-dimensional echocardiography revealed a thickened echogenic material (cross-section of the inflow cannula).

inside the left ventricle (Figure 1B), while two-dimensional transoesophageal echocardiography and real-time three-dimensional echocardiography revealed a thickened formation adherent to the wall of the device (Figure 1C, D).

The treatment of choice in such patients is explantation of the device.[4] However, this was excluded since it was considered that the patient would not tolerate being "support-free" until the infection was eradicated. Important factors in the decision were that myocardial recovery, including the structural and functional remodelling of the left ventricle, was not yet complete, and the fact that implantation of a new device in a contaminated environment is an absolute contraindication. Our final decision was, therefore, to approach the double LVAD infection in a conservative way. Based on the bacterial sensitivity tests, the patient received a triple combination of intravenous antibiotics, consisting of vancomycin, 1 g × 2 daily, rifampicin 600 mg × 1 daily, and garamycin, 1 mg/kg × 3 daily, while the infection at the exit was treated with local infusions of garamycin. This regimen was continued for a period of six weeks, during which the patient's clinical status gradually improved, laboratory

tests returned to normal, and blood cultures became free of microorganisms. He was discharged with an antibiotic combination of linezolid, 600 mg × 2 daily, and rifampicin, 600 mg × 1 daily *per os*, for 6 months. Local garamycin was also maintained, as cultures from the cutaneous exit site continued to be positive for pathogens.

Using this combination, the patient remained in a stable clinical state, without signs of re-infection, for a period of five months. However, at five months following the presentation of infection, while still on antibiotics, the patient died due to an extensive intracranial haemorrhage following an overdose of self-administered anticoagulants.

Discussion

Mechanical unloading of ventricles with LVADs, in combination with interventions that interrupt pathological remodelling is at present a common approach in the management of patients with refractory heart failure resistant to medical treatment; in fact, it is the only way to achieve reverse remodelling of the myocardium.[5,6]

Current options are well defined for the treatment of prosthetic material endocarditis, which includes endocarditis due to prosthetic valves and devices such as pacemakers and defibrillators.[7] There is not yet complete consensus regarding the optimal choice of treatment for LVAD infection. Recent algorithms have been suggested according to the site of infection, whether it is at the pump internal lining, the pump pocket site, or the drive line exit site.[3] One of the options includes device explantation, if the patient is in a stable condition, followed, after management of the infection, by implantation of an alternative device or cardiac transplantation. Meanwhile, in complicated situations such as in our patient where, on the one hand, open heart surgery was contraindicated by the septic environment and, on the other hand, heart transplantation was not an option, having been excluded before LVAD implantation, our decision to adopt a conservative approach appeared to be the most appropriate.

Despite the fact that LVAD implantation is a revolutionary treatment for patients with end-stage heart failure, device-related complications remain the main weaknesses of this therapeutic approach, compared to heart transplantation. Our belief is that further advances in technology, lower costs of these devices and larger clinical trials are needed to establish LVAD implantation as an alternative therapeutic option to heart transplantation in everyday clinical practice.

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