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Intracranial Mycotic Aneurysm Associated With Left Ventricular Assist Device Infection

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Abstract

Infection is a frequent complication of left ventricular assist device (LVAD) use in patients with severe heart failure. The development of bacteremia with “LVAD endocarditis” is a serious infection that has rarely been associated with mycotic aneurysm formation. We describe the first case of an LVAD-associated intracranial mycotic aneurysm. We suggest that this is an underappreciated condition that merits more aggressive evaluation and treatment in patients with chronic LVAD-associated infections.

Left ventricular assist devices (LVADs) are being increasingly used in patients with severe refractory heart failure as a bridge to heart transplantation or as destination therapy [1].

Implantation of an LVAD comes with significant risks, and up to 60% of patients develop an LVAD-related infection [2]. Chronic infections with biofilm formation of the driveline, pump pocket, and intravascular pump/cannula components may lead to bacteremia or fungemia that is very difficult to cure without removal or exchange of the device [3].

Mycotic aneurysms may develop in patients with bloodstream infections, particularly in the context of infective endocarditis. They are associated with significant morbidity and mortality [4], and the relatively high frequency of bacteremic events in patients with LVADs puts this population at high risk for mycotic aneurysms. We describe one of the first cases of an intracranial mycotic aneurysm in a patient with an LVAD.

A 64-year-old man with ischemic cardiomyopathy and an ejection fraction of 24% underwent implantation of a HeartMate II LVAD (Thoratec Corp, Pleasanton, CA) as a bridge to transplantation. He received appropriate anticoagulation for his LVAD. Approximately 3 months after implantation, he experienced fever. On examination, he was hemodynamically stable and in no distress. The driveline exit site was without evidence of infection, and the results of his examination were otherwise unremarkable. Laboratory studies revealed a (stable) creatinine level of 2.0 mg/dL and a white blood cell count of 16,000/mm³. Ultrasonography showed multiple small fluid collections along the driveline, raising a concern for abscesses, which subsequently resolved with antimicrobial therapy.

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Multiple blood cultures grew *Pseudomonas aeruginosa* attributed to infection of his driveline.

He was treated with ceftazidime intravenously for 2 weeks and then transitioned to long-term suppressive therapy with oral ciprofloxacin. However, he had multiple episodes of breakthrough *P. aeruginosa* bacteremia over the next 10 months. His antimicrobial agents at various times included ceftazidime, ciprofloxacin, tobramycin, and combinations of these agents, and some of the isolates developed resistance to those medications.

Six months after his initial infection, he experienced increased somnolence and dysarthria. A noncontrast computed tomographic (CT) scan of his head revealed a small subarachnoid hemorrhage in the right parietal lobe. Cranial CT angiography did not reveal an aneurysm or infarct. He recovered with supportive care. However, 4 months later he experienced similar symptoms, and CT angiography demonstrated a beaded appearance of a right occipital cortical vessel with surrounding intraparenchymal hemorrhage suggestive of a small mycotic aneurysm (Fig 1). His international normalized ratio was within the therapeutic range, approximately 2.0. The lesion was deemed too distal for endovascular repair and the patient unfit for surgical intervention. He was transitioned to hospice care and died shortly thereafter.

Comment

The use of an LVAD in patients with advanced heart failure results in a clinically meaningful survival benefit and improved quality of life [5]. Newer continuous flow devices have reduced complication rates, but sepsis still develops in as many as 36% of patients. Infection of the internal portions of the pump or cannula of LVADs may be considered “LVAD endocarditis” and often presents like prosthetic valve endocarditis [2, 3]. Complications include internal LVAD thrombosis, LVAD dysfunction, and septic emboli. Neurologic complications are also frequent, with an overall 11% incidence of first stroke at 1 year and 17% at 2 years [1]. Cerebral emboli occurred in 18% of LVAD patients in one study and may be more frequent in patients with bacteremia [5].

Mycotic aneurysms have only been described in surprisingly few patients with LVADs and were primarily related to direct inoculation of the aorta or left ventricle as a result of the LVAD itself [6, 7]. They can affect any vessel and typically arise in the setting of bacterial inoculation into an arterial wall at the time of vascular injury or seeding of an existing arterial injury, atherosclerotic plaque, or preexisting aneurysm. Intracranial mycotic aneurysms are almost exclusively described in the setting of infective endocarditis, as a complication in about 2% to 4% of endocarditis patients [4]; to our knowledge, this has not been reported in an LVAD patient.

Intracranial mycotic aneurysms tend to be clinically silent unless there is concomitant embolic infarction or aneurysmal rupture with intraparenchymal or subarachnoid hemorrhage. Mortality associated with mycotic aneurysms ranges from 5% to 36% but is significantly higher in the setting of rupture. Analogous to patients with valvular infective endocarditis, in whom screening for cerebrovascular complications detected a large number

of silent lesions [8], we suspect that this condition is underappreciated and undertreated in LVAD patients, given their risk factors, frequency of chronic infections, and proclivity for bacteremia with septic embolic events. A low threshold of suspicion for this condition with aggressive workup and treatment is crucial to improving outcomes in affected LVAD patients.

Treatment strategies for intracranial mycotic aneurysms are controversial and typically involve a combination of antimicrobial, surgical, or endovascular approaches [4]. Morbidity and mortality are higher in patients with ruptured aneurysms; antimicrobial therapy in combination with surgical intervention, endovascular intervention or both results in better outcomes in this setting. Patients with unruptured aneurysms may be treated with antibiotics alone, during close monitoring with serial imaging. Patients with LVADs are at particularly high risk for complications, given the need for anticoagulation; the risks and benefits of holding anticoagulation in this setting must be carefully weighed and have not been studied.

Treatment of LVAD endocarditis typically involves parenteral antibiotics for at least 2 weeks followed by long-term suppressive therapy with oral antibiotics (if appropriate) until the LVAD can be explanted for transplantation or potentially exchanged [2, 3]. Source control should be aggressively pursued if feasible. All patients with LVAD endocarditis and new neurologic symptoms should receive neuroimaging. If a suggestive abnormality is found, we recommend dedicated vascular imaging to evaluate for mycotic aneurysms. CT angiography is a reasonable first diagnostic modality, although magnetic resonance imaging and conventional angiography may be useful adjuncts to detect smaller lesions if the result of initial testing is negative and the index of suspicion is high [4].

In summary, we report the first case of an intracranial mycotic aneurysm in a patient with a chronic LVAD infection. This highly morbid condition is likely underrecognized in this patient population and warrants a low threshold for additional evaluation and treatment. Given the advancement in radiographic, surgical, and endovascular techniques for diagnosing and treating intracranial mycotic aneurysms, early identification of this disease in patients with LVADs may lead to improved outcomes.

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Fig 1.
Cerebral computed tomographic angiogram demonstrating intraparenchymal hemorrhage in the right occipital lobe around a beaded right occipital artery (arrow) consistent with a ruptured mycotic aneurysm.