Percutaneous Closure of a Patent Foramen Ovale to Prevent Paradoxical Thromboembolism in a Patient With a Continuous-Flow LVAD

Carlo R. Bartoli, PhD¹, Kelly C. McCants, MD², Emma J. Birks, MD², Michael P. Flaherty, MD, PhD², Mark S. Slaughter, MD³

ABSTRACT: Patent foramen ovale (PFO) may complicate left ventricular assist device (LVAD) therapy. We report a 70-year-old male with a HeartMate II LVAD at increased risk for thromboembolic stroke secondary to a PFO with right-to-left shunting and a large mobile thrombus on his right atrial pacing lead. Via percutaneous intervention, a 25 mm Cribriform Amplatzer atrial septal occluder was successfully deployed across the PFO without complications. This is the first reported case of percutaneous PFO closure to prevent paradoxical thromboembolism in a normoxic patient with an LVAD. In addition, arterial desaturation and the sequelae of chronic hypoxemia were prevented. Strategies to diagnose PFO at the time of LVAD implantation and physiological implications of a right-to-left atrial shunt during mechanical unloading of the failing left ventricle are reviewed.

J INVASIVE CARDIOL 2013;25(3):154-156

Key words: percutaneous, patent foramen ovale, paradoxical thromboembolism, pacemaker lead, left ventricular assist device

Over the past decade, left ventricular assist devices (LVADs) have emerged as a standard therapy for patients with advanced heart failure.¹ Multiple devices are commercially available or poised for clinical trials.²

Common pathologies may complicate prolonged mechanical circulatory support. For example, undiagnosed patent foramen ovale (PFO) predisposes a patient with an LVAD to life-threatening hypoxemia.³⁻¹⁴ As mechanical circulatory support gains prevalence, it is important to raise awareness of potential complications and to describe interventions that may be performed safely during LVAD support. In this report, we describe the first case of percutaneous PFO closure to prevent paradoxical thromboembolism in a patient with an LVAD. Strategies to diagnose PFO at the time of LVAD implantation and physiological implications of a right-to-left atrial shunt during mechanical unloading of the failing left ventricle are reviewed.

Case Description. A 70-year-old white male with a history of ischemic cardiomyopathy and a dual-chamber pacemaker was implanted with a HeartMate II LVAD (Thoratec Corporation). During the course of therapy, a left middle cerebral artery infarction produced mild dysarthria and right upper extremity weakness. After 7.5 months of LVAD support, the pump was exchanged for a second HeartMate II due to pump thrombosis. After 17 months of support, the patient presented with non-cardiac left upper quadrant pain surrounding the pump. The patient described the pain as intermittent and sharp with no radiation, shortness of breath, dyspnea, or diaphoresis.

On admission, lipase and amylase were mildly elevated. Abdominal computed tomography was normal. Transesophageal echocardiography (TEE) identified a previously undiagnosed PFO with mild right-to-left shunting during quiet respiration and marked shunting during Valsalva maneuvers (Figure 1A). A 2.1 x 0.6 cm mobile thrombus was noted on the right atrial pacing lead (Figure 1B). Due to the increased risk for thromboembolic stroke and/or spontaneous deoxygenation at rest, the PFO was repaired. In the cardiac catheterization laboratory, the patient received cefazolin 2 g, heparin 50 units/kg (target activated clotting time >250 s), clopidogrel 600 mg, aspirin 325 mg, and midazolam 1 mg. The patient was not intubated. The left groin was prepared and draped in a sterile fashion. Lidocaine 2% solution was administered for local anesthe sia. Venous access was obtained via modified Seldinger technique. Due to the potential risk of dislodging the pacing lead thrombus, a right heart catheterization was not performed to measure hemodynamics or shunt fraction.

A 6 Fr multipurpose catheter back-loaded on a 0.035 mm Wholey wire was placed in the fossa ovalis and easily traversed the interatrial defect. The multipurpose catheter was advanced into the left upper pulmonary vein. A 0.035” Amplatzer super-stiff wire was advanced through the multipurpose catheter. The multipurpose catheter was removed. An 18 mm Amplatzer sizing balloon was placed across the interatrial septum and measured the PFO at approximately 12 mm in diameter. Of note, we routinely balloon-size PFOs and ASDs to guide device selection. Balloon sizing demonstrates the relative size, shape, and morphology (aneurysmal, hypertrophic, eccentric, etc.) of the defect as well as the location in the atrial septum.

A 25 mm Cribriform Amplatzer septal occluder was advanced across the PFO. The left atrial disc was unheated. The right atrial disc was unheated. Successful deployment of the septal occluder across the interatrial defect was confirmed via cineangiogram (Figure 2A) with radio-opaque contrast material to delineate the right atrial border and exclude a residual shunt.¹⁵

The patient tolerated the procedure well. There were no perioperative or postoperative complications. The patient received clopidogrel 75 mg daily for 6 months and continued aspirin 81 mg daily and warfarin 12 mg daily. Postoperative day 1 transthoracic echocardiography (TTE) demonstrated the closure device across the atrial septum with no shunt via bubble study or color Doppler with

From the ¹MD/PhD Program, University of Louisville School of Medicine, Louisville, Kentucky, ²Division of Cardiovascular Medicine, University of Louisville, Louisville, Kentucky, and ³Division of Thoracic and Cardiovascular Surgery, University of Louisville, Louisville, Kentucky.

Disclosure: The authors have completed and returned the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Birks reports honoraria and a research grant from Thoratec. The other authors report no conflicts of interest regarding the content herein.

Manuscript submitted August 30, 2012, provisional acceptance given October 9, 2012, final version accepted December 5, 2012.

Address for correspondence: Mark S. Slaughter, MD, Division of Thoracic and Cardiovascular Surgery, University of Louisville, 201 Abraham Flexner Way, Suite 1200, Louisville, KY 40202. Email: mark.slaughter@louisville.edu
acutely increases right atrial pressure, decreases left atrial pressure, and provokes right-to-left shunting through a subclinical PFO. Positive end-expiratory pressure (PEEP) may aid in the diagnosis of a PFO by a similar mechanism. PEEP increases intrathoracic pressure, which temporarily increases pulmonary vascular resistance and right atrial pressure. In patients with a PFO, this technique increases the transatrial shunt fraction. Alternatively, intraoperative TEE performed with the LVAD running may facilitate diagnosis. During mechanical unloading with a continuous-flow LVAD, the left ventricular pressure-volume relationship collapses, and left ventricular pressure remains low throughout the cardiac cycle. Left atrial pressure decreases, and the pressure gradient across the interatrial septum reverses. If a PFO is present, a right-to-left shunt may emerge on intraoperative TEE. Indeed, in a patient with a PFO, when the LVAD was turned off, right-to-left shunting ceased.

Despite these techniques, a PFO may not be identified during LVAD implantation. Multiple reports have documented PFOs that complicated LVAD therapy. In these cases, right-to-left shunting predisposed to arterial desaturation, hemodynamic instability, paradoxical embolism, ventricular volume overload, myocardial hypertrophy, and (worsening of) heart failure. Patients typically reported periodic episodes of symptoms related to intermittent hypoxemia despite adequate pulmonary function and arterial blood flow. In these patients, mechanical unloading of the failing left ventricle reduced left atrial pressure below right atrial pressure. Since a PFO was present, deoxygenated venous blood followed the pressure gradient, entered the left atrium, and decreased systemic arterial oxygen saturation. The severity and onset of symptoms were unpredictable, but likely related to the pressure gradient across the interatrial septum, PFO size, shunt fraction, preload and afterload conditions (volume status), specific anatomy of the defect, and the influence of gravity with positional changes.

The hypercoagulable blood dyscrasia associated with prolonged blood contact with the interior of the LVAD may cause devastating thromboembolic events. Yet in most patients, the LVAD is not the only potential source for thrombus. Many patients with advanced heart failure are referred for LVAD therapy only after electrical resynchronization therapy has failed. As a result, most candidates for long-term mechanical circulatory support have intracardiac pacemaker leads in situ prior to LVAD implantation. Consequently, intracardiac leads may be an under-recognized nidus for thrombus formation. Indeed, thrombus

---

**Figure 1.** (A) In a patient with a HeartMate II left ventricular assist device, transesophageal echocardiography with color Doppler revealed a patent foramen ovale with a right-to-left shunt (white arrow). (B) A large, mobile thrombus on the right atrial pacing lead was also identified (white arrow). LA = left atrium; RA = right atrium.

**Figure 2.** (A) Due to the increased risk of paradoxical thromboembolism in this patient, percutaneous patent foramen ovale closure was performed. LAO projection: (1) In situ 25 mm Cribiform Amplatzer septal occluder; (2) HeartMate II left ventricular assist device inflow cannula; (3) right atrial pacing lead; (4) right ventricular pacing/defibrillation lead. (B) Postoperative day 1 transthoracic echocardiogram with color Doppler demonstrated the interatrial position of the atrial septal occluder (white arrow). (C) At 6-week follow-up, a close-up view of the interatrial septum with color Doppler demonstrated the interatrial position of the atrial septal occluder (white arrow) and the absence of an interatrial shunt.
is identified on 9%-32% of permanent transvenous pacemaker leads.25,26 Furthermore, paradoxical thromboembolic stroke attributed to a pacemaker lead thrombus has been described.27,28 These findings suggest that indwelling pacing leads should be considered as a source for unexplained thromboembolic complications, especially in patients in whom an LVAD may be blamed as the default source of thrombus.

Management of a patient with a history of cryptogenic stroke and evidence of a PFO includes thrombolysis, chronic anticoagulation, and closure of the PFO. Percutaneous PFO closure is a standard treatment for patients with hypoxemia secondary to right-to-left shunting.29 Importantly, percutaneous PFO closure to correct hypoxemia has also been described in hypoxic adult6,9,10,12,13 and pediatric3 patients with an LVAD. In these reports, PFO closure resulted in rapid resolution of hypoxemia and permanent symptom resolution.

In the absence of hypoxemia, a PFO is not an absolute indication for prophylactic closure unless recurrent paradoxical thromboembolism is suspected.30 In our normoxic patient with a history of embolic stroke, a large mobile thrombus on his right atrial pacing lead, and a PFO with intermittent shunting, recurrent cryptogenic stroke was a major concern. Consequently, percutaneous PFO closure was an appropriate prophylactic intervention.

Of note, when pacemaker leads reside in close proximity to a PFO, lead entrapment in the atrial septal occluder may interfere with adequate anchoring of the device within the interatrial septum. Incorrect device deployment may prevent closure of the foramen and impair endothelialization of the device.31,32 In patients who undergo percutaneous closure of a PFO, entrapment of pacing leads in the right atrial disc of the atrial septal occluder should be avoided.

In summary, we have presented the first case of percutaneous PFO closure to prevent paradoxical thromboembolism in a normoxic patient with an LVAD. Prior history of cryptogenic stroke should raise suspicion for a PFO. Intracardiac pacing/defibrillator leads may be a source of thrombus. If a PFO is identified in a patient undergoing long-term mechanical circulatory support, prophylactic closure should be considered.

**Conclusion.** A PFO discovered during the implantation of an LVAD is routinely closed to prevent sequelae of right-to-left shunting during prolonged mechanical circulatory support. Simple screening maneuvers may assist in the intraoperative diagnosis of a PFO via TEE. In a normoxic patient in whom a PFO is identified during ongoing LVAD therapy, percutaneous PFO closure may be appropriate if there is concern for paradoxical thromboembolism.

**Acknowledgments.** The authors acknowledge and thank the University of Louisville Division of Cardiology VAD Coordinators.

**References**