Pulmonary artery dissection in a patient with right sided mechanical circulatory support and LVAD.

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1. Case Report

The use of left ventricular assist device (LVAD) is an essential treatment option for patients with advanced heart failure, as both a bridge to transplant and destination therapy. It is important to consider the risks associated with an LVAD, however, as complications can present with a range of severity and chronicity. In the perioperative setting of LVAD implantation, right ventricular failure (RVF) can occur in an estimated 20% of patients.[1,2] RVF increases both morbidity and mortality, and it is reported that the 1-year survival rate is 60% in patients requiring biventricular support devices.[1,2] Temporary percutaneous right ventricular assist devices (RVAD) can be used until hemodynamic stability is achieved, or until the patient is bridged to permanent RVAD or cardiac transplantation. In recent years, TandemLife ProtekDuo (TandemLife, Pittsburg, PA) (TPD) or CentriMag (Abbott, Chicago, IL) have been introduced as options for temporary right ventricular support. However, temporary RVADs can also cause a variety of complications, including but not limited to tricuspid regurgitation, hemolysis, cannula migration, or

cerebrovascular insults.[1,3] When indicated, patients who require biventricular devices are at much higher risk of complications and adverse events, as compared to those with LVAD or RVAD alone. Despite the documented risk, temporary RVADs remain one of very few options to treat RVF peri-operatively and must be considered.

Here, we report a case of acute right heart failure immediately following LVAD implantation. The patient was supported initially on CentriMag, then subsequently on TPD. He then presented nearly 2 years later with pulmonary artery dissection (PAD). We describe this rare complication and review management strategies.

The patient is a 49-year-old-male with medical history of anterior ST elevation myocardial infarction requiring a coronary stent to the left anterior descending artery. He presented 7 months post-procedure with refractory cardiogenic shock. Transthoracic echocardiogram (TTE) was notable for left ventricular ejection fraction of 16%. Right heart catheterization (RHC) showed cardiac index of 0.9 L/min/m^2, cardiac output of 2L/min, and elevated wedge pressure of 30 mmHg. Pulmonary artery pressure was 37/14 mmHg (mean 25), which indicated only mild pulmonary hypertension. Patient was initially stabilized by the placement of an Impella 5.0 (Abiomed Inc, Danvers, Massachusetts, USA), and subsequently underwent LVAD implantation. Intraoperatively after weaning from bypass machine, right heart started to dilate with deterioration of right heart function. The flow on the LVAD decreased to less than 2L. He then suffered a cardiac arrest, necessitating open cardiac massage to achieve return of spontaneous circulation. Given that the LVAD flow was less than 3L and right ventricular function remained severely reduced, temporary RVAD (CentriMag) was implanted emergently. The outflow drainage cannula of the RVAD exited the right atrium and the inflow canula entered the main pulmonary artery. CentriMag provided approximately 5L/min of flow, which allowed the LVAD flow to increase above 4L/min.

Transesophageal echocardiogram (TEE) performed immediately after CentriMag placement was notable for a decompressed right ventricle. Despite medical optimization and continued support on CentriMag for several days, there was no improvement in right heart function. Thus, the decision was made to exchange CentriMag for 31 French TPD via internal jugular vein on day 4. TPD was placed without complications, and post-operative TEE was unremarkable. TPD provided up to 5 L/min of flow at 4600 rpms. His right ventricular function gradually recovered, and the TPD was removed following 20 days of support. His index hospitalization was further complicated by a cerebrovascular accident post-LVAD placement. Although the patient underwent successful thrombectomy, he had residual left-sided hemiparesis and significant debility. Of note, computed tomography (CT) scan of the chest without contrast obtained 2 months after LVAD implantation demonstrated an unremarkable pulmonary artery without any evidence of dissection.

The patient re-presented 22 months after LVAD implantation with intense chest pain that he described as the sensation of "muscle tearing". Emergent computed tomography angiography (CTA) scan of the chest was notable for a dissection of the left pulmonary artery. The dissection flap began immediately distal to the main pulmonary artery and involved proximal to distal left pulmonary artery. It extended to the level of the left lower interlobar branch (Figure 1). The maximum overall diameter of the flap measured 1.1cm, whereas the true lumen at that level of the pulmonary artery measured 5mm. There was no thrombus on radiographic exam, and the dimensions of the pulmonary artery were within normal limits.

Multi-disciplinary discussions evaluated all possible treatment options, including open surgical repair, endovascular stenting, and medical management. Given his significant comorbidity, including left-sided hemiparesis, the patient was assessed to not be a candidate for surgical intervention. Therefore, medical management was offered. His acute chest pain was managed with

morphine. It completely resolved by 48 hours after presentation, without recurrence. Repeat CT scan completed 3 days after the diagnosis of PAD confirmed the dissection to be stable, without any identified progression. He was discharged from the hospital with close outpatient follow-up. CTA (Figure 2) of the chest was repeated 3 months later, which again revealed stable left PAD without progression. For the past 12 months, the patient has remained without any complications of the pulmonary artery dissection. He continues to follow closely with the advanced heart failure clinic for optimization of heart failure medications and other medical management.

2. Discussion

Data on pulmonary artery dissection is extremely limited. It is a rare condition with less than 200 cases identified in the literature. Herein, we report the first case of PAD in a patient with a left ventricular assist device. Most cases of PAD are attributed to pulmonary hypertension and a dilated pulmonary artery. It has also been documented in individuals with acquired and congenital heart diseases, most often affecting patients with a patent ductus arteriosus.[4] Anatomically, when a tear in the intima occurs, it can progress into the mid or deep media. This can eventually lead to dissection of the pulmonary artery, which then tends to propagate into the intramedial planes.[5] If the media is exposed to elevated blood pressures, a false lumen can result.[5] In most cases of PAD, rupture is more likely to occur than the creation of a re-entry pathway. This is due to the significantly thinner media in the pulmonary artery, when compared to the aorta.[4, 5] Rupture can occur in the pericardium, mediastinum, lungs, or pleural cavity.[6]

D.M.G Fernando et al published an extensive review of 150 PAD cases reported in the literature that demonstrated significance between gender and age at diagnosis.[4] Males are most likely to be diagnosed with PAD in the third or fourth decade of life, whereas incidence in females is most common in the fifth or sixth decades of life.[4] More than 50% of patients report the presenting

symptom to be chest pain or dyspnea, which was emulated by the chief complaint in our patient.[4] The preferred imaging modality for diagnosis is CT scan, when available.[4]

There is no evidence-based consensus for the acute management or chronic treatment strategy for PAD. Several approaches such as open surgical repair, endovascular stenting, and medical management have been reported in the literature with varied success. In the review published by D.M.G Fernando et al, management guidelines were included in just over half of the cases. Medical and surgical management strategies were split almost evenly.[4] Medical management included vasodilators (ACE inhibitors, calcium channel blockers), diuretics, and beta-blockers.[4] The success rate was 67% among patients treated with conservative management, although 3 patients did ultimately require surgical intervention. Although D.M.G Fernando et all reported successful outcomes in 90.3% of cases treated surgically, other included cases series suggested that medical treatment is preferred when surgical risk is high. Alternative cases further supported that conservative management is acceptable for asymptomatic patients. This was pertinent to the medical decision making for our reported patient. Given his LVAD and significant comorbidities, he was a poor surgical candidate. Accordingly, it was decided that medical management was indicated. One year after PAD diagnosis, he remains on losartan, carvedilol, and spironolactone without any complications.

To date, there are no reported case of PAD caused by an iatrogenic etiology in patients with normal caliber pulmonary arteries, normal pulmonary pressures, or anatomically normal heart structure. However, pulmonary artery (PA) catheters have been reported as a potential source for PA rupture and pseudoaneurysm.[7] Alternatively, there are cases of traumatic aortic canulation during cardiopulmonary bypass resulting in aortopulmonary window and PAD.[8] Although the exact etiology of pulmonary artery dissection in our patient is unclear, it is possible that PAD resulted

from previous interventions to the pulmonary artery. Prior to his diagnosis of PAD, he underwent pulmonary artery catheterization, emergent placement of CentriMag, followed by TPD for over 3 weeks. Following TPD removal, however, no PAD or pulmonary artery aneurysms were identified. Additionally, the patient had no evidence of pulmonary artery hypertension prior to LVAD placement, and pulmonary artery dimensions have been normal on all imaging to date. A TTE performed 3 months after TPD removal was without any evidence of pulmonary hypertension or valvular abnormalities. The patient has also remained free of overt signs or symptoms of pulmonary artery hypertension or worsening right heart failure (RHF) such as progressive dyspnea, lower extremity edema, or congestive hepatopathy. No other intervention or hemodynamic change caused by his LVAD explain the presentation of PAD in our patient. It is documented that weakening of the aortic wall predisposes to aortic dissection. It is possible that the pulmonary artery wall may have been damaged by previous interventions, thereby predisposing it to acute dissection.

In summary, we report the first documented case of PAD in a patient with a history of LVAD implantation. He was managed medically and has remained healthy for the past 12 months. PAD ranges in severity, although it can be fatal. Postmortem studies have confirmed PAD to cause hemopericardium, hemothorax, pulmonary hemorrhage, hemomediastium, and death. Perioperative attention to this potential complication of right sided mechanical circulatory support may reduce its incidence. When possible, early diagnosis of PAD with CT scan can guide medical and surgical interventions. As mechanical circulatory support continues to advance in technology and expand in clinical practice, it is important to consider PAD as a rare complication of prior intervention or instrumentation in the pulmonary artery.

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Figures:

1. Figure 1: Computed tomography angiography (CTA) scan of the chest showing the maximal diameter of the PAD flap (arrow) measuring about 1.1cm with true lumen measuring about 5mm.

2. Figure 2: CTA scan of the chest with intravenous contrast at 3-month follow-up with no notable change in PAD (arrow) per formal radiology read.