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Berlin Excor Cannulation of Left Atrial Appendage in Left Ventricular Restrictive Physiology: A Novel Bailout Strategy

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Abstract: Ventricular assist device (VAD) management continues to be a challenge in the presence of restrictive physiology. Left atrial (LA) decompression is not satisfactory even with good function and position of the left ventricular cannula. We describe an alternate approach with LA cannulation *via* the left atrial appendage (LAA) as a rescue strategy in a patient who had restrictive physiology, in our case was secondary to viral myocarditis acute systolic heart failure with subsequent insidious diffuse endomyocardial fibrosis and superimposed massive calcification, causing inadequate emptying of the left ventricle despite optimal VAD apical cannula position. *ASAIO Journal* 2021; 67;e157–e159

Key words: myocarditis, acute heart failure, ventricular assist device, Berlin Heart Excor, left atrium, left atrial appendage, inflow cannula

Extracorporeal ventricular assist device (VAD) support is commonly used in young children with severe heart failure to support them to recovery or heart transplant as the children are too small to be supported with adult-type intracorporal VADs. Some pediatric centers prefer the centrimag/pedimag for shortterm support given more experience with this device. However, the Berlin Heart Excor has become the preferred VAD in other centers for children who require medium to long-term VAD. As worldwide experience with this type of VAD has expanded, there have been improved outcomes¹ likely related to changes in the anticoagulation management, patient selection, a better understanding of VAD management, and also attributed to increased surgical experience with the implant techniques which improves hemodynamics.

VAD management continues to be a challenge in the presence of restrictive physiology. Left atrial decompression is not

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satisfactory even with good function and position of the left ventricular cannula. To combat this problem, techniques have been developed to cannulate the left atrium (LA) directly, particularly in hypertrophied ventricles or when the size of the ventricle would not allow for the appropriate position of the inflow cannula.² The surgical technique of LA cannulation has been described previously,³ and typically involves cannulation of the rightward aspect of the LA directly or *via* the right pulmonary vein.

Herein, we describe an alternate approach with LA cannulation *via* the left atrial appendage (LAA) as a rescue strategy in a patient who had restrictive physiology secondary to viral myocarditis acute systolic heart failure with subsequent insidious diffuse endomyocardial fibrosis and superimposed massive calcification, causing inadequate decompression of the left ventricle (LV) despite optimal VAD apical cannula position.

CASE REPORT

A 6-year-old, 17 kg female presented to the emergency department with fever, tachycardia, and respiratory distress. Echocardiogram revealed a severely depressed bi-ventricular function. She rapidly decompensated with the development of acute cardiorespiratory failure and required intubation, and initiation of inotropic support with dopamine and epinephrine. Progression with acute pulmonary edema and ongoing severe cardiac failure refractory to maximal medical therapy required advanced mechanical support with peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO) from femoral vessels approximately 12 hours after presentation. Because of left atrial distention on echocardiography, the patient was taken to cardiac catheterization for atrial septostomy. Her medical/surgical history was significant for renal transplantation at 2 years of age secondary to congenital renal dysplasia. Her post-renal transplant course was complicated by chronic graft dysfunction secondary to recurrent antibodymediated rejection, Epstein Barr Virus (EBV) viremia, and posttransplant lymphoproliferative disorder.

Further evaluation of her decompensation revealed most likely enteroviral myocarditis (serum PCR positive for enterovirus). On VA-ECMO, the flows were ranging from 2 to 2.5 L/ minute (LPM); however, despite the atrial septostomy the patient developed pulmonary hemorrhage and echocardiography showed evidence of ongoing severe left atrial hypertension with NT-pro B-type natriuretic peptide (BNP) 91.388 pg/ mL and troponin peak 151.966 µg/ml. Therefore, the patient was transitioned 10 days later from peripheral VA-ECMO to a Thoratec CentriMag (Abbott) Bi-VAD support with cannulation of the right atrium (RA)/pulmonary artery (PA) for the right VAD and the left atrium(LA)/Ascending Aorta (Ao) for the left VAD (LVAD). With this cannulation strategy, the patient was

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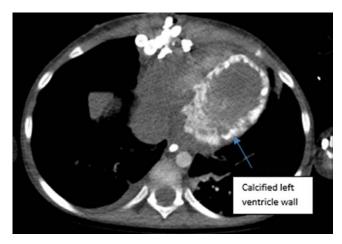


Figure 1. Axial computerized tomography (CT) scan showing markedly abnormal, diffusely calcified left ventricular wall.

well supported with excellent oxygenation, renal function, and adequate bi-VAD flows ranging from 2 to 2.5 LPM on the LVAD and 1.5 to 2.3 LPM on the RVAD. Conversion to longer-term mechanical support was needed 2 weeks later due to lack of myocardial recovery with ongoing systolic heart failure with left ventricular ejection fraction <20% preoperative head computed tomography (CT) scan showed subacute right middle cerebral artery stroke and foci of acute intraparenchymal hemorrhage within right frontal and parietal lobes. These findings were stable on repeat head CT 1 week later, and so the decision was made to proceed with Berlin Heart Excor (Berlin Heart Inc, The Woodlands, Tx) implant. After discussion with the technical support of Berlin Heart, we opted for a 25-mL Berlin Heart Excor Bi-VAD support, via 9mm RA and left ventricular apical cannulae, and 6mm pulmonary artery and ascending aortic cannulae. The settings on the Berlin Heart were adjusted to maintain optimal filling and emptying of both RVAD and LVAD, with initial settings with LVAD rate: 90 BPM, systolic pressure 210, diastolic pressure -40, and 35% systole, with RVAD Rate: 87 BPM, systolic pressure 175, diastolic pressure -25 and 35% systole.

Despite initial optimal filling and emptying of both VADs, in the following days, we encountered persistent difficulty with the filling of the LVAD with worsening pulmonary edema. This inadequate filling was not responsive to any adjustments of the Berlin settings in terms of diastolic pressure, rate of the device, and synchrony between the two VADs. Furthermore, the native heart was relatively bradycardic (maximum 80 beats/minute), which notoriously is a favorable factor for Berlin LVAD mechanics. The inadequate filling was thought to be secondary to restrictive physiology from the development of diffuse endomyocardial fibrosis and massive calcifications in the left ventricle (LV) myocardium (Figure 1). Because of this, the plan was to take the patient back to the operating room to change the left side inflow cannulation from the left ventricular apex to the LA. This was accomplished with the addition of a LA cannula through the LAA and using a Y-connection with the left ventricular apical cannula to a CentriMag continuous flow pump in exchange for the Berlin left-sided pump. The RVAD was also changed to a Centrimag pump at this time. The reason this unusual configuration was adopted was to avoid another run of cardiopulmonary bypass (CPB) in a very unstable patient. This improved the VAD filling and flow with guick resolution of the pulmonary edema (Figure 2), therefore in a further surgery 4 days later the LA cannula was then exchanged to a Berlin Heart 9 mm atrial cannula (Figures 3 and 4) and the LV apical cannula was removed (Figure 5) and apex of the heart repaired. She was then converted back to pulsatile Berlin Bi-VAD support with this new cannulation strategy. She was well supported for 2 months only requiring one RVAD pump exchange for thrombus, however unfortunately she had infectious and neurologic complications including seizures and diminished neurologic status, which eliminated her transplant candidacy; thus support was withdrawn.

DISCUSSION

Experience with VAD support has grown tremendously in the past 10 years, with now close to 40% of pediatric patients requiring mechanical support as a bridge to transplantation.⁴ Despite this, patients who have restrictive physiology (diastolic heart failure) continue to be a challenge as there is often inadequate unloading of the left atrium in these cases. Although our patient presented with systolic failure, she had evidence of diastolic dysfunction secondary to a severely calcified/fibrotic ventricle. In this case, the only way to adeguately unload the left atrium was with direct cannulation. However, from personal communications with Berlin Heart, we were aware of the increased risk of clot formation and thrombosis in left atrial cannulation as historically reported with the Berlin Heart Excor. This has been attributed to the turbulence of flow from LA cannulation despite optimal anticoagulation management. Despite optimal anticoagulation

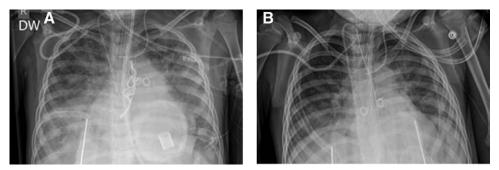


Figure 2. Intraoperative photo showing the heart is rotated to the right with 9 mm Berlin inflow cannula being sewn into left atrial appendage with 5-0 prolene sutures. A. Chest X-ray showing pulmonary edema with Berlin Heart 9 mm apical LV cannulation. B. Chest X-ray showing improved pulmonary edema with Berlin Heart 9 mm LA cannulation. LA cannulation, left atrial cannulation; LV, left ventricle.

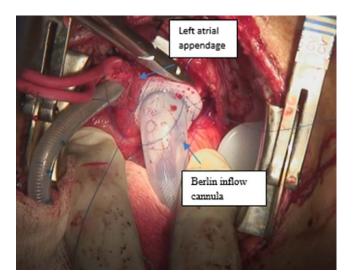


Figure 3. Intraoperative photograph showing the heart is rotated to the right with 9 mm Berlin inflow cannula being sewn into left atrial appendage with 5-0 prolene sutures.

and no direct evidence for thrombosis in LA cannula or LVAD, this patient experienced a devastating neurologic injury that was likely multifactorial and could be secondary to an infection, hypoperfused states throughout her course, or embolic events as evidenced by head CT changes even before the LA cannulation.

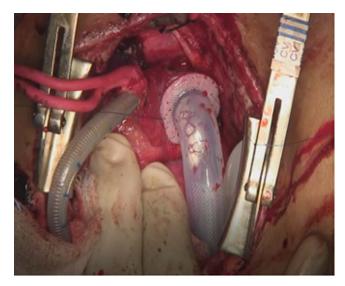


Figure 4. Intraoperative photograph showing 9mm Berlin inflow cannula tip positioned posteriorly and medially towards the left atrial cavity.

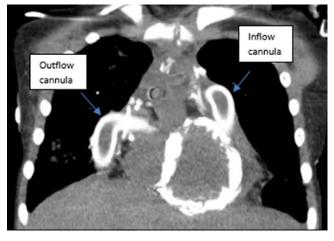


Figure 5. Coronal plane CT scan showing diffuse left ventricular wall calcification and both left atrial outflow and aortic inflow cannulae in position. CT, computerized tomography.

In other reports, the left atrium is cannulated *via* the right superior pulmonary vein or with the creation of atrial septal defect (ASD) and placement of an interposition graft to the cannula.⁵ This approach can lead to pulmonary vein stenosis, even after a transplant, and the approach with the creation of ASD requires CPB. The alternate approach we described above avoids pulmonary vein stenosis and allows an approach from the left side to minimize the risk of superior vena caval obstruction. In addition, our approach allowed us to cannulate the LA off the bypass in an otherwise very unstable patient and we utilized the same exit point on the chest of the left ventricular apical cannula. We can argue this cannulation strategy can be expanded for adequate left-sided heart drainage and a better chance of LV recovery and probably less risk for fibrosis/calcification injury development when there is inadequate unloading of the LV.

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