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Ligation of the Outflow Tract for the Treatment of Pump Thrombosis of a Left Ventricular Assist Device

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Abstract

Ventricular assist device thrombosis is a dreaded complication after implantation. Acute device thrombosis within one month of placement creates additional surgical complexities for the patient. Ligation of the outflow tract while leaving the ventricular assist device in place is an option.

Keywords: Ventricular assist; Anticoagulation; INTERMACS; Heartware

Introduction

Left ventricular assist devices have significantly changed the way physicians manage advanced heart failure. Ventricular Assist Device (VAD) thrombosis can occur for a variety of causes, including: inadequate anticoagulation and/or malpositioning of the inflow cannula. Traditional approaches to treat pump thrombosis include anticoagulation and/or exchange of the VAD [1]. We report a unique treatment of pump thrombosis management.

Case Report

A 67 year old male presented to an outside hospital with an anterior wall ST Elevation Myocardial Infarction (STEMI), had a cardiac catheterization and a drug eluting stent placed in the Left Anterior Descending artery (LAD) and diagonal arteries. He was transferred to our hospital for further care. During this time he also had two episodes of ventricular fibrillation requiring cardioversion. He was stabilized with a continuous amiodarone infusion at a rate of 1 mg/min and decreased to 0.5 mg/min. A transthoracic echocardiogram showed an ejection fraction of 24% with right ventricular systolic pressures of 40-45 mmHg and regional wall motion abnormalities in the LAD, circumflex, and right coronary artery territories. A subsequent, heart catheterization demonstrated patent stents in the LAD and the diagonal arteries and an elevated right ventricular pressure of 39/12 mmHg and a pulmonary artery pressure of 37/21 mmHg with a mean pulmonary capillary wedge pressure of 21. The left ventricular pressure was 95/3 mmHg. An Intra-aortic Balloon Pump (IABP) was placed.

During the first day he had an episode of bradycardia; dobutamine was started at 2.5 mcg/kg/min and increased to 5 mcg/kg/min. The dobutamine was eventually stopped and the IABP was removed. He went into cardiogenic shock and the IABP was replaced within 12 h and he was started on norepinephrine and milrinone at 0.25 mcg/kg/h. The norepinephrine was weaned off; however, the milrinone and IABP were unable to be discontinued. He was in cardiogenic shock with a New York Heart Association (NYHA) Class IV heart failure with a reduced ejection fraction with a life expectancy of less than 6 months and his Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) profile 2. A repeat echo showed an ejection fraction of 20% and regional wall abnormalities in the left anterior descending and right coronary artery territories. In addition, he had intermittent arrhythmias, including supraventricular tachycardia and

atrial fibrillation with rapid ventricular rate. The patient needed a left ventricular assist device in order to improve and be discharged.

The patient was taken to the operating room and a Heartware left ventricular assist device (Heartware Inc., Framingham, MA) was placed a month after the initial STEMI, without cardiopulmonary bypass. An echo was performed the following day which showed no systolic opening of the aortic valve at 2300-2500 revolutions per minute and minimal opening of the aortic valve at 2200 revolutions per minute. The IABP was removed on post-operative day 1 and he was extubated on post-operative day 4. The milrinone was weaned off and he was discharged home on post-operative day 18 on amiodarone 200 mg daily, furosemide 20 mg daily, losartan 25 mg daily, spironolactone 25 mg daily, and warfarin 4 mg daily.

The patient presented to an outside hospital with complaints of his ventricular assist device alarming 13 days after discharge. The International Normalized Ratio (INR) was sub-therapeutic at 1.2 and the Lactate Dehydrogenase (LDH) level was greater than 1000 Units/L. He was started on therapeutic enoxaparin at 90 mg every 12 h and transferred to our institution. There was concern for an acute LVAD thrombosis. The power on the LVAD was 15 Watts, the flow was >10 L, and the pump speed was 2,400 RPMs. A transthoracic echo showed an ejection fraction of 40-45% with mild left ventricular dilation. A cardiac catheterization was performed and he was administered 25 mg of alteplase in the left ventricle. His power decreased to 3.7 Watts and flows to 6.5 L. He received a bolus of eptifibatide at 180 mcg/kg and then a continuous infusion was started at 2 mcg/kg/min for 48 h along with a heparin infusion at 1,430 units/h.

Initially, the patient showed a response; however, on hospital day 3 the flow increased to >10 L and the power increased to 8.3 Watts. His arterial line was pulsatile and he was started on dobutamine at 3 mcg/

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kg/min. During this time, he was given another bolus of eptifibatide at 180 mcg/kg and then a continuous infusion at 2 mcg/kg/min for an additional 48 h. The LVAD flow continued to remain at 10 L with a power at 4.4 Watts, pump speed at 2400 RPMs, and the LDH level increased to 900 Units/L. There was continued concern the thrombosis had not resolved. A transthoracic echo was performed that showed an ejection fraction of 43% and aortic valve opening 1:1 at 2400 revolutions per minute.

The patient was approximately 1 month after the initial LVAD placement. The patient was higher risk for a redo sternotomy. He was about 4 weeks from his initial surgery; therefore, at increased risk of adhesions and reactive tissue bleeding. In addition, exchanging or explanting the LVAD would come with a higher morbidity. During this time the LVAD settings were able to be weaned to where he would not require the LVAD. Due to the recent echo findings, increased risk of bleeding from the recent Tissue Plasminogen Activator (tPA), and increased morbidity with a redo sternotomy, it was decided to ligate the outflow tract and leave the LVAD in place via subxiphoid approach. This treatment would provide the patient the best optimal outcome.

The patient was taken to the operating room. A pulmonary artery catheter was placed along with an arterial line and left subclavian central line. A limited subxiphoid incision was made and the outflow tract was identified. The dobutamine was increased to 5 mcg/kg/min and the outflow tract was encircled and clamped. The patient tolerated clamping the outflow tract well. The intraoperative transesophageal echocardiogram showed that the global right and left ventricular function was normal with an ejection fraction of 50%. There were no left ventricular wall motion abnormalities. The outflow tract was stapled off using a 30 mm TA stapler with a vascular load. The inflow line and LVAD were left intact. The drive lines were removed.

Postoperatively, he was extubated the same day and the dobutamine was weaned to 3 mcg/kg/min and discontinued on postoperative day 2. The rest of his hospital course was complicated by acute kidney injury (AKI) with his creatinine rising to 1.8 mg/dL and blood urea nitrogen (BUN) to 29 mg/dL The AKI resolved with a creatinine of 1.35 mg/dL before discharge.

The patient was discharged to home on post-operative day 10 on clopidogrel 75 mg daily, amiodarone 100 mg daily, digoxin 125 mcg daily, losartan 25 mg daily, spironolactone 25 mg daily, torsemide 40 mg and 20 mg alternating daily, and warfarin 5 mg once a day. At his three month visit, he was doing well. His NYHA functional classification was Class II. He had a transthoracic echocardiogram at the time that showed an ejection fraction of 55% with normal right ventricular pressures. Warfarin was discontinued at this time. At seven months from the operation, he was back working 12 h shifts three days a week. His NYHA functional classification remained at Class II.

Discussion and Conclusion

LVAD pump thrombosis occurs at a rate of approximately 0.08 events per patient-year [2]. Causes of pump thrombosis can be a result of a myriad of issues: sub-therapeutic anticoagulation, technical errors in implanting the ventricular assist device, low flow states, previous thrombus in the left atrium, and hypertension. Medical management with tPA has been attempted in the past with about 50-60% success rates [3]. In the remaining cases, the LVAD must be removed and/or exchanged. When pump thrombosis occurs in a relatively short time interval after the initial implantation and the patient is not dependent on the assist device, outflow ligation and removal of drive lines from a subxiphoid incision is an excellent surgical option for treatment of LVAD thrombosis with a lower surgical risk, and morbidity than an LVAD explanation and/or exchange.

Techniques for device removal have been well described. Redo sternotomies pose high morbidity, with an increased risk within the first few months of the index operation. This approach of outflow tract ligation through a subxiphoid incision is an alternative to a difficult and rare dilemma. The advantage of a subcostal approach along with leaving the device in place: Avoids a redo sternotomy; therefore, minimizes bleeding, transfusions, and operative time [4,5].

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