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Peer-Reviewed Case Report

A Unique Case of Inflow Cannula Obstruction by a Tissue Membrane

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Abstract

The use of a left ventricular assist device has increased and is a primary surgical treatment for heart failure. However, one major complication of left ventricular assist device support is an obstruction in the blood flow pathway. Pump thrombosis and outflow graft occlusion are some of the common causes of such obstructions. Here, we describe a unique case of HeartMate II (Abbott Laboratories) inflow cannula obstruction from a membranous structure without evidence of thrombus. The histology showed evidence of fibrous tissue and heart muscle tissue in the membrane. The patient underwent a successful device exchange with a HeartMate 3 (Abbott Laboratories) and is doing well.

Background

The left ventricular assist device (LVAD) has become one of the mainstream treatments for end-stage heart failure (HF). However, it is not free of complications. One of the dreaded complications is an obstruction in the blood flow pathway, which defeats the very purpose of the device. Thrombus formation and extrinsic compression of the outflow graft are some of the well-reported causes of such obstruction.[1-3] Further, inlet cannula obstruction from a thrombus formation can contribute to low flows and is well described.[4] Here, we report a rare case of complete inflow cannula obstruction from tissue ingrowth that caused the device to malfunction.

Case Report

A 57-year-old female received a HeartMate II (HMII, Abbott Laboratories, Abbott Park, IL) for end-stage HF from non-ischemic cardiomyopathy. The patient had significant other comorbidities, including diabetes mellitus, morbid obesity, remote history of thyroid cancer, chronic kidney disease (baseline creatinine of 1.8-2.5 mg/dL), and hypertension. Chronic driveline infections were treated with oral antibiotics, and therapeutic anticoagulation was maintained throughout her seven-year follow-up. Lactate dehydrogenase levels remained around 400-500 IU.

Even though the patient did well overall, routine device interrogation during clinic visits for the last two years showed random low flow alarms lasting only a few seconds. The patient was mostly asymptomatic through these low flow alarms. A workup was done using an institutional algorithm (Figure 1). Computed tomography angiography (CTA) showed extrinsic compression of the proximal portion of the outflow graft. Further evaluation of the extrinsic compression with cardiac catheterization showed no significant gradient. The peak systolic gradient between the origin and termination of the compression of the graft was 19 mmHg, and medical management was selected for the course of action.

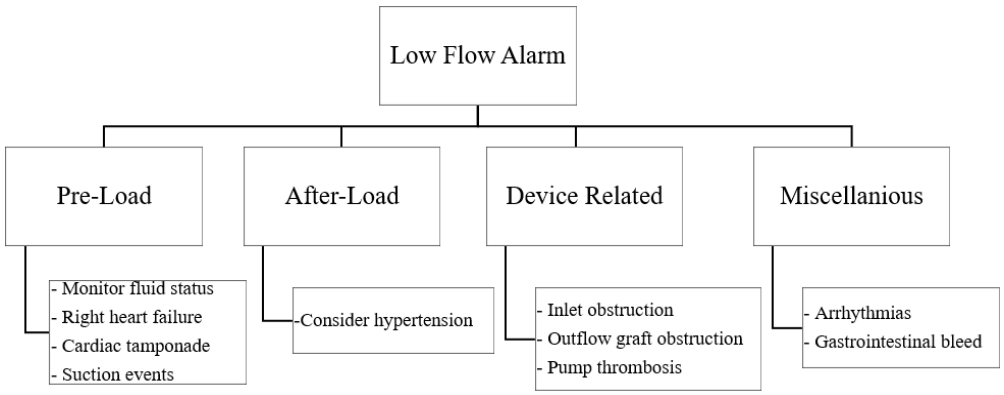

Figure 1. *Left ventricular assist device low-flow alarm workup process considerations.*

A follow-up CTA was completed a year later and showed no progression; however, the random low flow alarms continued. The transient, asymptomatic low flows noted on routine device interrogation were attributed to uncontrolled hypertension. The patient was noncompliant with medications, and her mean arterial pressures (MAPs) were consistently elevated to the hundreds during clinic visits. The device log files captured the intermittent transient low flow events while on the mobile power unit; thus, technical services were contacted. They concluded that this was a short-to-shield issue with the driveline and recommended that the patient use battery power or an ungrounded cable moving forward.

The patient presented to the emergency room one year later with continuous low flow alarms. Log files showed random low-flow alarms, low-speed advisories, and a pump-off event. The patient was admitted to the hospital for further workup. She experienced intermittent prolonged episodes of pump stoppages during the hospitalization. The CTA was notable for eccentric low density within the proximal aspect of the outflow graft. A narrowing of the lumen was noted (1.6 x 0.9 cm versus the normal luminal diameter of ~1.8 cm), and the results had not changed compared to the previous imaging studies. An x-ray of the chest showed device malposition compared to a radiograph at the time of implantation (Figure 2). An echocardiogram revealed a dilated left ventricle (end-diastolic diameter = 6.2 cm) with a severely reduced left ventricular ejection fraction of 15-20%. The patient's heart rate was 100 beats/minute, MAP was 55 mmHg, and oxygen saturation was 95% on two liters of oxygen. Dobutamine was started as inotrope support for hemodynamic instability.

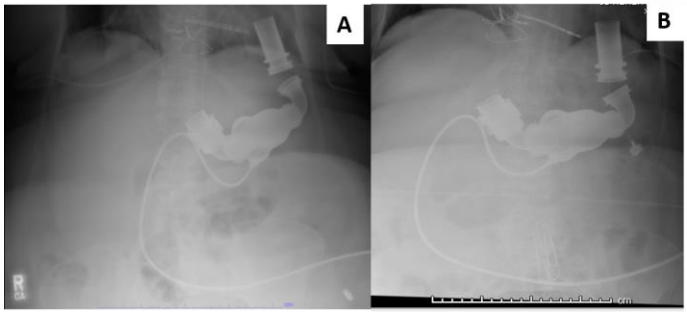

Figure 2. *A) X-ray obtained following HeartMate II implantation. B) X-ray obtained seven years later showed the inflow cannula's malposition.*

The next day, the patient underwent a successful exchange to HeartMate 3 (HM3, Abbott Laboratories). Initially, the heart and LVAD were dissected. Intraoperative transesophageal echocardiogram displayed the cannula directed towards the septum with dense material around it (Figure 3). The inflow cannula had started to core into the septum; thus, it was removed with difficulty. The inflow cannula

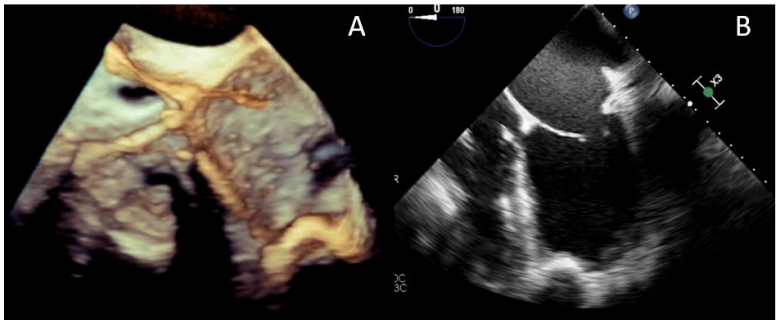

Figure 3. *Transesophageal echocardiography images of the inflow cannula in the A) 3D and B) 2D views show the cannula adhered to the septum.*

strongly adhered to the myocardium. The apical cuff for the HMII was used and modified by cut down. The HM3 inflow cannula was placed into the HMII cuff, and a tie band was used. The outflow graft for the HMII was cut about 10 cm from the aorta, and an end-to-end graft anastomosis was done with the HM3 outflow graft. The direction of the cannula was repositioned in two ways. The HMII was housed in the preperitoneal fat. The HM3 was placed intrapericardially, which changed the position of the inflow cannula. Also, a stitch was placed through the Gortex membrane that was placed around the proximal outflow graft and tacked to the caudal portion of the pericardium; this allowed for a change in the angulation of the inflow graft.

A thick piece of tissue obstructed the inflow cannula of the HMII. The gross specimen showed a single circular brown-black piece of tissue covering the inlet completely and measuring 1.8 x 1.9 x 0.7 cm. Upon disassembly of the pump, the textured, blood-contacting surface of the inlet tube revealed a red tissue-like formation that occluded most of the blood pathway (Figure 4).

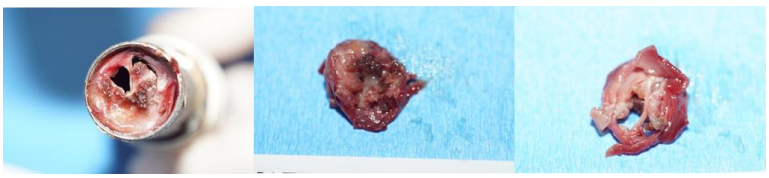

Figure 4. *The deposit in the proximal lumen of the inlet tube was sent to histopathology. It was identified as a plug of pre-existing fibrous and cardiac muscle tissue. The two lumens seen on the membranous structure were made upon explant of the left ventricular assist device.*

The deposition was well adhered to the textured blood-contacting surface and likely developed over an undetermined amount of time while the device was operating. The deposition would have obstructed flow through the device and likely contributed to the low-flow alarms captured in the log file. Histopathology of this

material showed mainly fibrous tissue with myocardial cells and no thrombotic masses (Figure 5). The engineers concluded that this was a plug of pre-existing fibrous and cardiac muscle tissue with only a small amount of fibrin at the perimeter, essentially plugging the inlet tube and impeding the flow.

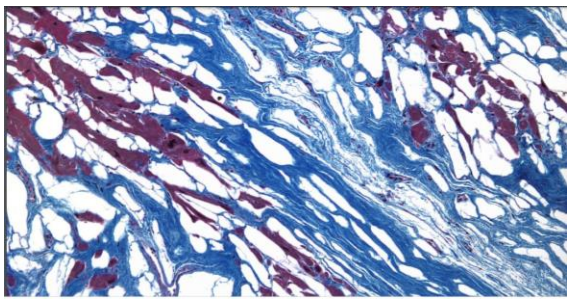

Figure 5. *Histopathology of the membrane shows cardiomyocytes and fibrous tissue.*

Discussion

Pump thrombosis is a frequent complication of HMII, with a reported incidence of >10%.[1] Pump obstruction from compression of the outflow graft of the LVAD by thrombotic masses accumulating within the protective Goretex wrap is a less common complication and has been well-studied.[2-4] Rarely, other causes of the pump obstruction (i.e., seroma or fungal infection) are reported.[5-8] A principally new type of pump obstruction of the HMII inflow cannula by a membranous structure with no evidence of thrombosis is described in this case report.

To our knowledge, this is the first case reported in the literature. The histopathology showed mature tissue of mainly fibrous material and myocardial cells. A thrombus was not seen. The etiology of such a finding is unclear. However, it is well known that prosthetic valves develop pannus over time, which can impede blood flow and obstruct the valve. Pannus formation is a chronic process usually seen on the ventricular side of the valve. It is thought to be a neointimal proliferation from tissue migration, the proliferation of myofibroblasts, and the extracellular matrix with vascular components.[9] We propose that what we observed in our patient is similar to the tissue growth seen around the inlet of the LVAD. The other possibility is that the inflow cannula could have imbibed into the septum; hence the cannula was stuck and had to be forcefully separated. This could also explain the presence of muscle tissue in histopathology.

Our patient had the HMII implanted for nearly seven years, and therapeutic anticoagulation was maintained throughout this period. Interestingly, the serum lactate dehydrogenase level was only mildly elevated (~400-500 IU), and there were no spikes or gradual increases before the admission for low-flow alarms and

pump stoppages. It is known that inflow cannula angulation is a risk factor for pump thrombosis.[4] In our patient, the inlet cannula position had changed over time with angulation towards the septum. This likely occurred because of a significant and intentional weight loss (135 kg to 115 kg) as the patient tried to gain eligibility for cardiac transplantation. Intraoperatively, the inlet cannula had to be forcefully separated from the ventricular septum. It is possible that the septal tissue proximation of the inflow cannula triggered tissue ingrowth. In prosthetic valves, shear stress is also known to contribute to pannus formation.[10] Alteration in the inflow angulation could have caused blood flow turbulence around the inflow cannula and resulted in tissue growth in this case.

Traditionally, pump thrombosis is treated with an intensification of the anticoagulation regimen.[11] In some cases, there are reports of using a fibrinolytic as well.[12] Since the definitive treatment of such tissue overgrowth is only operative, using an antithrombotic or fibrinolytic agent will not have a therapeutic yield.

In conclusion, we report a case of complete inflow cannula obstruction of an HMII from membranous tissue growth. The pannus-like formation can be seen in the inflow cannula and likely caused the device to malfunction. Alternatively, the obstruction could have occurred if the inflow was imbibed into the septum. We believe that it is important to be aware of such a phenomenon as it can potentially be seen with HM3 or any other assist device with an inflow cannula.

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