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Peer-Reviewed Case Report

Acute Pump Thrombosis within One Hour of a HeartMate 3 Implantation: Case Report

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Abstract

A 39-year-old male with a history of non-ischemic cardiomyopathy underwent a HeartMate 3 (Abbott Laboratories) implantation. Within an hour of implantation, low flow alarms were noted before exiting the operating room. The alarm was secondary to acute pump thrombosis. This is the first reported case of thrombosis immediately post-implant.

Background

Left ventricular assist device (LVAD) thrombus can develop *de novo* in the VAD or could arise elsewhere, such as in left ventricle (LV) and then extend to the VAD.[1] In general, pump thrombosis is due to pump-related, patient-related or management-related factors. Pump-related factors include interactions at the interface between blood flow and the components of the pump. The impeller mechanism involves interactions, which include surface interfaces, shear stress, and stasis. For the inflow portion, thrombus formation at the cannulation site and cannula malposition can lead to pump thrombosis. For the outflow portion, factors include outflow graft impingement by the bend relief, graft kinking or twisting, and obstruction of the outflow anastomosis.[2] Patient-related factors include pre-existing LV thrombus, atrial fibrillation, infection, right-sided heart failure, hypercoagulable state or non-compliance. Management-related factors include sub-therapeutic international normalized ratio, low flow due to low-speed setting or sub-optimal hypertension management.[2]

Case Report

A 39-year-old male with multiple comorbidities, including non-ischemic cardiomyopathy and heart failure with reduced left ventricular ejection fraction (10-15%), was referred to our center for advanced heart failure therapies. He experienced several months of progressive decline in functional status and was found to be in cardiogenic shock. After a multidisciplinary team discussion, the patient was deemed a suitable candidate for an LVAD implantation as a destination therapy. His INTERMACS profile was 2 at the time of the implantation. The pre-implant complete blood count, therapeutic activated partial thromboplastin time, and fibrinogen were all within normal ranges. Preoperatively, the patient was on continuous intravenous heparin, which was stopped four hours before surgery.

A thoracotomy and hemi-sternotomy approach were utilized. The LV end-diastolic dimension via intra-operative transesophageal echocardiography (TEE) was 6.8 cm. The coring device was used to take out the apical core. There was an adherent apical LV clot and some minor trabeculations, which were thoroughly removed. As the heart was free of debris and any obstruction, the HeartMate 3 (HM3) was seated onto the sewing ring, and the locking mechanism was closed. 35,000 units of heparin were administered intraoperatively. Heparin was reversed with standard protamine at the conclusion of cardiopulmonary bypass. No blood products or pro-coagulants were administered. The patient's HM3 implantation was successful. At the time of implant, the LVAD speed was 5200 rpm, flow was 3.8 L/min, power was 3.5 Watts, and the pulsatility index was 4.4.

Approximately 60 minutes after the end of the surgery, low flow alarms started without any significant change in speed and power. Intraoperative TEE was still in place and revealed no pericardial effusion or tamponade. The HM3 cannula position was appropriate with no septal contact (Figure 1). Maneuvers, including the increase of ramp speed and dose of inotropes, had no effect. The patient's right ventricular function was adequate, but his aortic valve could not be closed.

After sternal reopening, the absence of tamponade was confirmed, and there was no kinking of the bend relief or the outflow graft. Given the acute change in pump flow combined with no power changes with steady RPMs, there was a strong suspicion of significant inlet cannula obstruction. The pump was unlocked from the sewing ring and inspected. The inflow cannula had a fresh, nearly occlusive thrombus present (Figure 2). The outflow side of the pump was similarly involved. The thrombus appeared to involve all of the sintered surfaces of the pump.

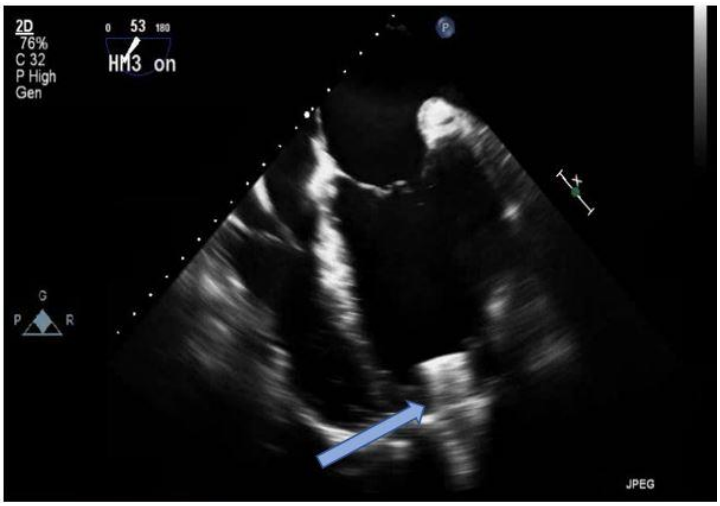

Figure 1: *The arrow shows the HeartMate 3 (Abbott Laboratories) cannula position during intra-operative transesophogeal echocardiogram.*

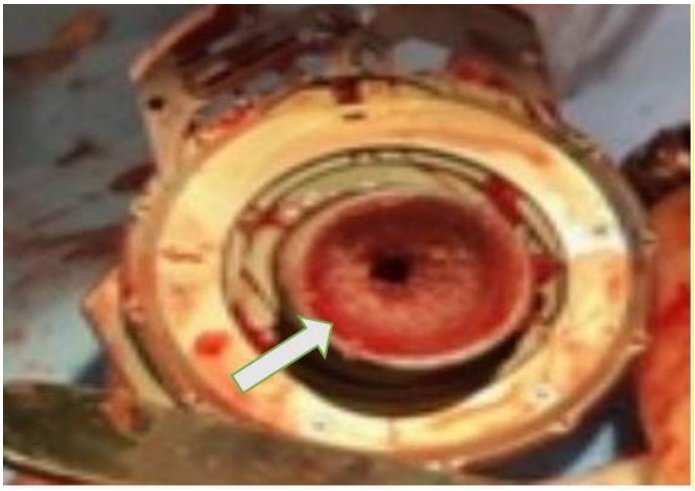

Figure 2: *The arrow indicates the nearly occlusive thrombus in the HeartMate 3 left ventricular assist device system (Abbott Laboratories).*

A pump exchange without reversal of heparin was performed. The thrombosed pump was sent to the manufacturer for inspection, and no mechanical defects were found. Postoperatively, the patient had a negative Factor V Leiden screen, and he tested negative for hexagonal lupus anticoagulant. Normal MA kaolin, R

kaolin, RK kaolin, K kaolin, lysis 30 and angle kaolin levels were documented two days after surgery. The patient was discharged home on Postoperative Day 13.

Discussion

The HM3 uses a fully levitated, self-centering rotor with large blood flow pathways (Maglev Flow Technology) that is more hemocompatible than the prior axial flow technology that was used in the HeartMate II (HMII) device (Abbott Laboratories). In the MOMENTUM 3 trial, there was a greater than 90% relative risk reduction of suspected/confirmed pump thrombosis when compared to HMII.[3] Moreover, in the HM3 CE Mark study, there were no cases of pump thrombosis after two years of follow up.[4] The earliest reported cases of HM3 pump thrombosis were at Postoperative Day 3.[5,6] In one patient, apical calcium was noted intra-operatively, and the proposed mechanism was the ingestion of some calcium from the apex leading to misplacement of the rotor and subsequent thrombosis.[5] In the other case, the left ventricular cavity size decreased from 5.9 cm pre- operatively to 4.0 cm post-operatively, causing septal contact with the inflow cannula and eventually pump thrombosis.[6]

Our patient had pump thrombosis within the first hour of implantation. There was no apical calcium noticed before LVAD implantation and no septal contact during intraoperative TEE. A possible etiology in this case could be a previous adherent apical LV clot site that might have acted as a nidus for the development of a thrombosis leading to acute pump thrombosis. Histological analysis confirmed the presence of platelet and red blood cell deposition with very few fibrin strands, suggestive of acute pump thrombus (Figure 3).

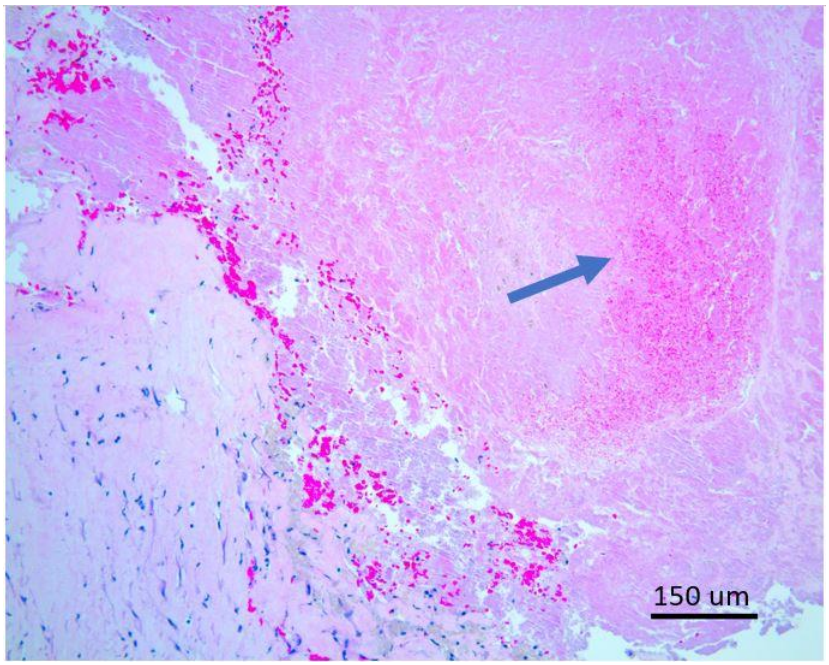

Figure 3: *The arrow highlights the acute thrombus on histological analysis.*

This case represents a unique instance of very early pump thrombosis and should be kept in mind if new, low flow alarms are encountered—even immediately post-implant

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