Acute Pump Thrombosis in the Early Postoperative Period After HeartMate 3 Implantation

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There are no reports of acute pump thrombosis in the latest, continuous flow left ventricular assist devices type HeartMate 3, other than thrombus ingestion. We present a case of early thrombosis of the pump and outflow graft, necessitating acute pump and outflow graft replacement. A combination of low-flow episodes and subtherapeutic levels of anticoagulation was the most likely cause. *ASAIO Journal* 2019; 65:e72–e74.

Key Words: heart assist devices, heart failure, anticoagulation

Left ventricular assist device support may infrequently be complicated by acute thrombosis. So far, there have been no reports of reoperation for pump thrombosis in patients with a HeartMate 3 (HM3).[1,2] We present a case of pump and outflow graft thrombosis in a patient with a HM3 device in the very early postoperative phase, needing urgent replacement of the pump and outflow graft.

Case Description

A 44-year-old man with end-stage heart failure because of dilated cardiomyopathy (left ventricle [LV] end diastolic diameter 62 mm, left atrium 43 mm, no signs of LV thrombus) underwent implantation of a HM3 device as a bridge to transplantation. Preoperatively, the patient was on continuous heparin intravenously, with therapeutic activated partial thromboplastin time (APTT), which was ceased 4 hours before surgery. He did not use platelet inhibitors, platelet count was 202×10^9/l, prothrombin time international normalized ratio 1.4. On postoperative day 1, transoesophageal echocardiography (TEE) showed no signs of right ventricular failure, normal turbulence at in- and outflow cannulae, and no spontaneous echo contrast in the LV. However, a structure was seen in the left posterior aortic sinus, attached to the left coronary cusp (**Figure 1A**, and Supplementary Movie 1 and 2 [see Video 1, Supplemental Digital Content, http://links.lww.com/ASAIO/A370 and Video 2, http://links.lww.com/ASAIO/A371]). The differential diagnosis was an evolving thrombus, because of stasis and absence of aortic valve opening, for which conservative management with follow-up imaging was decided. The patient was extubated late on postoperative day 1.

Heparin was started on postoperative day 2, 10,000 units/day by continuous infusion. On day 3, the patient was discharged from the ICU with a heparin ratio of 1.2. However, later that day, there was a low-flow episode that resolved with a 500 ml fluid bolus. Echocardiographically, a tamponade was suspected. Heparin was ceased, and the patient went for urgent rethoracotomy. Intraoperative TEE showed a normal aortic valve and aortic sinus. Some pericardial effusion was evacuated, although a tamponade could not be confirmed and in retrospect the suspected large pericardial effusion was a pleural effusion (600 ml). There were no signs of thrombus in the outflow graft. It was decided not to restart the heparin until the next day.

However, in the night, the patient deteriorated, cold and perspirating, with clinical features of cardiogenic shock and acute respiratory failure. The display of the HM3 console showed an increase in pump power (8W), high calculated flow (8.5 L/min) and normal pulsatility index (2.2). Lactate dehydrogenase level was increased to 411 u/l. The patient was reintubated, and urgent TEE and CT scan were performed. TEE showed no flow in both outflow and inflow graft (**Figure 1**, B and C). The CT scan showed an occluded outflow graft, but no signs of kinking or external compression (**Figure 1**, D and E). Log file interrogation showed rotor imbalance and increase in noise in the previous 18 hours (**Figure 2**). The patient underwent urgent HM3 pump and outflow graft replacement, using the original left apical button. Macroscopic inspection revealed extensive pump and outflow graft occlusion with thrombotic material (**Figure 1**, F and G). Postoperatively, HM3 flows were stable and no major new events occurred. Heparin was restarted on postoperative day 1, acetylsalicylic acid 80 mg/day was started on postoperative day 5, and the patient was discharged on oral vitamin K antagonist (target INR 2–3) and acetylsalicylic acid. Hematologic work-up did not reveal any signs of antiphospholipid syndrome. The patient recovered prosperously and is currently New York Heart Association I after 12 months of follow-up.

Discussion

We present a case of early failure of a HM3 device, due to thrombosis in the pump and outflow graft. Device malfunctioning has been described before in case reports, but in these cases this was caused by bleeding within the bend relief compressing the outflow graft, kinking, or thrombosis in the pump because of suspected embolus ingestion.[1,3–5] In our case, both the pump and the outflow graft were fully thrombosed, without

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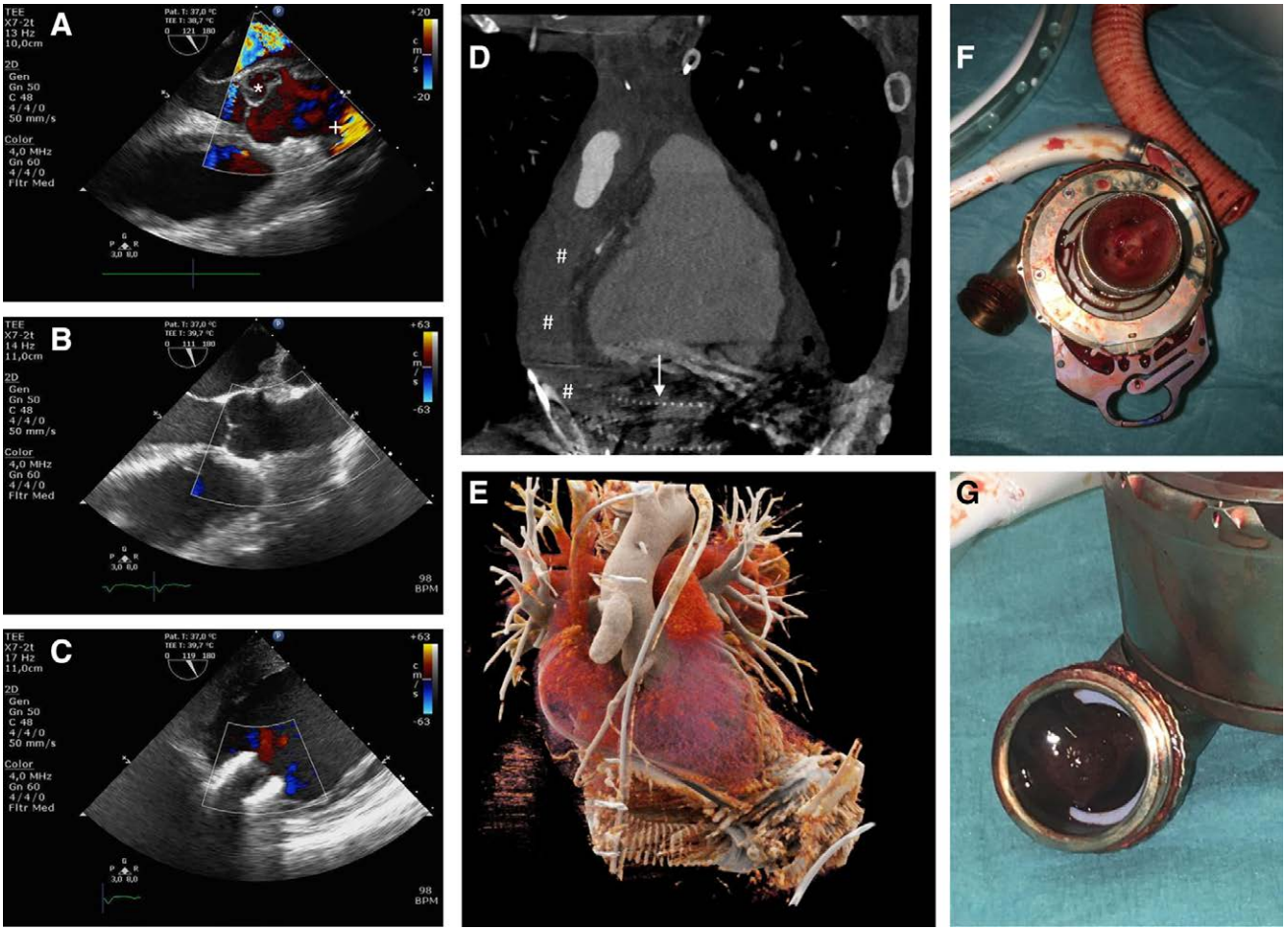

Figure 1. Images of the functioning and thrombosed LVAD. A: TEE on postoperative day 1, note the structure attached to the aortic valve (*), and the colour jet of the inflow of the HeartMate (HM) 3 flow in the ascending aorta (+). The aortic valve remained closed during the whole heart cycle. B: TEE on postoperative day 4, showing no colour jet of HM3 flow in the ascending aorta. Note that there is no sign of the structure seen in (A) attached to the aortic valve. The echocardiogram showed full opening and closing of the aortic valve. C: TEE on postoperative day 4, showing no flow in the direction of the HM3 inflow canula. D and E: CT images on postoperative day 4, showing the occluded ouflow graft (#), with contrast only in the distal part of the outflow graft. Arrow: Bend relief of the proximal part of the ouflow graft, coming from the HM3. E: Three-dimensional, volume rendered reconstruction of (D). With no contrast, the outflow graft itself is not visible as a seperate structure. F and G: Photograph of the explanted HM3 pump, the outflow graft has been removed, showing thrombus in the inflow side and pump of the heartmate (F) as well as in the outflow side of the HM3 (G). CT, computed tomography; TEE, transoesophageal echocardiography.

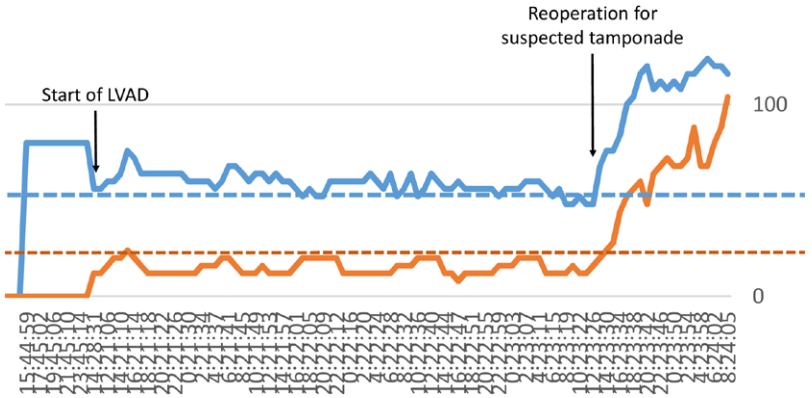

Figure 2. Log files of HM3 from the start of therapy, until log file interrogation was done for suspected pump occlusion on postoperative day 4. The red line shows motor noise (μm), the blue line shows motor displacement (μm). Note the rotor noise and displacement start to increase from the moment of low-flow alarms. HM, HeartMate.

any signs of kinking or external compression, both on CT and intraoperatively. The HM3 was sent to the manufacturer for further analysis. No mechanical defects could be found.

Most likely, thrombosis occurred because of a combination of not yet therapeutic anticoagulation and low-flow episodes. The HM3 manufacturer advises to start heparin after 12–24 hours and start aspirin on postoperative day 2–3. However, following our local anticoagulation policy, continuous heparin is started in low dose on postoperative day 1–2 (day 2 in this patient), and aspirin on postoperative day 4–6. This regime was chosen to reduce the high rate of early rethoracotomy we experienced.[6] Because of this conservative anticoagulation approach, and (retrospectively unjustified) rethoracotomy, APTT levels had not yet reached target values by the time pump thrombosis occurred. It seems unlikely that the structure seen in the aortic sinus had migrated in the inflow cannula, because in that scenario the structure should have crossed an open aortic valve against normal flow direction.

In conclusion, this case report illustrates that thrombosis of a HM3 device can occur. In absence of a clear cause, anticoagulation still seems mandatory in the early phase, especially when there are low-flow episodes.

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