Early inflow pannus development requiring left ventricular assist device exchange: More to come?

Alexander Ghannam, MD,[a] Larissa Check, MD,[b] Ecem Akdogan, MD,[b] Jennifer Hajj, RN,[b] Brian Houston, MD,[b] Vishal Rao, MD,[b] Ryan Tedford, MD,[b] and Arman Kilic, MD,[a] Charleston, SC

From the [a]Division of Cardiothoracic Surgery, Department of Surgery, and [b]Division of Cardiology, Department of Medicine, Medical University of South Carolina, Charleston, SC.

Informed Consent: Publication of this case report was done so under the consent of the involved patient.

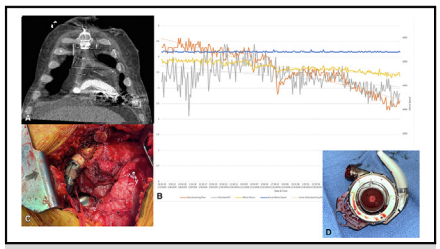
Received for publication Feb 21, 2024; revisions received March 21, 2024; accepted for publication March 22, 2024; available ahead of print April 16, 2024.

Address for reprints: Arman Kilic, MD, Division of Cardiothoracic Surgery, Department of Surgery, Medical University of South Carolina, 30 Courtenay Dr, MSC 295, Suite BM279, Charleston, SC 29425 (E-mail: kilica@musc.edu).

JTCVS Techniques 2024;25:97-9

2666-2507

https://doi.org/10.1016/j.xjtc.2024.03.024



A, CT scan. B, Log file graph X (days), Y (L/min). C, Outflow. D, Inflow pannus.

CENTRAL MESSAGE

The log file data and clinical presentation can help in future similar cases with pinpointing the etiology of pump failure and devising a treatment plan.

HeartMate 3 (HM3) (Abbott Laboratories) is a left ventricular assist device (LVAD) implanted in patients with heart failure. HM3 is the only durable LVAD on the market because of its excellent hemocompatibility resulting in low incidence of stroke and thromboembolic complications.[1] Retrospective studies demonstrated that a conservative anticoagulation strategy of initiating vitamin K antagonist directly during the postoperative period, rather than bridging with heparin, decreases bleeding events and length of stay.[2] A prospective randomized trial demonstrated that avoiding aspirin after HM3 was associated with a lower rate of bleeding events and no increase in thromboembolic events.[3] We present the case of a patient who experienced HM3 pump failure from pannus formation inside the inflow cannula 2 months postoperatively requiring HM3 to HM3 exchange.

CASE REPORT

The patient is a 67-year-old man with a history of nonischemic cardiomyopathy, atrial fibrillation, diabetes mellitus, chronic kidney disease, deep venous thrombosis (DVT), and pulmonary embolism (PE). He had no evidence of hypercoagulable disorders. He was evaluated by a multidisciplinary committee and was deemed a candidate for HM3 placement rather than transplant due to malignant colon polyps. Publication of this case report was done under the consent of the involved patient; institutional review board approval was not required.

The patient underwent implantation of a HM3 via median sternotomy. The HM3 was set to 5400 rpm with flows of 4.0 to 4.5 L and a normal pulsatility index. Heparin was started postoperatively due his history of atrial fibrillation and DVT/PE. He was bridged to warfarin with an international normalized ratio (INR) goal of 2 or 3 without aspirin. He was discharged to a rehabilitation facility on postoperative day 22 with a normally functioning HM3.

Three weeks after discharge, he presented with low flow alarms and volume overload. Echocardiography demonstrated an appropriately positioned inflow cannula and poor LV unloading. A computed tomography angiography demonstrated no outflow graft obstruction (Figure 1, *A*). He had a stable lactate dehydrogenase level and no power spikes.

He was taken for a right heart catheterization that showed that with increasing to 7400 rpm he continued to have poor LV unloading, 2.5 L of flow, elevated filling pressures, and low cardiac output. Angiography with contrast injected retrograde through the outflow graft and a left ventriculogram revealed no evidence of inflow cannula or outflow graft obstruction. Review of his HM3 log file data demonstrates a progressive decline in flow, stable pump power, and stable pulsatility index over the week before presentation (Figure 1, *B*). Device interrogation by engineers confirmed normal pump function.

The working diagnosis given his clinical picture and log files was that he had a pre-rotor inflow occlusion problem. He was taken to the operating room for a HM3 to HM3 exchange via left thoracotomy. The HM3 was exposed and the bend relief was removed from the device to confirm there was no twisting, hematoma, or gelatinous material causing external compression (Figure 1, *C*). The right femoral artery

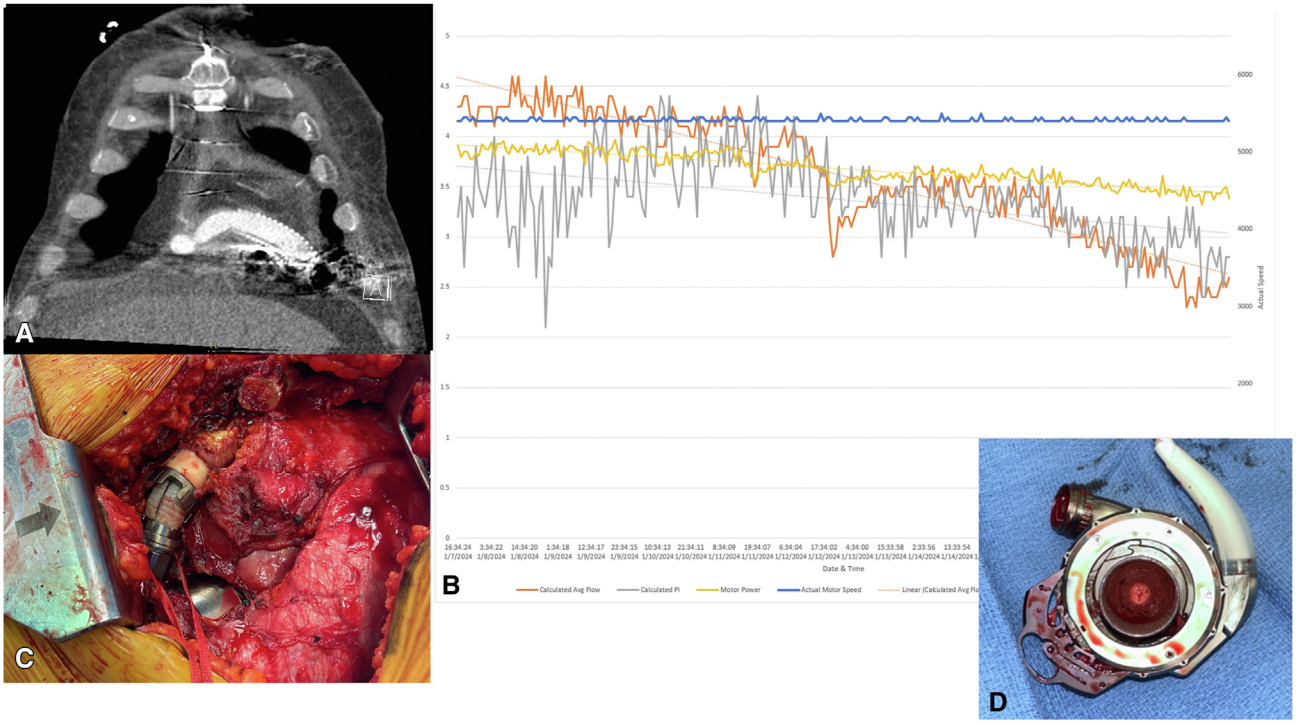


FIGURE 1. A, Computed tomography scan. B, Log file graph X (days), Y (L/minute). C, Outflow. D, Inflow pannus.

and vein were cannulated for cardiopulmonary bypass. The HM3 was removed from the LV apex. The LV was found to be free of trabeculations or tissue that could cause inflow obstruction. The HM3 inflow cannula was nearly occluded at the most narrow portion of the inflow by a white pannus (Figure 1, *D*). A new HM3 device was placed in the existing sewing ring. He was weaned from bypass without difficulty at 5400 rpm with flows >4 L and normal pump parameters. He was initiated on intravenous heparin and aspirin postoperatively and discharged home on day 11 with an INR goal of 2 or 3.

DISCUSSION

There are a few case reports of patients requiring HM3 to HM3 exchange in the literature.[4] To our knowledge, this is the first case of HM3 exchange being performed for pannus formation inside the inflow cannula.

Our practice is to initiate warfarin once the patient is downgraded from the intensive care unit and to avoid bridging with intravenous heparin unless the patient has another indication. Our patient had a history of DVT/PE and atrial fibrillation and was bridged. His HM3 had excellent flows through his index hospitalization. In Figure 1, *B*, there is a clear progressive decline in his flow over a period of 1 week before presentation to the hospital, during which time his INR was within an appropriate range. This gradual decline in flow, lack of power spikes, and stable lactate dehydrogenase level supports the theory of a progressive pannus formation rather than a single ingestion event.[4] Further, the lack of power spikes suggests the inflow issue was at the pre-rotor and not rotor level. Although it is difficult to image the proximal outflow graft due to artifact on computed tomography scan, the short time course since original implant made it less likely that there was compression of the outflow graft in this area. We have exchanged LVADs due to compression of the outflow graft, all of which were due to the accumulation of gelatinous material from so-called graft sweat. In the operating room, the outflow graft was found to be aligned correctly and had no debris compressing it under the bend relief (Figure 1, *B*).

The ARIES (Aspirin and Hemocompatibility Events With a Left Ventricular Assist Device in Advanced Heart Failure) trial was a multicenter randomized trial with a median follow up of 14 months of HM3 patients off aspirin that showed no increase in thrombotic events.[3] A biologic framework for the development of pannus on the titanium components of the HM3 has been described; however, it is unclear why it occurred in this patient.[5]

This case highlights an unusual phenomenon in the HM3 era of early inflow occlusion necessitating exchange. This is underscored by the challenge in finding the exact problem because imaging was normal. It is conceivable in these cases that there is hesitation to take the patient to the operating room without a clear cause. We believe our approach of ensuring no outflow problem on imaging, followed by

visual inspection of the area between the bend relief and the outflow graft in the operating room, and finally with commencing with device exchange, was the appropriate course and could serve as a guide in future cases.

Conflict of Interest Statement

Dr Kilic is a speaker and consultant with Abbott, Abiomed, 3ive, and LivaNova. Dr Tedford is a consultant for Abbott and Medtronic and has consulting relationships with Acorai, Aria CV Inc, Acceleron/Merck, Alleviant, Boston Scientific, Cytokinetics, Edwards LifeSciences, Gradient, and United Therapeutics as well as serving on steering committees for Merck, Edwards, and Abbott and as a research advisory board member for Abiomed. Dr Tedford also does hemodynamic core lab work for Merck. Ms Hajj is part of speaker's bureaus for Medtronic and Abbott. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

References

1. Mehra MR, Goldstein DJ, Cleveland JC, et al. Five-year outcomes in patients with fully magnetically levitated vs axial-flow left ventricular assist devices in the MOMENTUM 3 randomized trial. *JAMA*. 2022;328(12):1233-1242. https://doi.org/10.1001/jama.2022.16197
2. Damman K, van den Broek SAJ, Mecozzi G, et al. Conservative initial postoperative anticoagulation strategy after HeartMate 3 left ventricular assist device implantation. *Neth Heart J*. 2022;30(10):466-472. https://doi.org/10.1007/s12471-022-01671-1
3. Mehra MR, Netuka I, Uriel N, et al. Aspirin and hemocompatibility events with a left ventricular assist device in advanced heart failure: the ARIES-HM3 randomized clinical trial. *JAMA*. 2023;330(22):2171-2181. https://doi.org/10.1001/jama.2023.23204
4. Hanke JS, Dogan G, Schmitto JD. A lateral approach to exchange a left ventricular assist device. *J Thorac Cardiovasc Surg*. 2019;158(2):e39-e40. https://doi.org/10.1016/j.jtcvs.2018.12.081
5. He W, Butcher JT, Rowlands GW, Antaki JF. Biological response to sintered titanium in left ventricular assist devices: pseudoneointima, neointima, and pannus. *ASAIO J*. 2023;69(1):1-10. https://doi.org/10.1097/MAT.0000000000001777