Commentary: You are what you eat—Pump thrombosis and device exchange of a HeartMate 3

Amit Iyengar, MD, MS, and Pavan Atluri, MD

The HeartMate 3 (HM3; Abbott Laboratories, Abbott Park, Ill) left ventricular assist device has undoubtedly demonstrated encouraging results regarding biocompatibility. The device has several design points engineered to reduce blood trauma and improve end-organ perfusion, including a fully magnetically levitated centrifugal rotor to reduce points of friction and a fixed artificial pulse.1,2 The Multi-center Study of MagLev Technology in Patients Undergoing MCS Therapy With HeartMate 3 (MOMENTUM 3) trial randomized 366 patients to HeartMate II versus HM3 implantation and showed improvements in primary end point in the HM3 arm, driven by a marked reduction in pump thrombosis.1 Only 2 suspected cases were noted at 2-year follow-up (1.1%), although devices were never explanted and thrombosis never confirmed.3 Extremely few other case reports of pump thrombosis with the HM3 exist.4,5

In this issue of the Journal, Hanke and colleagues6 add to this experience with their report of pump thrombosis and subsequent device exchange. The patient was a 64-year-old man who underwent HM3 implantation through a thoracotomy in 2015. During subsequent follow-up at 4 months, a new left atrial thrombus was incidentally found. At 1-year after implantation, the patient presented with clinical pump thrombosis despite a therapeutic international normalized ratio, and the former atrial thrombus had disappeared on repeat imaging. The device was successfully exchanged without issue. Whether the report represents a de novo pump thrombosis of the HM3 in a patient receiving recommended anticoagulant therapy or rather ingestion of an intracardiac thrombus is unknown, although Hanke and colleagues6 surmise the latter.

A related case report describes a 46-year-old man with pump thrombosis 8 days after off-pump HM3 implantation.7 Copious ventricular clot was noted on reoperation after inspection of the cavity on bypass, and despite maximal clot removal and pump exchange, the patient had recurrent prepump thrombosis and subsequently died. That report is a clearer story of clot ingestion into the left ventricular assist device and associated pump thrombosis but is germane to the current report of Hanke and colleagues.6

Overall, these cases illustrate the concept that, despite improvements in left ventricular assist device design, no amount of biocompatibility can fix an existing thrombus that is ingested into the inflow graft. The ideal management of intracardiac thrombus in patients in stable condition with ventricular assist device support requires further investigation. For routine postoperative management, the manufacturer recommends anticoagulation with warfarin for a target international normalized ratio of 2.0 to 3.0 and antiplatelet therapy with aspirin.1 The utility of increasing this goal, addition or substitution of such alternative agents as direct-acting oral anticoagulants or P2Y12 inhibitors, or catheter-based or surgical intervention in patients with preexistent thrombus is unknown. More aggressive anticoagulation needs to be weighed against a persistent incidence of hemorrhagic stroke and gastrointestinal bleeding, and some discussion regarding potentially reducing anticoagulation levels in patients with HM3s, given the low thrombosis events, as a means of further reducing bleeding complications. As a preemptive therapy, many surgeons advocate the routine ligation of the left atrial appendage at time of device implantation, whereas others reserve it for those with comorbid atrial arrhythmias. Regardless, diligent surveillance for this complication and awareness of the
possibility of thrombus ingestion into the device must be considered, as illustrated by these cases. After all, we are what we eat.

References