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CASE REPORT: CLINICAL CASE SERIES

Aortic Root Thromboembolism and Associated Acute Myocardial Infarction in Patients With Contemporary Durable LVADs

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ABSTRACT

Patients with HeartMate 3 left ventricular assist devices may develop aortic root thrombus, yet its prevalence and associated risks are unknown. We present 2 patients who developed aortic root thromboembolism and acute coronary occlusions. We additionally present heart transplantation as viable treatment for thromboembolic disease and refractory right ventricular failure. (JACC Case Rep 2024;29:102441) Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Acute myocardial infarction (AMI) in the presence of an aortic root thrombus (ART) is a rare thrombotic complication among patients with HeartMate 2 left ventricular assist devices (LVADs)[1] and has only been reported once in patients with HeartMate 3 (HM3) LVADs.[2] Despite reduced thrombotic risk in HM3 compared with HM2 LVAD,[3] the prevalence of postoperative ART and complications remain unclear. We present 2 HM3-supported patients who developed ART complicated by embolic coronary artery occlusion, AMI, and right ventricular (RV) failure.

LEARNING OBJECTIVES

- To understand complications of aortic root thrombus in patients with LVADs.
- To describe diagnostic challenges in acute myocardial infarction among patients with LVADs.
- To review treatment strategies for acute myocardial infarction in patients with LVADs.

CASE 1

A 65-year-old man with nonischemic dilated cardiomyopathy and end-stage heart failure underwent HM3 LVAD implantation as a bridge to transplantation. He presented on postoperative day 105 after 2 syncopal episodes and persistent low-flow alarms within 20 minutes of emergency room arrival. Presenting clinical data are shown in **Table 1** and **Figure 1A**. Transthoracic echocardiography (TTE)

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ABBREVIATIONS AND ACRONYMS

AMI = acute myocardial infarction

ART = aortic root thrombus

HM2 = HeartMate 2

HM3 = HeartMate 3

LVAD = left ventricular assist device

RCA = right coronary artery

RV = right ventricle

TTE = transthoracic echocardiogram

showed new-onset severe RV dysfunction and immobile aortic valve (AV). Thrombus was visualized anteriorly in the sinotubular junction (**Figure 1B**, Video 1). The patient underwent chest computed tomographic angiography which revealed a new focal filling defect in the right sinus of Valsalva with associated right coronary artery (RCA) occlusion (**Figure 1C**). The patient was considered a high risk for coronary intervention and was treated conservatively with intravenous heparin and dobutamine given severe RV failure. Unfortunately, attempts to wean dobutamine led to recurrent syncope. Invasive hemodynamics showed severe derangements in filling pressures (right atrial/pulmonary capillary wedge ratio 3:1, cardiac index 1.86) despite inotropic support. Previously, he was not suitable for transplantation due to active inhaled cannabis use before requiring urgent LVAD. However, the patient had since abstained post-LVAD and was determined suitable for urgent transplant listing as he was high risk for imminent clinical deterioration and demise either from extension/embolization of ART, cannula-septum interaction from underfilled left ventricle, or insufficient hemodynamic support due to severe RV failure. He underwent LVAD explantation and orthotopic heart transplantation 5 days later. No residual ART was visualized either in proximal outflow tract or native ascending aortic arch. His functional capacity clinically improved following transplantation.

CASE 2

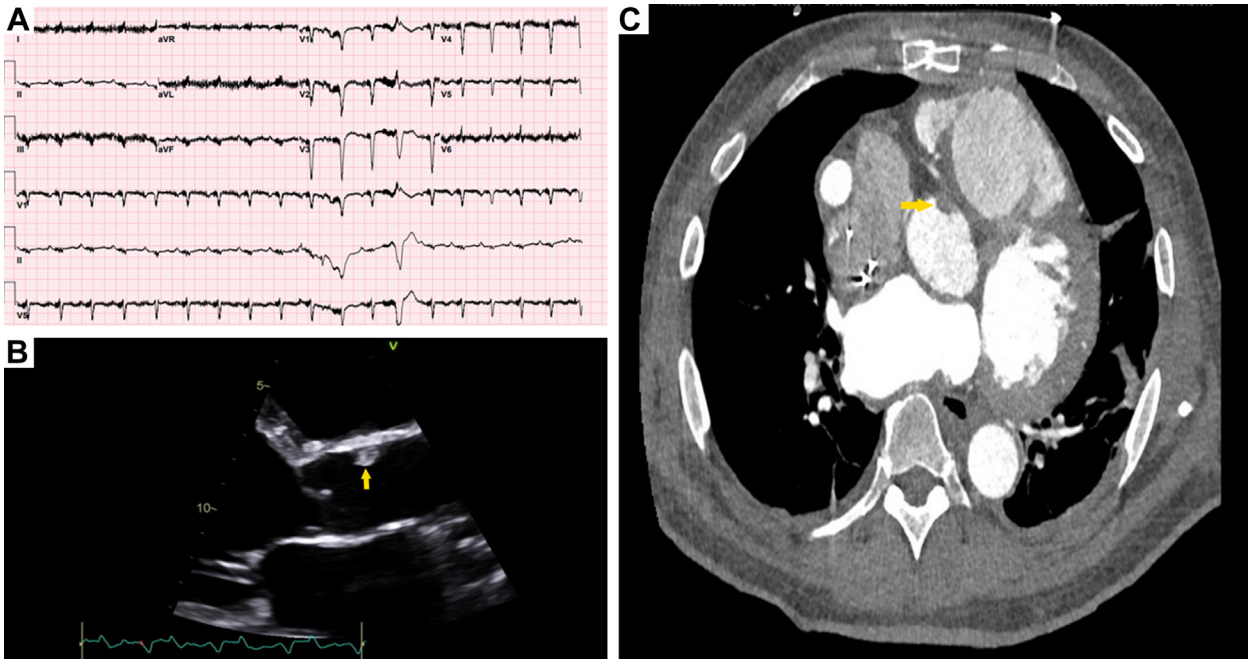
A 72-year-old man presented with ischemic cardiomyopathy and end-stage heart failure. He underwent HM3 LVAD implantation as destination therapy with Park's stitch for aortic insufficiency. He was not a transplant candidate due to advanced age and chronic lung disease. His postoperative course was complicated by mediastinal hematoma and multiple occurrences of hemodynamically significant gastrointestinal bleeding requiring cessation of antiplatelets/anticoagulation. He presented on postoperative day 148 with ventricular tachycardia requiring defibrillation and chest pain. Initial clinical and laboratory data are shown in **Table 1** and

TABLE 1 Presenting Clinical Characteristics and Course Following Diagnosis of Aortic Root Thrombus and Coronary Artery Occlusion

	Case 1	Case 2
Presenting symptoms	Recurrent syncope and low-flow alarms	ICD shock and chest pain
Past medical history	Hypertension, ischemic stroke, and seizure disorder	Chronic kidney disease and chronic obstructive lung disease
History of ischemic cardiomyopathy	No	Yes
Initial vital signs	Mean arterial pressure 78 mm Hg, heart rate 101 beats/min, respiratory rate 22 breaths/min, afebrile.	Mean arterial pressure 82 mm Hg, heart rate 86 beats/min, respiratory rate 18 breaths/min, afebrile.
Troponin peak at time of presentation	>50,000 ng/L	>50.000 ng/L
INR	2.2	1.2
Electrocardiogram findings	Q-wave formations from V_1 through V_3; similar to postimplantation electrocardiogram	ST-segment depressions in inferior leads and V_4 through V_6
Echocardiogram findings	Small thrombus in sinotubular junction, immobile aortic valve, new onset RV dysfunction, and severe tricuspid regurgitation	New onset RV dysfunction, immobile aortic valve (Park's stitch)
LVAD settings at baseline	Pulsatility index 8 to 9.4 Power of 3.3 W Flow under 2.5 L/min Speed at 5,000 rpm Multiple low-flow episodes	Pulsatility index 2.2 Power of 3.6 W Flow of 3.5 L/min Speed at 5,200 rpm Multiple low-flow episodes
Angiography	Computed tomography showed focal filling defect in the right sinus of Valsalva and associated proximal right coronary artery occlusion.	Invasive angiography showed large thrombus in aortic root extending into right coronary artery.
Invasive hemodynamics	RA/PCWP 3:1 Thermodilution cardiac index 1.86 PVR 2.2 Wood units	Not available
Initial treatment	Intravenous dobutamine and diuretics	Intravenous heparin
Long-term outcome	Orthotopic cardiac transplantation due to refractory RV failure.	Resumed anticoagulation with improving symptomatic lightheadedness.

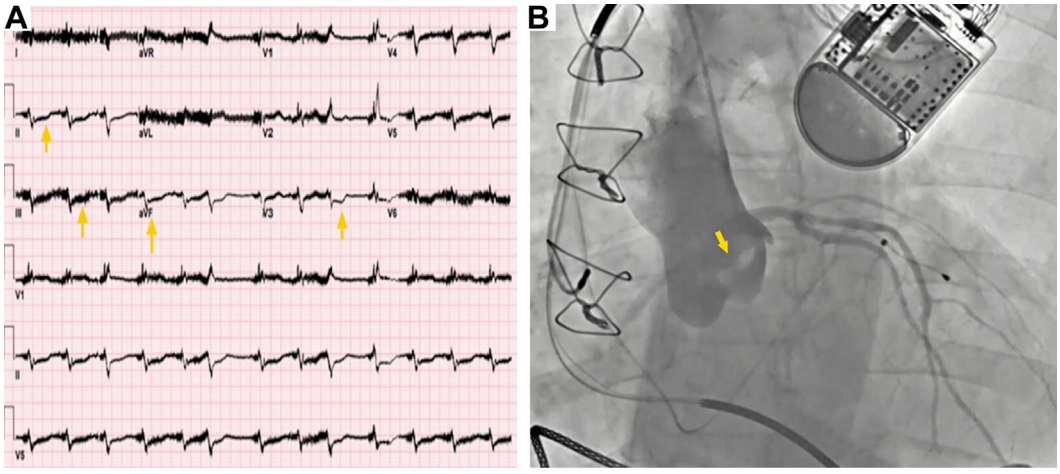
ICD = implantable cardioverter-defibrillator; INR = international normalized ratio; LVAD = left ventricular assist device; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; RA = right atrium; RV = right ventricle.

FIGURE 1 **Patient With HM3 LVAD Presenting With Syncope and Low-Flow Alarms**



(A) Electrocardiogram showing Q waves in the anterior leads. (B) Transthoracic echocardiography in parasternal long-axis view showing thrombus attached to the aortic root near right coronary artery ostium (yellow arrow). (C) Coronary computed tomography angiography reveals a filling defect in the right sinus of Valsalva extending into the proximal right coronary artery (yellow arrow). HM3 LVAD = HeartMate 3 left ventricular assist device.

FIGURE 2 **Patient With HM3 LVAD Presenting With Ventricular Arrhythmias and Chest Pain**



(A) Electrocardiogram showing ST-segment depression (yellow arrow) in inferior leads and V_3 through V_6. (B) Invasive aortogram reveals large thrombus in the aortic root (yellow arrow). HM3 LVAD = HeartMate 3 left ventricular assist device.

Figure 2A. Echocardiography showed new-onset RV failure, immobile AV, and mild aortic insufficiency. In the setting of AMI, the patient was referred for emergent aortic angiography which showed a large ART with mobile extension into the RCA (**Figure 2B**, Video 2). Because of multiple prior bleeding episodes, the patient was determined to be at high risk for percutaneous or surgical thrombectomy or coronary intervention. He was treated conservatively with intravenous heparin.

Following initial presentation, the patient remained stable and his RV failure was medically managed with TTE surveillance. He was discharged home on warfarin.

DISCUSSION

ART-associated AMI has been reported previously in HM2 patients despite therapeutic anticoagulation.[1,4,5] In contrast, HM3 has shown a favorable thrombotic profile compared with the HM2.[3] This improvement has been attributed to its broad blood flow passages and lack of mechanical bearings, resulting in a frictionless environment. Additionally, it is programmed to enable swift adjustments in rotor speed, allowing for the creation of an inherent artificial pulse, which operates independently of the natural heartbeat, diminishing stasis within the pump.[3] The present case series shows the serious complications of aortic root thromboembolisms involving RCA occlusions in 2 patients who received HM3 that resulted in AMI and RV failure.

Despite left-sided support with an LVAD, AMI can still pose a life-threatening risk if it affects the RV territory or causes significant arrhythmias. Additionally, electrocardiogram interpretation in patients with LVADs can present diagnostic challenges due to LVAD speed-related artifacts or presence of conduction disturbances or pacemaker-related ventricular rhythms. Neither of the presented patients showed ST-segment elevation; however, both patients had marked elevations in high-sensitivity troponin assays which supported diagnosis of AMI. This complication occurred in 2 different clinical conditions: 1) a patient with therapeutic anticoagulation and previous AV mobility; and 2) a patient with Park's stitch AV closure and cessation of anticoagulation due to adverse bleeding events. As such, providers must have a high index of suspicion for ART and AMI despite anticoagulation strategies and absence of presenting anginal symptoms among patients who present supported with HM3.

Extended AV closure in LVAD patients can result in commissural fusion and aortic insufficiency.[6,7] Furthermore, turbulent backflow originating from the outflow cannula establishes a low-flow environment conducive to thrombus formation.[6] In both presented cases, AV was observed to be closed at the time of acute presentation. However, the AV in case 1 was observed opening every beat on serial postoperative TTEs. The finding of AV closure upon acute presentation was likely due to AMI resulting in severe biventricular dysfunction. Surprisingly, ART occurred despite therapeutic anticoagulation in case 1. Despite anatomic and medical differences, both patients exhibited RV failure following coronary occlusion. Incident embolic events affecting the RCA or left circumflex artery in a left-dominant system may contribute to RV dysfunction, significantly decreasing LVAD preload, resulting in poor forward flow that may have precipitated cardiogenic shock in the present cases. We hypothesize that the subsequent reduced left ventricular preload secondary to RV failure may have resulted in decreased overall LVAD flow and resulted in higher pulsatility index. The severity of RV failure was worse in case 1, likely contributing to a higher pulsatility index than observed in case 2. Among patients observed to have chronically immobile AV, LVAD speed may be reduced to permit intermittent AV opening, potentially as a preventive measure against aortic root thrombus formation.[4] However, prevalence of aortic root thrombosis across LVAD speeds remains uncertain.

There exists no definitive evidence for raising therapeutic anticoagulation thresholds among patients with LVAD who develop incident ART. Standard anticoagulation for the HM3 is an international normalized ratio (INR) of 2 to 3,[3] and a recent multicenter randomized clinical trial determined that warfarin use alone compared with aspirin plus warfarin in patients supported with HM3 LVADs resulted in significantly lower major nonsurgical bleeding events and no significant increase in thromboembolic risk.[8] ART embolization to the left circumflex artery on postoperative day 9 after HM3 implantation has been reported despite therapeutic anticoagulation.[2] However, the investigators described that aspiration thrombectomy and raising the anticoagulation goal to INR 3 to 3.5 were ineffective in preventing early mortality on postoperative day 60.[2] Additional treatment options for ART may include systemic anticoagulation, percutaneous or surgical thrombectomy, or percutaneous coronary

intervention with stent in the event of thromboembolism. In cases of refractory RV failure following thromboembolism and coronary occlusion, emergent orthotopic heart transplantation may be considered and is feasible. However, invasive treatment options must be considered on an individual basis as they may present or worsen an increased risk of bleeding or embolic cardiovascular events. The presence of LVADs may also present technical challenges for percutaneous interventions and associated long-term medical therapies.

CONCLUSIONS

Acute aortic root thromboembolism can occur in patients supported with HM3 with or without therapeutic anticoagulation. Coronary artery embolisms can lead to AMI, RV failure, and cardiogenic shock. Further characterization of the prevalence and associated complications of post-LVAD ART are needed.

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KEY WORDS acute myocardial infarction, aortic root thrombus, HeartMate 3 left ventricular assist device, right ventricular failure

APPENDIX For supplemental videos, please see the online version of this paper.