



Peer-Reviewed Case Report

ST-Segment Elevation Myocardial Infarction in Patient with Left Ventricular Assist Device

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Abstract

Acute myocardial infarction is a rare event in a patient with a left ventricular assist device (LVAD). We present a case of ST-segment elevation myocardial infarction in a LVAD patient.

Keywords

Ventricular assist device; Heart failure; Acute myocardial infarction

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A 53 year-old male with a HeartMate II (Thoratec Corporation, Pleasanton, CA) left ventricular assist device (LVAD) presented to an outside emergency department with acute onset of chest pain. LVAD placement occurred 5 months prior to patient's presentation due to end-stage ischemic cardiomyopathy. The patient's history was significant for refractory gastrointestinal bleeding while on LVAD support which resulted in complete cessation of all anticoagulation including aspirin. ECG demonstrated significant ST elevation in the anterior leads. The patient was given aspirin and started on intravenous heparin and transferred to our institution for urgent left heart catheterization. Catheterization showed 100% thrombotic occlusion of the mid left anterior descending artery (Figure 1). Aspiration thrombectomy and percutaneous transluminal coronary angioplasty were performed with successful result and minimal residual disease (Figure 2). Mild to moderate disease was found elsewhere consistent with his history of known ischemic heart failure. The patient was loaded with 600 mg of clopidogrel. Chest pain completely resolved.



Fig 1. Total left anterior descending artery occlusion on coronary angiogram. Video at: <https://vimeo.com/130852738>

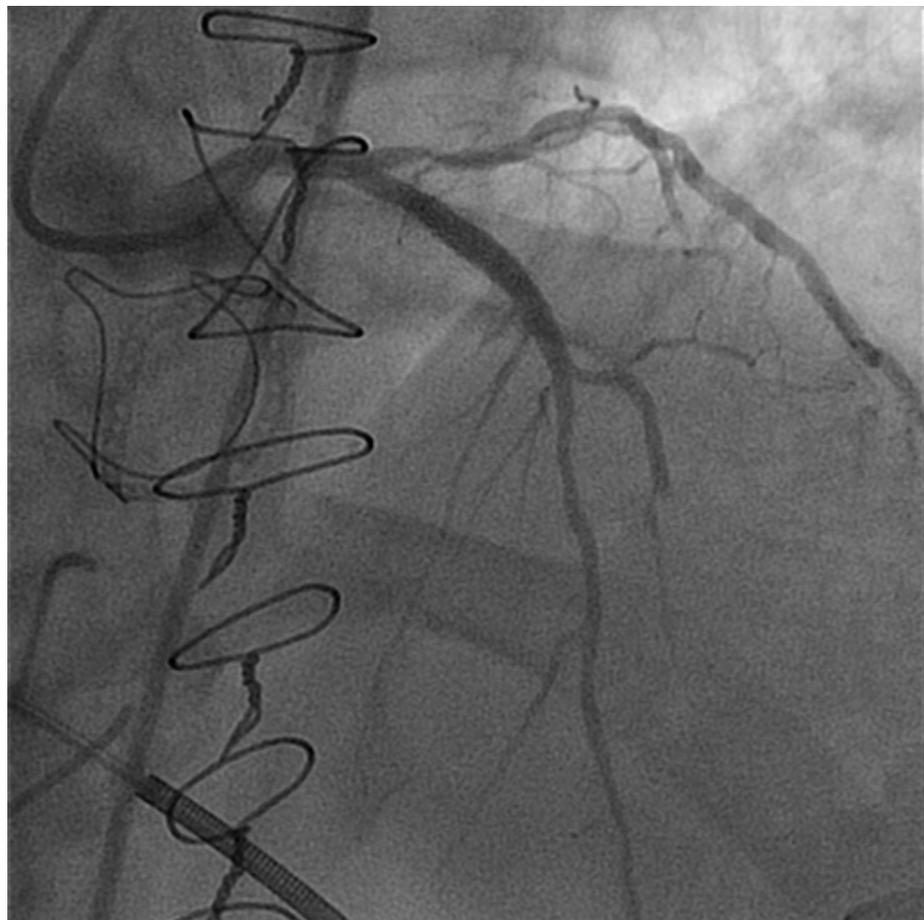


Fig 2. Restoration of flow in left anterior descending artery following successful thrombectomy and angioplasty.



However, the day following the procedure, the patient developed acute onset of right facial droop, expressive aphasia, and right-sided weakness. Emergent head CT with angiography showed a thrombus within the left middle cerebral artery (M1 segment). Urgent cerebral angiography was performed and the patient underwent successful mechanical thrombectomy of the left middle cerebral artery with restoration of 85% of the left middle cerebral circulation. The patient's neurological status dramatically improved.

Both of these events appeared to be embolic in nature, possibly contributed by the patient's lack of anticoagulation. Upon admission, lactate dehydrogenase (LDH) was elevated at 466 U/L compared to 232 U/L 1 month prior to admission. Free hemoglobin was elevated as well at 45 mg/dL. Transthoracic echocardiogram showed no visible thrombus in left ventricular cavity and Doppler velocities through inflow cannula were normal. Parameters were normal on LVAD interrogation and no power spikes were noted.

Given that he had no active signs of gastrointestinal bleeding, the patient was started on intravenous heparin and warfarin and aspirin were also initiated. The patient did well initially with continued improvement in his neurological deficits. However eight days after admission to the hospital, he developed new aphasia and hemiparesis once again. Urgent head CT showed the new development of an intraparenchymal hematoma centered in the temporal lobe with mild mass effect. The patient was not believed to be a candidate for neurosurgical intervention. As a result, anticoagulation was stopped, including clopidogrel. Despite the new neurological insult, over the next several days the patient once again was improving from a neurological standpoint. However, 16 days following hospital admission, his mental status acutely deteriorated and he became unresponsive. Head CT showed a new acute infarction in the right middle cerebral artery territory. Given his general poor prognosis, the family elected to proceed with comfort measures and he died on hospitalization day #20. The family declined autopsy, and the LVAD was not sent to the manufacturer.

Acute myocardial infarction following LVAD implantation is a rare phenomenon. The latest annual INTERMACS report showed that myocardial infarction occurred in the first 12 months after LVAD implant at a rate of 0.08 events per 100 patient-months.¹ Compared to other adverse events such as bleeding, right heart failure, arrhythmias, and infection, myocardial infarction is far less common. ST-segment elevation myocardial infarction has been reported in an LVAD patient soon after implant due to thrombotic occlusion of the left coronary cusp.² In the present case, we hypothesize that our patient's myocardial infarction and stroke were likely embolic events from a thrombus in the blood pump pathway. The exact location of the thrombus was not certain. LDH and free hemoglobin were both elevated, but other LVAD parameters including power were normal. However, it is well known that LDH can often be the only sign of impending LVAD thrombosis.³ Lack of anticoagulation due to refractory gastrointestinal bleeding likely was the major factor contributing to thrombus formation. Recent data suggests that rates of LVAD thrombosis are increasing and clinicians should be aware that acute myocardial infarction may be evidence of this complication.⁴ We hope that this case report adds awareness to this



potential complication which may become more common as patients are living longer with continuous-flow LVADs.

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