Left ventricular assist device thrombosis presenting as an acute coronary syndrome

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Video clip is available online.

Left ventricular (LV) assist device (LVAD) thrombosis is a major cause of morbidity among LVAD recipients.¹ Hemodynamic instability, heart failure symptoms, and complications related to systemic thromboembolism are common manifestations of acute LVAD failure.² We report a case of presumed LVAD thrombosis presenting as an acute coronary syndrome.

CLINICAL SUMMARY

A 63-year-old woman with ischemic cardiomyopathy required placement of a HeartMate II (Thoratec Corporation, Pleasanton, Calif) LVAD as destination therapy. Two years later, she came to her local emergency department with acute onset of crushing chest pain and dyspnea. An admission electrocardiogram showed ventricular pacing, only without new ST-segment elevation. A continuous hum throughout the precordium was noted, along with bibasilar rales. Laboratory analysis showed an elevated troponin I value and hemolysis (Table 1). Echocardiography showed

a dilated LV and opening of the aortic valve with every cardiac cycle.³ Coronary angiography was deferred, and right heart catheterization showed low right and left heart filling pressures and a low normal cardiac index (Table 1). Initial management included unfractionated heparin, aspirin, and clopidogrel for a possible cardioembolic myocardial infarction and presumed LVAD thrombosis. After initial stabilization on medical therapy, the patient had hypotension and worsening dyspnea without chest pain develop on hospital day 5 (Table 1). Transesophageal echocardiography showed no aortic or LV thrombus and limited Doppler flow across the LVAD inflow conduit (Video 1). Coronary angiography through the right radial artery was unchanged relative to a previous study (Figure 1, A, and Video 2). Initial LV pressure was 79/8 mm Hg, confirming incomplete LV unloading (Figure 1, B and C). Left ventriculography showed patent inflow and outflow LVAD conduits (Video 3). Collectively, these findings suggested the diagnosis of presumed LVAD thrombosis. LVAD exchange was deferred because of severe peripheral vascular disease and multiple prior sternotomies. Alteplase (1 mg/min) was infused into the LV without an initial bolus through a pigtail catheter with intermittent monitoring of LV pressures (Figure 1, A). After 28 minutes of infusion, LV pressure decreased to 19/2 mm Hg, with an aortic pressure of 110/85 mm Hg, indicating successful LVAD thrombolysis (Figure 1, B and C). Alteplase was continued for a total of 42 minutes. LVAD parameters showed increased flow, a reduced pulsatility index, and closure of the aortic valve (Table 1). During the next several days, hemolysis parameters improved (Figure 2), and the patient was discharged on hospital day 12 with aspirin, clopidogrel, and warfarin. Lactate dehydrogenase levels remained less than 500 IU/L 1 month after discharge.

DISCUSSION

Nearly 1500 LVADs are implanted annually in the United States alone.⁴ The diagnosis and management of LVAD

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TABLE 1. Hemodynamic, laboratory, and left ventricular assist device parameters

	Hemodynamic variables						Laboratory studies				LVAD parameters				
	MAP (mm Hg)	HR (beats/min)	RA (mm Hg)	PAP (mm Hg)	PCWP (mm Hg)	CI (L/min/m ²)	LDH (IU/L)	TB (mg/dL)	pf Hb (mg/dL)	Tn I (ng/dL)	Speed (rpm)	Flow (L/min)	Power (W)	PI	AV
Admission	70	97	2	19/8	3	2.3	1380	1.1	33.6	27.4	—	—	—	—	—
Pre-tPA	74	80	2	20/12	16	2.6	4136	2.3	75.1	9.25	9200	4.1	8.7	5.2	Opening
Post-tPA	90	80	2	22/6	4	4.5	1957	0.3	6.4	8.47	9800	6.1	7.1	3.5	Closed

LVAD, Left ventricular assist device; MAP, mean arterial pressure; HR, heart rate; RA, right atrial pressure; PAP, pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; LDH, lactate dehydrogenase; TB, total bilirubin; pf Hb, plasma free hemoglobin; Tn I, troponin I; PI, pulsatility index; AV, aortic valve; tPA, alteplase.

FIGURE 1. Intracavitary left ventricular (LV) assist device thrombolysis. A, Radial artery access with a 6F pigtail catheter in the left ventricle and a 3-way stopcock for intermittent left ventricular hemodynamic monitoring with a fluid-filled manometer during thrombolytic infusion. B, Left ventricular pressures at baseline and during thrombolytic infusion, showing a gradual reduction in both left ventricular systolic and diastolic pressures (shown as systolic/diastolic, end-diastolic pressure). C, Left ventricular hemodynamic tracings before and after alteplase (tPA) infusion, followed by a pullback sequence from the left ventricle to the aorta, showing a step up in systolic and diastolic pressures and suggesting improved left ventricular unloading by the left ventricular assist device.

dysfunction remains poorly understood. Our report has several potentially important clinical implications. First, we identify acute coronary syndrome as a potential manifestation of LVAD failure. Acute coronary syndrome in patients with LVADs may be due to coronary thrombosis, cardioembolus, or primary LVAD failure leading to demand ischemia. Second, emergency coronary revascularization in this complex clinical scenario carries a risk of bleeding, systemic thromboembolism, and worsening hemodynamic instability. For this reason, initial angiography was deferred, and instead the diagnosis of presumed LVAD thrombosis, likely caused by bearing thrombosis, was investigated by studying LVAD parameters, laboratory evidence of hemolysis, imaging studies to rule out aortic root or intracavitary thrombosis, and invasive hemodynamic

measurements. Third, this case demonstrates that right heart catheterization alone may be insufficient to diagnose LVAD dysfunction. Instead, left ventriculography with intracavitary hemodynamics may be a helpful alternative to evaluate LVAD function. Finally, fibrinolytic therapy is often a temporary solution and carries a high rate of recurrent thrombosis. LVAD exchange remains the criterion standard for the treatment of LVAD thrombosis; however, not all patients are candidates for LVAD exchange. We report our approach to intracavitary thrombolysis through the radial artery with intermittent monitoring of LV pressures.

In conclusion, acute LVAD thrombosis complicated by an acute coronary syndrome may be successfully managed by intracavitary thrombolysis in select cases. A careful assessment of LVAD function and management options must involve consultation with an interventional cardiologist, a heart failure specialist, and a cardiac surgeon.

FIGURE 2. Serum lactate dehydrogenase (LDH) levels before and after intracavitary thrombolysis.

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