A 38-year-old man with a history of ischemic cardiomyopathy was admitted to the hospital for shortness of breath 6 months after receiving a HeartMate II left ventricular assist device (LVAD; Thoratec Corporation, Pleasanton, CA). The patient had volume overload with elevated jugular venous pressure, edema, and bilateral rales. The pump speed was set at 9000 rpm, and the power was 6.5 W. His lactate dehydrogenase (LDH) level, which had been 350 to 450 IU/L, increased to 450 to 650 IU/L, and his creatinine level had increased from 1.7 to 2.0 mg/dL. Because the patient had shortness of breath, the pump speed was increased to 10000 rpm, but his symptoms barely improved. The power increased minimally to 7 W. During the next 48 hours, his LDH levels increased to 1000 to 1300 IU/L.

A review of the pump history did not show any power spikes or low-flow alarms. A speed change echo showed no change in end-diastolic diameter despite increasing revolutions per minute. The pump speed was reduced to 9000 rpm, and the LDH decreased to 600 to 700 IU/L, with a power of 6 to 6.5 W.

A computed axial tomographic scan of the chest showed that the pump had migrated from the normal orientation (Figure [A]) to one that distorted the outflow, kinking the proximal portion of the outflow graft (Figure [B]). The left ventricular assist device was exchanged, and the outflow connection was repositioned.

After the pump was exchanged and the kink eliminated, the LDH decreased to 300 to 400 IU/L. In the perioperative period, the pump speed was set at 8000 rpm and was gradually increased to 8200 rpm. The patient remained asymptomatic. He recovered and was discharged. At the next clinic visit, the pump speed was increased to 9200 rpm. Both the LDH (<400 IU/L) and creatinine levels returned to baseline. He continues to do well and is asymptomatic. The flow has been stable at 5 L/min, with a pulsatility index of 5.5 and power of 5.7 W.

The absence of power spikes in the setting of a high LDH implies a differential diagnosis of turbulent flow through the system, either in the inflow or outflow. Thrombus or fibrin in the pump would cause a power increase. The absence of low-flow alarms seemed to rule out recurrent suction events or severe flow restriction out of the left ventricle, which would also have caused a more pronounced power change. In our case, the inflow cannula seemed to be in a good position. We were able to identify a kink in the outflow graft because of migration of the pump. After pump exchange, the patient had no symptoms of heart failure, and the flow was stable at a lower pump speed.

In summary, increased LDH, especially with a hemodynamic change, can be a sign of turbulent and often reduced blood flow. Change in pump position is a potential cause.

Disclosures

None.

Keywords: HeartMate II ◼ ventricular assist device ◼ ventricular assist device complications

Received November 27, 2012; accepted March 6, 2013.
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(Circ Heart Fail. 2013;6:e30.)

Circ Heart Fail is available at http://circheartfailure.ahajournals.org
DOI: 10.1161/CIRCHEARTFAILURE.112.000020
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_Circ Heart Fail._ 2013;6:e30
doi: 10.1161/CIRCHEARTFAILURE.112.000020
_Circulation: Heart Failure_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3289. Online ISSN: 1941-3297

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