A 59-year-old man with nonischemic cardiomyopathy, advanced heart failure, and group 2 pulmonary hypertension underwent HeartWare left ventricular assist device (LVAD) implantation as bridge to transplantation. Off-pump LVAD implantation was performed via minimally invasive left thoracotomy and upper hemisternotomy with an estimated blood loss of 200 mL. Postoperative VAD settings were as follows: speed, 2600 rpm; power, 4 W; flow, 5.5 to 5.7 L/min; and pulsatility index (PI), 4. Aspirin 325 mg daily and warfarin were begun on postoperative day 1. Over the course of the next few days, he was extubated and optimized hemodynamically with continued inotropic support (milrinone), and a therapeutic international normalized ratio (2–3) was achieved. Before his transfer out of the intensive care unit, he underwent a transthoracic echocardiogram, showing a small anterior pericardial effusion (0.8 cm) without hemodynamic compromise. He was transferred to the step-down unit on postoperative day 8 in stable condition with LVAD speed uptitration to 2700 rpm (flow, 5–6.5 L/min; power, 3.5–4 W; PI, 7). On postoperative day 16, he complained of postural dizziness, substernal chest pressure, and worsening drowsiness. Examination revealed an elevated jugular venous pressure (15 cm) and normal VAD hum. LVAD parameters were as follows: speed, 2700 rpm; power, 3.8 to 5.4 W; flow, 5 to 8 L/min; and PI, 2.5 to 5. A unique finding of significance was the respiratory variation of PIs on interrogation (Movie I in the online-only Data Supplement). Urgent transthoracic echocardiography, showing a large anterior pericardial effusion (3.3 cm) with diastolic right ventricular collapse and distended inferior vena cava (Movies II–IV in the online-only Data Supplement). The patient was taken to the operating room for pericardial window and drainage of the effusion with immediate improvement in hemodynamics and upper hemisternotomy with an estimated blood loss of 200 mL. Postoperative VAD settings were as follows: speed, 2600 rpm; power, 4 W; flow, 5.5 to 5.7 L/min; and pulsatility index (PI), 4. Aspirin 325 mg daily and warfarin were begun on postoperative day 1. Over the course of the next few days, he was extubated and optimized hemodynamically with continued inotropic support (milrinone), and a therapeutic international normalized ratio (2–3) was achieved. Before his transfer out of the intensive care unit, he underwent a transthoracic echocardiogram, showing a small anterior pericardial effusion (0.8 cm) without hemodynamic compromise. He was transferred to the step-down unit on postoperative day 8 in stable condition with LVAD speed uptitration to 2700 rpm (flow, 5–6.5 L/min; power, 3.5–4 W; PI, 7). On postoperative day 16, he complained of postural dizziness, substernal chest pressure, and worsening drowsiness. Examination revealed an elevated jugular venous pressure (15 cm) and normal VAD hum. LVAD parameters were as follows: speed, 2700 rpm; power, 3.8 to 5.4 W; flow, 5 to 8 L/min; and PI, 2.5 to 5. A unique finding of significance was the respiratory variation of PIs on interrogation (Movie I in the online-only Data Supplement). Urgent transthoracic echocardiography, showing a large anterior pericardial effusion (3.3 cm) with diastolic right ventricular collapse and distended inferior vena cava (Movies II–IV in the online-only Data Supplement). The patient was taken to the operating room for pericardial window and drainage of the effusion with immediate improvement in hemodynamics and resolution of the marked respiratory variations in PI (Movie V in the online-only Data Supplement).

Discussion

Pericardial effusion, especially localized, is very common after open heart surgery as a result of intrapericardial inflammation and bleeding. Patients receiving LVAD are especially at higher risk of pericardial bleeding and hence cardiac tamponade (CT) because they require early postoperative anticoagulation and antiplatelet therapies. CT is a medical emergency requiring rapid diagnosis and urgent intervention; however, the presence of an LVAD may mask common clinical findings associated with CT. A reduction in PI, a sudden increase in right atrial pressures, or nonresponsiveness to fluid challenge can be observed in LVAD recipients with CT. Respiratory variability in PIs has not been described as a sign suggestive of CT in LVAD recipients. Traditionally, transthoracic echocardiography has been a standard diagnostic tool to assess the severity of pericardial effusion with established criteria to assess tamponade physiology, including exaggerated respiratory variation in mitral and tricuspid inflow pattern. This respiratory variation in the inflow pattern has been attributed to ventricular interdependence and dissociation of intrathoracic and intracardiac pressure, which results not only in an overall reduction in inflow velocities but also in a reduction in mitral E velocity during inspiration and a reciprocal increase during expiration. A similar physiological response is likely to explain the respiratory PI variation observed in our patient on LVAD interrogation. The HeartWare VAD records beat-to-beat PI, thus allowing immediate evaluation of respiratory variation when CT is a concern. We observed that the PI decreased during inspiration and increased during expiration, along with an overall reduction in amplitude caused by the exaggerated decrease in left ventricular volume during inspiration. To the best of our knowledge, this is the first description of the use of the PI to diagnose CT in LVAD recipients. Such PI findings can assist clinicians in the rapid diagnosis and treatment of tamponade.

Disclosures

None.

References


Rapid Diagnosis of Cardiac Tamponade Using Pulsatility Index Variability in a Patient With a HeartWare Ventricular Assist Device
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