Rapid progression of carotid artery atherosclerosis and stenosis in a patient with a ventricular assist device

James A. Saltsman III, MD, MPH,[a] Reid A. Ravin, MD,[b] Peter L. Faries, MD,[b] and **Rami Tadros, MD,[b]**
New York, NY

This case describes the management of cerebrovascular disease in a patient with a left ventricular assist device (LVAD) who was awaiting cardiac transplantation. It demonstrates several unique features in managing vascular disease in patients with cardiac assist devices. First, we detail the difficulties in using duplex ultrasound to assess patients with altered hemodynamic physiology. Second, we report an instance of rapid progression of known carotid stenosis in a patient with a recently placed LVAD. This case suggests that patients with any degree of carotid stenosis before LVAD placement should be monitored closely for progression after the LVAD is placed. (J Vasc Surg Cases 2016;2:40-2.)

As mortality from myocardial infarction decreases, heart failure and ischemic cardiomyopathy are becoming more prevalent in the United States.[1] For patients with refractory heart failure, cardiac assist devices including left ventricular assist devices (LVADs) are increasingly being used as either a destination therapy or a bridge to cardiac transplantation.[2-4]

LVADs are implantable pumps with an inflow cannula that accepts blood from the left side of the heart and then pumps that blood out through an outflow cannula into the ascending aorta. Patients undergo a variety of physiologic changes, depending on the type of assist device and the time since implantation.[4-7] Modern devices function by use of an internal rotor that continuously pumps blood in a nonpulsatile fashion.[5] In the early postimplantation period, patients may have little or no blood ejection from the heart, and the aortic valve may not open with the cardiac cycle. In this situation, there may be minimal or no arterial pulsatility. Depending on the patient's unique physiology and time from implantation, some patients do regain some degree of physiologic blood ejection from the left ventricle.[6]

This case describes the management of cerebrovascular disease in a patient with an LVAD who was awaiting cardiac transplantation. It demonstrates several unique features in diagnosis and management of vascular disease in patients with cardiac assist devices. The patient described in this case report has consented to the publication of this article.

CASE REPORT

A 53-year-old man with a history of hypertension, insulin-dependent diabetes, coronary artery disease, atrial fibrillation, and ischemic cardiomyopathy treated with an automatic implantable cardioverter-defibrillator and then an LVAD presented to the emergency department with 1 day of abdominal pain and hematochezia. He was admitted and treated for acute ischemic colitis due to low flow from the ventricular assist device, which had been placed 1 month before admission. Transesophageal echocardiography confirmed no evidence of an embolic source from the heart or the LVAD. On hospital day 7, the patient developed sudden weakness of the left lower extremity. He denied other symptoms, and findings of a full neurologic examination were normal with the exception of 0/5 strength in the left lower extremity. A noncontrast-enhanced computed tomography scan of the head was obtained immediately and showed no acute abnormality. Weakness resolved spontaneously about 16 hours after onset. Duplex ultrasound examination of bilateral carotid arteries was obtained, which showed severe stenosis on B mode. Our institution's vascular laboratory uses validated institution-specific criteria to classify the degree of carotid artery stenosis. The degree of stenosis was quantified at 80% to 99% bilaterally. On the right side, peak systolic velocity (PSV) was 523 cm/s, and internal carotid artery (ICA)/common carotid artery (CCA) ratio was 9.98; on the left side, PSV was 371 cm/s, and ICA/CCA ratio was 9.88 (Fig 1). Computed tomography angiography of the head and neck confirmed the findings of severe narrowing and calcification of ICAs bilaterally, greater on the left side than on the right (Fig 2). Diagnosis was made of transient ischemic attack (TIA) due to symptomatic right ICA stenosis causing transient left-sided weakness.

Three months before the current admission, bilateral carotid artery duplex ultrasound examination was completed at our institution's validated vascular laboratory as part of the patient's preoperative evaluation before LVAD placement (Fig 1). At that time, a

From the Department of Surgery[a] and Division of Vascular Surgery, Department of Surgery,[b] Icahn School of Medicine at Mount Sinai.

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Correspondence: Rami Tadros, MD, Division of Vascular Surgery, Department of Surgery, Mount Sinai School of Medicine, 5 E 98th St, Box 1273, New York, NY 10029 (e-mail: rami.tadros@mountsinai.org).

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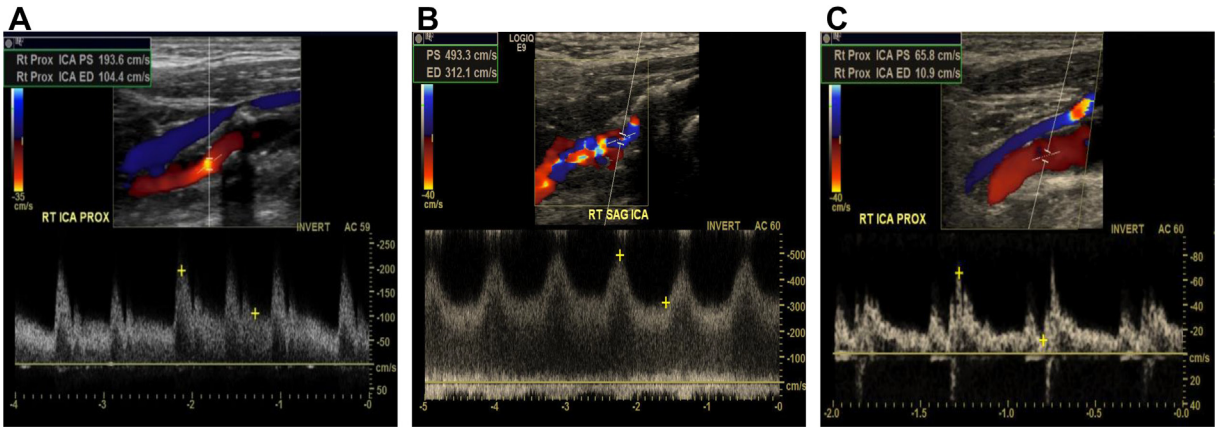

Fig 1. Representative images from duplex ultrasound examination of the right internal carotid artery (*ICA*) obtained at various time points. **A,** Before left ventricular assist device (LVAD) placement, duplex ultrasound shows elevated velocities consistent with moderate stenosis. *ED,* End diastole; *PS,* peak systole. **B,** At the time of transient ischemic attack (TIA) and before carotid endarterectomy, duplex ultrasound demonstrates high peak systolic velocity (PSV) as well as elevated end-diastolic velocity, decreased pulse pressure, and flow turbulence, indicating severe stenosis. **C,** After carotid endarterectomy and cardiac transplantation, duplex ultrasound demonstrates more normal flow dynamics.

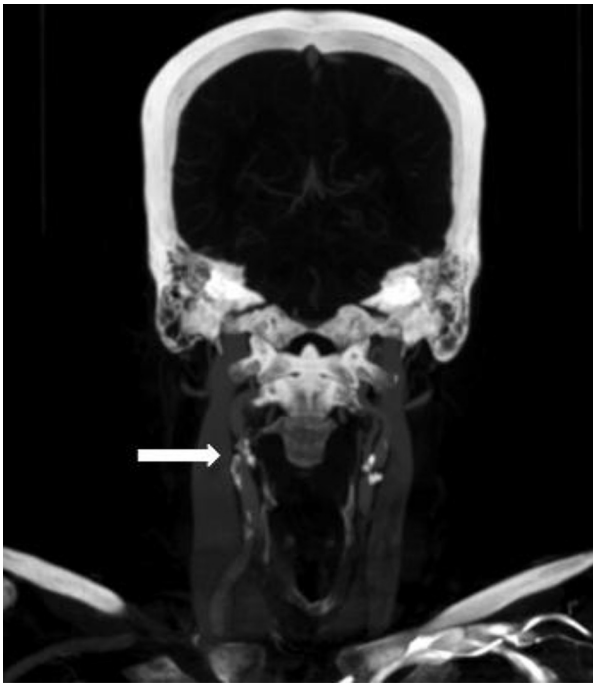

Fig 2. Representative image from computed tomography angiography of the neck obtained after the patient developed left leg weakness consistent with transient ischemic attack (TIA). The *arrow* indicates an area of significant carotid plaque and stenosis.

20% to 39% stenosis of the left ICA was observed (PSV, 71 cm/s; ICA/CCA ratio, 1.67) along with 70% to 79% stenosis of the right ICA (PSV, 213 cm/s; ICA/CCA ratio, 6.14). As such, the patient's presentation of TIA on this admission was consistent with significant progression of stenosis compared with previous duplex ultrasound examination 3 months before. Given the patient's recent symptom of left leg weakness attributed to the right-sided lesion, the decision was made to treat the right side first. The patient underwent right-sided carotid endarterectomy on hospital day 14. The procedure was uneventful, and the patient was discharged on postoperative day 11 after he was stable from cardiac issues and ischemic colitis.

The patient was then scheduled for left-sided carotid endarterectomy approximately 1 month later. At the time of readmission for the second procedure, the patient had not experienced any additional neurologic symptoms since the episode of leg weakness during his previous admission. The patient underwent left carotid endarterectomy, and the procedure was again uneventful. He was discharged home on oral anticoagulation on postoperative day 9 after his cardiac status was stable.

Several months after his second carotid endarterectomy, the patient underwent a heart transplantation and LVAD removal and has been doing well on follow-up. On duplex surveillance, his carotid arteries are widely patent, and the patient's heart failure symptoms have completely resolved (Fig 1).

DISCUSSION

This case illustrates a challenging diagnostic dilemma in carotid artery disease among patients with ventricular assist devices. There are currently no validated duplex velocity criteria for use in patients with ventricular assist devices; however, screening carotid duplex examinations are frequently obtained in this population of patients. Second-generation devices produce continuous, nonpulsatile blood flow without systole or diastole. LVADs are also known to cause alterations in duplex velocities, including an artificially decreased PSV as well as an increase in end-diastolic velocity.[7] Despite any LVAD-related reductions, in our patient we observed PSVs >400 cm/s in both lesions. Increased turbulent blood flow and blunting of waveforms are also characteristic in LVAD patients,[7] and

these are well demonstrated in ultrasound images from the time of TIA shown in Fig 1, *B*.

Given the lack of validated velocity criteria in LVAD patients, our vascular laboratory expressed a lack of confidence in quantifying the exact degree of stenosis in this case but noted that severe stenosis was suggested on B-mode ultrasound and that velocities in the ICAs were increased relative to the CCAs. The duplex ultrasound examination was interpreted cautiously, and the results were weighed in conjunction with the computed tomography angiography scan and the patient's symptoms. This case reinforces the need for further investigation into the use of carotid duplex ultrasound in the evaluation of patients with cardiac assist devices.

CONCLUSIONS

This is an interesting case of rapid progression of carotid artery atherosclerosis and stenosis in a patient with a ventricular assist device, which led to a cerebrovascular event. Most strokes in this population are associated with multiple factors, including inadequate anticoagulation leading to clot embolization from the LVAD pump or the native left ventricle.[8,9] Rapid progression of symptomatic carotid disease is not well described in LVAD patients, but there are interesting theoretical explanations for the phenomenon. For instance, it has been shown that shear stress is one factor that inhibits the development of atherosclerotic plaque, and areas of low shear stress are associated with greater atherosclerotic burden.[10] In this case, the patient had a lack of pulsatile flow due to the LVAD and thus lower arterial shear forces throughout the entire arterial system. It is possible that the low shear environment created by the patient's ventricular assist device contributed significantly to the rapid rate of progression in his carotid artery atherosclerotic disease between the time of LVAD placement in June and the development of stroke symptoms in July. This finding suggests that patients with any degree of carotid stenosis before LVAD placement should be monitored closely for progression of stenosis after the LVAD is placed.

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