A 32-year-old African American man with a medical history of hypertension and gunshot wound to the abdomen 8 years prior presented to the emergency department complaining of several weeks of exertional dyspnea, episodes of midsternal chest pain, and lower-extremity edema (more pronounced in the left leg). He stated that the dyspnea had progressed to that point at which he could only walk 2 blocks before having to rest, and he was unable to participate in his normal recreational activities. He also claimed that the lower-extremity swelling was a new development during the past several months. His social history was notable for smoking 1 pack a day for 15 years and a recent escalation of ethanol intake during the past 4 months, up to a pint or more of liquor a day. He denied drug use but had a history of cocaine use 5 to 6 years ago. On arrival, the patient was afebrile, tachycardic (110 bpm), and hypertensive (180/103 mm Hg); his breathing rate was 16/min. His physical examination was remarkable for a 3/6 holosystolic murmur heard across the entire precordium, bounding pulses, faint bibasilar crackles, and a prominent abdominal bruit. Swelling was noted in the bilateral lower extremities but was more pronounced in the left leg. Acute coronary syndrome, deep venous thrombosis, and pulmonary embolism were excluded in the emergency department. An infectious process was unlikely because he lacked fever or leukocytosis. An electrocardiogram demonstrated sinus tachycardia with left atrial enlargement, left axis deviation, and nonspecific anterior T-wave changes. His brain natriuretic peptide was elevated at 600 pg/mL, and a chest X-ray film (Figure 1) showed evidence of severe cardiomegaly with pulmonary vascular congestion; this was a marked change compared with his films from 4 years earlier. The patient was admitted for additional workup of new-onset congestive heart failure, which the primary team hypothesized to be secondary to alcohol-induced cardiomyopathy, given his increased consumption and lack of other heart failure-related risk factors.

**Diagnostic Studies**

The echocardiogram demonstrated a preserved ejection fraction of 60%, with normal global systolic function and no valvular abnormalities. It revealed pulmonary arterial systolic pressures of 60 mm Hg and evidence of high-velocity turbulent flow through the aortic isthmus. Based on his studies, examination, and history of traumatic abdominal gunshot wound, a computed tomographic angiogram was ordered to evaluate his aorta and downstream vasculature. The computed tomographic scan did not support coarctation. Instead, it exposed a massively dilated inferior vena cava and left pelvic venous system, along with a suspected left internal iliac AV fistula, with recruitment from adjacent vessels and a 1.6-cm calcified pseudoaneurysm just distal to the origin of the internal iliac artery (Figure 2A). Prior computed tomographic images from the time of his gunshot wound showed a closed-entry wound at the time of his gunshot (Figure 2B). The team hypothesized that the pseudoaneurysm was caused by an expandable missile or an impacted foreign body, which had produced a focal site of injury, leading to progressive dilatation and pseudoaneurysm formation.

**Figure 1.** An admission chest X-ray demonstrating an enlarged cardiac silhouette and prominent pulmonary vasculature.

**Figure 2.** A, A computed tomographic angiogram showing a dilated left-sided venous system and early venous return through the AV fistula into the engorged veins, as well as the calcified pseudoaneurysm. B, Comparison scan done 8 years prior when seen initially for a gunshot wound; a closed-entry wound can be seen at the top.
wound showed no evidence of fistula or vascular distension (Figure 2B). A diagnosis was made of progressive high-output heart failure from a traumatic AV fistula, likely from shrapnel migration, which explained his unilateral leg edema, abdominal bruit, and prevalent cardiac murmur.

**Treatments/Interventions**

A multidisciplinary team composed of vascular surgery, cardiology, and radiology deemed an endovascular intervention as the most appropriate initial strategy, given the obstacles his previous trauma surgical procedures presented to an open approach. An angiogram was performed to further characterize the fistula and collateral circulation, which confirmed a large left internal iliac AV fistula (Figure 3A and 3B). There was marked engorgement of the L4-5 left lumbar, sacral, and circumflex femoral arteries, with brisk retrograde flow feeding the fistula and distended pelvic venous system. After visualizing the fistula, a 13-mm × 5-cm Gore Viabahn-covered stent was deployed across the ostium. Follow-up images revealed marked improvement; showing antegrade flow in the distal internal iliac artery and resolution of any early venous return, as well as abrupt collapse of previously visualized collaterals (Figure 4A and 4B). The decision was made at that time not to embolize the vessel(s) because such dramatic results were achieved with stenting alone. The patient tolerated the procedure well. He was diuresed of his excess volume during the next few days; by the time of discharge, he experienced complete resolution of his initial examination findings and his clinical syndrome had completely resolved.

**Disclosures**

None.

**Key Words:** cardiomyopathy ■ collateral circulation ■ fistula ■ tomography ■ high output heart failure

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**Figure 3.** A and B, Pelvic arteriogram showing time progression of contrast through the AV fistula into the venous system, filling the inferior vena cava (seen as the lightly opacified large-caliber vessel just left of the aorta). B, demonstrates extensive vascular collaterals that have been formed on the patient’s left side.

**Figure 4.** A, Angioplasty of the Viabahn-covered stent in the left internal iliac artery just distal to the bifurcation. B, Follow-up arteriogram demonstrating no significant flow through the AV fistula or early venous return, as well as collapse of the collateral circulation seen in images from Figure 3.
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