High-Output Cardiac Failure and Coronary Steal With an Arteriovenous Fistula

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Creation of an arteriovenous access for hemodialysis can provoke a sequence of events that significantly affects cardiovascular hemodynamics. We present a 78-year-old man with end-stage renal disease and concomitant coronary artery disease previously requiring coronary artery bypass grafting including a left internal mammary graft to the left anterior descending artery, ischemic cardiomyopathy with left ventricular systolic dysfunction, and severe aortic stenosis who developed hypotension unresponsive to medical therapy after recent angioplasty of his ipsilateral arteriovenous fistula for high-grade outflow stenosis. This case highlights the long-term effects of dialysis access on the cardiovascular system, with special emphasis on complications such as high-output cardiac failure and coronary artery steal syndrome. Banding of the arteriovenous fistula provided symptomatic relief with a decrease in cardiac output. Avoidance of arteriovenous access creation on the ipsilateral upper extremity in patients with a left internal mammary artery bypass graft may prevent coronary artery steal syndrome.

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Teaching Cases focus on interpretation of pathology findings, laboratory tests, or imaging studies to educate readers on the diagnosis or treatment of a clinical problem.

Introduction

Cardiovascular disease is an important source of mortality and morbidity in patients with chronic kidney disease.[1,2] Data from the US Renal Data System revealed that risk for death in a dialysis patient with heart failure is 33%, 46%, and 57% at 12, 24, and 36 months, respectively, after dialysis therapy initiation.[3,4] There are a number of cardiovascular effects of arteriovenous (AV) fistula (AVF)/graft (AVG) creation, including increased cardiac output, increased blood volume, and increased pulmonary blood flow. High-output heart failure and coronary steal are rare complications of AVF creation.[5,6] Few prospective studies have examined the impact on the heart of AVF creation. We present a case of the adverse hemodynamic impact of both high-output heart failure and coronary steal from an AVF in a patient with previous coronary artery bypass graft (CABG) with aortic stenosis and left ventricular (LV) systolic dysfunction.

Case Report

Clinical History and Initial Laboratory Data

A 78-year-old white man with end-stage renal disease from uncontrolled diabetes mellitus, who had been treated by hemodialysis since 2013 through a left arm brachiocephalic AVF created in 2011, presented from his outpatient dialysis unit with hypotension (systolic blood pressures, 60s-70s mm Hg) unresponsive to intravenous fluid resuscitation. Additionally, he reported exertional dyspnea and presyncope during the past few weeks.

Cardiovascular disease history included coronary artery disease requiring CABG in 1994, including a left internal mammary artery (LIMA) to the left anterior descending artery; and atrial fibrillation on warfarin therapy. Before initiation of dialysis therapy, echocardiography from 2009 revealed a mildly dilated left ventricle, but with preserved ejection fraction of 60% to 65% and mild mitral and tricuspid regurgitation.

AVF intervention to maintain patency included angioplasty performed before dialysis therapy initiation (outflow cephalic vein stenosis) in 2012 without cardiovascular disease symptoms at the time. One year after dialysis therapy initiation (2014), LV ejection fraction decreased to 45% to 50%, and moderate aortic stenosis (valve area, 1.3 cm^2) was present. Ever since, the patient was repeatedly admitted for shortness of breath, at times complicated by demand ischemia.

No additional intervention of the AVF was made until 3 days before presentation, for high-grade outflow stenosis. Angioplasty was performed for 50% stenosis of the cephalic arch (9 mm × 8 cm balloon with 18 atm of pressure for 1 minute, residual stenosis < 10%) and 90% stenosis of the upper-arm cephalic vein (7 mm × 8 cm balloon, then 9 mm × 8 cm balloon with 50% residual stenosis) further requiring a 10 cm × 100 mm self-expanding stent (after it was dilated with a 9-mm balloon with no residual narrowing). Access flows before angioplasty ranged from 889 to 1,748 mL/min. Postangioplasty values were not obtained while hospitalized, but after banding, the value was 889 mL/min.

Following the latest angioplasty of the AVF, the patient developed severe chest discomfort with diaphoresis treated with sublingual nitroglycerin and morphine. On examination, blood pressure (BP) was 109/65 mm Hg, heart rate was 110 beats/min, and he had 14-cm jugular venous distention, bibasilar crackles, and trace lower-extremity edema. An electrocardiogram revealed new anterolateral ST depressions. Troponin I concentration was elevated to 77 (reference, <0.06) ng/mL. A chest radiograph showed an enlarged cardiac silhouette and pulmonary vascular congestion compatible with congestive heart failure. Echocardiography demonstrated a reduction in LV ejection fraction to 35%, now severe aortic stenosis (mean gradient, 22 mm Hg with valve area of 0.9 cm^2), and a dilated right ventricle with severely reduced systolic function.

Additional Investigations

Vasopressor therapy was initiated for worsening hypotension (BP as low as 60/30 mm Hg). Cardiac catheterization was emergently performed, revealing moderate to severe fixed aortic stenosis with a minor component of pseudoaortic stenosis from the underlying ischemic cardiomyopathy with LV systolic dysfunction. With a patent AVF and on norepinephrine treatment to maintain an arterial BP of 80/40 mm Hg, baseline Fick cardiac output (with an assumed oxygen consumption index of 125 mL/min/m^2) was elevated at 6.0 L/min, mean aortic valve gradient was 26 mm Hg, and calculated aortic valve area was 1.05 cm^2.

During occlusion of the AVF using a BP cuff, norepinephrine treatment was discontinued given the increase in arterial BP. Systolic BP increased to as high as 120 to 130 mm Hg (with occlusion of the AVF and off norepinephrine treatment; Fig S1A) from 100 to 110 mm Hg (with a patent AVF and on norepinephrine treatment; Fig S1B). Repeat Fick cardiac output decreased to 4.0 L/min, mean aortic valve gradient was unchanged at 26 mm Hg (shaded area between the aortic and LV pressure tracings in Fig S1), and calculated aortic valve area was reduced to 0.87 cm^2. This was consistent with a component of pseudoaortic stenosis seen with low-flow low-gradient aortic stenosis and underlying LV systolic dysfunction in which the valvular area and leaflet opening may increase with maneuvers that increase cardiac output and transvalvular flow by increasing LV stroke volume, such as the lower vascular resistance with the patent fistula. The full set of the patient's hemodynamic parameters both pre– and post–AVG occlusion are shown in Table 1. Without the vasopressors and in the setting of a patent AVF, BPs, central BPs, and systemic vascular resistance would have been lower, with likely a stable or mild increase in cardiac output.

Coronary subclavian steal was demonstrated in our patient during native left coronary angiography performed with an open AVF. Retrograde filling and reversal of flow in the patent LIMA with contrast was seen filling the left subclavian artery (Movie S1; Fig S2).

Interventions were performed the next day for recurrent chest pain, hypotension requiring norepinephrine, and electrocardiogram abnormalities (anterolateral ST depressions). Drug-eluting stents were placed for 80% stenoses in the native first obtuse marginal artery through a patent saphenous vein graft, and native distal right coronary artery. Balloon aortic valvuloplasty for the aortic stenosis using a 20-mm diameter balloon resulted in only a slight reduction in mean gradient from 30 to 26 mm Hg and increased valve area from 1.0 to 1.1 cm^2.

Diagnosis

With the finding that mean arterial pressures consistently increased with compression of the AVF, high-output failure was thought to be from high shunt flow precipitated by the recent dilation of the AVF stenosis. The lack of benefit from the coronary intervention and balloon valvuloplasty suggested that coronary steal through the patent LIMA and the patent AVF, not the obstructive coronary artery disease or aortic stenosis, was primarily responsible for the recurrent angina and electrocardiogram changes.

Clinical Follow-up

Given the persistent hypotension requiring vasopressors and continued AVF occlusion, dialysis was instead performed through a tunneled dialysis catheter during the hospital admission. Five days after dialysis catheter insertion, vascular surgery banded the AVF, with a reduction in diameter from 11 to 6 mm, after which vasopressor treatment was successfully discontinued and the patient's angina resolved. AVF use was resumed 2 days after banding, allowing for subsequent removal of the tunneled dialysis catheter.

Discussion

AVFs and AVGs are considered the optimal choices for hemodialysis access. However, creation of an AV access for hemodialysis imparts increased complexity to the physiology of cardiovascular hemodynamics. AV access creation can result in a reduction in total peripheral vascular resistance, which in turn increases sympathetic activity, leading to increased contractility and stroke volume. The overall effect is an increase in cardiac output, leading to expansion of blood volume, causing elevated atrial natriuretic peptide and brain natriuretic peptide concentrations. This increase in cardiac output can lead to LV hypertrophy.[5,7,8] The incidence of LV hypertrophy is reported to increase from 67% to 83% and 90% at 1 and 3 months, respectively, after AV access creation, suggesting that hemodynamic changes from the AV access contribute to LV hypertrophy progression.[9] Regression of LV hypertrophy has been observed after kidney transplantation and with the loss of AV access function.[10]