

The Arteriovenous Fistula: An Often Overlooked Precipitant of High Output Heart Failure

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Abstract

Heart failure usually occurs in the setting of a low cardiac output, however in rare cases it may be associated with a high cardiac output. We present a rare and intriguing case of a patient with end-stage renal disease (ESRD) on hemodialysis that developed high output heart failure from his arteriovenous fistula (AVF). A 36 year old African American male with ESRD secondary to hypertension, on hemodialysis for six years, presented to emergency room with a history of chest pain and shortness of breath. He was diagnosed with congestive heart failure secondary to coronary artery disease. His transthoracic echo showed an ejection fraction of 65% and both the stress test and cardiac enzymes were negative for ischemia. Further workup revealed a cardiac output of 10.6 L/min and access flow of 2.37 L/min. We made a diagnosis of high output heart failure secondary to the AVF. He was taken to surgery for a minimally invasive limited ligation endoluminal-assisted revision (MILLER) procedure after which his symptoms resolved. High output heart failure, though a rare complication of AVF placement is an important differential in hemodialysis patients who present with symptoms of congestive heart failure. An accurate diagnosis will prevent morbidity and unnecessary hospital admissions.

Keywords: Heart failure; Low cardiac output; High cardiac output; Arteriovenous fistula

Introduction

Heart failure usually occurs in the setting of a low cardiac output; however in rare cases it may be associated with a high cardiac output. This is otherwise referred to as high-output heart failure. High-output heart failure may be caused by a variety of underlying conditions; these include hyperthyroidism, beriberi, anemia and pregnancy. This discussion focuses on high-output heart failure in the dialysis population secondary to arteriovenous fistula (AVF) creation. When a dialysis patient with an AVF presents repeatedly with volume overload not improved with medical therapy or dialysis, it is imperative to consider high output heart failure secondary to increased blood flow from the access as a possible etiology. We present a rare and intriguing case of a patient with renal failure on hemodialysis that developed heart failure from his AVF.

Case Report

A 36 year old African American male with end-stage renal disease (ESRD) secondary to hypertension, on hemodialysis for six years, presented to emergency room with a one day history of chest pain and shortness of breath. He described a left sided, sharp pain which was 7/10 in intensity. The pain radiated to the left shoulder and improved with leaning forward. He noted that the chest pain and shortness of breath worsened during dialysis. The patient initiated dialysis six years prior to presentation and his previous access was a left brachiocephalic AVF. His current access was a right brachiocephalic fistula. He had a past history of coronary artery disease and obstructive sleep apnea. On physical examination, he was tachycardic with a baseline pulse of 105/min and a widened pulse pressure of 82. His chest was clear to auscultation and cardiovascular exam was unremarkable. He had a right brachiocephalic AVF with a prominent thrill. It had an appropriate augmentation of flow and it collapsed on raising the arm above the level of the heart. However, on manually occluding the AVF, his heart rate decreased instantaneously from 105 bpm to 70 bpm. Electrocardiography on presentation showed sinus tachycardia with asymmetrical T-wave inversion in lateral leads. A chest X-ray demonstrated cardiomegaly with no acute abnormality. His complete blood cell counts, coagulation

profile, thyroid and liver function tests were within normal limits. His cardiac enzymes were negative for ischemia.

He was admitted to the hospital with a diagnosis of congestive heart failure secondary to coronary artery disease and dialyzed. A stress test done was negative for ischemia. His transthoracic echocardiogram showed severe concentric left ventricular hypertrophy with an ejection fraction of 65%. There were no regional wall motion abnormalities detected but he had significant diastolic dysfunction. A computer tomography done was negative for a pulmonary embolus. At this point, we had to consider other possible causes for his CHF. We calculated his cardiac output (CO) and vascular access flow (Qa) using ultrasound dilution Transonic Hemodialysis Monitor HD02. The CO was elevated at 10.6 L/min at various times. The vascular access flow (Qa) was 2.37 L/min constituting a Qa: CO ratio of 0.22. Vascular surgery was consulted and the patient was taken to surgery for a minimally invasive limited ligation endoluminal-assisted revision (MILLER) procedure. After the initial procedure and a subsequent revision, both the vascular access flow and the cardiac output decreased with resolution of the patient's symptoms.

Discussion

The creation of the AVF sets up a cascade of events beginning with a reduction in peripheral resistance, causing an increase in cardiac output. The rise in the cardiac output does not immediately result in overt heart failure. With time, as the cardiac output increases, circulating blood volume also increases. This results in increase in right-sided pressures including right atrial pressure, pulmonary

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arterial pressure and inferior venacava pressure. Eventually, the left ventricular end diastolic volume increases. The dilatation of the left ventricle ultimately compromises the ejection fraction and eventually the patient develops symptomatic heart failure [1-5]. The timing from the creation of the AVF varies from six weeks to years.

The access flow in the hemodialysis patients with high output heart failure from AVF is typically 5-6 L/min. The greater the access flow, the higher the risk for heart failure and patients with flows above 4 L/min are at increased risk. The access flow rate Q_a can be measured with an ultrasound using a dilution technique. Although there are no KDOQI guidelines as to the upper limit of inflow rates of an AVF, in general higher access flows may be predictive of increased risk. Some studies have also associated previous cardiac disease as a risk factor for development of this disease. Begin et al showed that access flows depends on location with upper arm fistulas experiencing twice as much blood flow compared with lower arm fistulas however there is controversy to if this predisposes patient to the development of high output heart failure [6-10].

These patients are usually misdiagnosed with volume overload secondary to chronic under dialysis or exacerbation of congestive heart failure secondary to underlying atherosclerotic disease. It is therefore important to understand the symptoms and signs associated with high output failure associated with AVF. The symptoms include shortness of breath, especially on exertion, non-productive cough and lower extremity swelling. Physical findings are increased jugular venous pressure, tachycardia usually with a third heart sound, bilateral bibasilar crackles and a wide pulse pressure. The examination of the AVF may yield additional clues to diagnosis. It may have a palpable thrill. It may be dilated or hypertrophied to accommodate the high flow volumes. There may be signs or symptoms of vascular steal syndrome such as rest pain distal to the fistula, pallor and diminished pulses on the affected extremity [6]. One could also attempt the Nicola-doni-Branham test as we did which is positive if there is a reflex bradycardia following the occlusion of the arterial inflow of the fistula [11]. The occlusion causes an increase in peripheral vascular resistance and afterload. The increased afterload causes a reflex bradycardia. Laboratory data may reveal an elevated atrial natriuretic peptide and brain natriuretic peptide [12,13]. Clinical suspicion can be confirmed by a transonic examination of the fistula site which determines the blood flow.

A transthoracic echo may reveal a high, low or normal left ventricular ejection fraction depending on the extent of disease progression. Earlier on in the disease process the ejection fraction is normal but as time progresses there may be further reduction in the systolic function leading to lower ejection fractions. Some authors recommend a right heart catheterization which would show a low systemic vascular resistance and pulmonary hypertension as a result of the large preload volumes. The pulmonary vascular resistance may be normal [12]. Pandeya et al. introduced the concept of using a ratio of access flow to cardiac output. They studied stable dialysis patients and calculated their average Q_a : CO to be 22%. This is the ratio of flow between the access (Q_a) and the CO. A Q_a : CO ratio > 0.3 has been shown in case reports to be a potential risk of developing high-output cardiac failure [14,15].

Management

The initial management is conservative medical management. Definitive treatment options include either limiting the inflow through a banding procedure or sacrifice of the access by ligation. The first priority should be to preserve the hemodialysis access where

possible. Banding is a surgical intervention which involves placing a polytetrafluoroethylene band next to the arterial anastomosis in order to reduce the rate of blood in-flow. If the band is too tight; it causes reduced blood flow and poor dialysis; if is loose, there is no symptomatic improvement [6]. Our patient had a minimally invasive limited ligation endoluminal-assisted revision (MILLER) procedure banding the done to reduce the fistula flow rate. It is a modified form of banding that offers the added advantage of regulating the inflow size through the use of an endoluminal balloon. Usually a size of 4 or 5 mm is chosen [6]. If the patient's symptoms are intractable, ligation of the fistula is the last resort. High output heart failure, though a rare complication of AVF, is an important differential in hemodialysis patients who present with symptoms of congestive heart failure, particularly as these patients often have cardiovascular disease as a co-morbid condition. An accurate diagnosis prevents morbidity and unnecessary hospital admissions.

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