The Arteriovenous Fistula: An Often Overlooked Precipitant of High Output Heart Failure
Titilayo O Ilori*, Jyothi Pinnaka, Mark Kats and Vandana Dua Niyyar
School of Medicine, Emory University, Atlanta, USA

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 Hemodilometry 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...
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 it is loose, there is no symptomatic improvement [6]. Our patient had a minimally invasive limited ligation endoluminal-assisted revision (MILLER) procedure banding the donor 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.

References

1. Sy AO, Plantholt S (1991) Congestive heart failure secondary to an arteriovenous fistula from cardiac catheterization and angioplasty. Cathet Cardiovasc Diagn 23: 136-138.
2. Ozaki K, Kubo T, Hanayama N, Hatada K, Shimagawa H, et al. (2005) High-output heart failure caused by arteriovenous fistula long after nephrectomy. Heart Vessels 20: 236-238.
3. KDOQI (2006) Clinical Practice Guidelines and Clinical Practice Recommendations 2006 Updates Hemodialysis adequacy Peritoneal Dialysis Adequacy Vascular Access. Am J Kidney Dis 48: S1.
4. MacRae JM, Pandeya S, Humen DP, Kirvitski N, Lindsay RM (2004) Arteriovenous fistula-associated high-output cardiac failure: a review of mechanisms. Am J Kidney Dis 43: e17-22.
5. MacRae JM, Levin A, Bilenkii I (2006) The cardiovascular effects of arteriovenous fistulas in chronic kidney disease: a cause for concern? Semin Dial 19: 349-352.
6. MacRae JM, Levin A, Bilenkii I (2006) The cardiovascular effects of arteriovenous fistulas in chronic kidney disease: a cause for concern? Semin Dial 19: 349-352.
7. Goel N, Miller GA, Jotwani MC, Licht J, Schur I, et al. (2006) Minimally Invasive Limited Ligation Endoluminal - assisted Revision (MILLER) for treatment of dialysis access-associated steal syndrome. Kidney International 70: 765-770.
8. Langer S, Paulus N, Koepell TA, Greiner A, Buhl A, et al. (2011) Cardiovascular remodeling during arteriovenous fistula maturation in a rodent uremia model. J Vasc Access 12: 215-223.
9. Iwashima Y, Horio T, Takami Y, Inanaga T, Nishikimi T, et al. (2002) Effects of the creation of arteriovenous fistula for hemodialysis on cardiac function and natriuretic peptide levels in CRF. Am J Kidney Dis 40: 974-982.
10. Begin V, Ethier J, Dumont M, Leblanc M (2002) Prospective evaluation of the intra-access flow of recently created native arteriovenous fistulae. Am J Kidney Dis 40: 1277-1282.
11. Wijnen E, Keuter XH, Planken NR, van der Sande FM, Tordoir JH, et al. (2005) The relation between vascular access flow and different types of vascular access with systemic hemodynamics in hemodialysis patients. Artif Organs 29: 960-964.
12. Yakes WF, Rossi P, Odirik H (1996) How I do it. Arteriovenous malformation management. Cardiovasc Intervent Radiol 19: 65-71.
13. Stern AB, Klemmer PJ (2011) High-output heart failure secondary to arteriovenous fistula. Hemodial Int 15: 104-107.
14. Jin H, Alfonso L, Singh A, Migdal S, Spears JR (2006) Case report: recurrent heart failure with preserved ejection fraction but markedly elevated BNP in a 51-year-old female on hemodialysis with oversized AV fistula. Int J Cardiol 110: 429-430.
15. Chemla ES, Morsy M, Anderson L, Whitmore A (2007) Inflow reduction by distalization of anastomosis treats efficiently high-inflow high-cardiac output vascular access for hemodialysis. Semin Dial 20: 68.