

Case Report High-output Heart Failure Secondary Due to Large Arteriovenous Fistula

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ABSTRACT

Background: Arteriovenous fistula (AVF) creation is a commonly performed procedure for patients who suffered from end-stage renal disease (ESRD) and require a permanent vascular access in order to receive long-term hemodialysis. However, these AVF may have a significant deleterious effect on cardiac hemodynamic functions due to increasing cardiac output (CO) and can lead into high output heart failure. Case Summary: Female, 36 years old complained dyspnea on effort, ascites and lower extremity oedema since 6 months ago. She had history of chronic kidney disease and routinely undergoing hemodialysis with brachiocephalic AVF that enlarged since 2 years ago. Physical examination revealed increased jugular vein pressure, hepatomegaly, ascites and giant draining vein of left with brachiocephalic AVF Nicoladoni-Branham positive sign. Echocardiography examination showed dilated right atrium and right ventricle, left ventricle diastolic D-shaped, normal left ventricular ejection fraction with increase right ventricle CO and cardiac index (CI) 7,8 L/minutes/m², moderate pulmonary regurgitation, severe tricuspid regurgitation (TR) and high probability of pulmonary hypertension. Vascular ultrasound revealed enlarged draining vein with high AVF blood flow rate. Patient then referred to vascular surgeon and decided to undergo ligation of AVF draining vein. After ligation, patient's right heart failure symptoms were improved and had a better quality of life. upon echocardiography control examination revealed significant improvement of left ventricle diastolic Dshaped. Conclusion: High output heart failure is one of a potential serious complication upon creating AVF hemodialysis vascular access. Routine screening of AVF blood flow rate, identifying high risk patients, and early management is very important to prevent irreversible myocardial damage.

Highlights:

1. This case illustrates that high-output heart failure from an excessively flowing AVF—though rare in younger patients—can be dramatically reversed with AVF ligation, highlighting the importance of flow assessment and timely intervention in dialysis patients with unexplained right heart failure.

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Introduction

Arteriovenous fistula (AVF) is the preferred vascular access in dialysis patients given their higher blood flow rates, superior patency, and low incidence of infections compared with either arteriovenous grafts or catheters. However, creation of AVF lead to hemodynamic consequence that led to an increasing cardiac output and development of high output heart failure may be an underappreciated complication in patients who have undergone this procedure. This is mediated by a fall in total peripheral vascular resistance, increased venous return due to blood flow shunt, and increased myocardial contractility secondary to sympathetic nervous system activation. As a result, there is a rise in stroke volume which in time leads to an increase in the right atrial and ventricular pressures. Over time this hemodynamic consequence can lead into heart failure.[1,2]

Incidence of high output heart failure (HOHF) secondary due to AVF is very rare. Patient may present with symptoms of cardiac failure (dyspnea either at rest or with varying degrees of exertion, orthopnea, paroxysmal dyspnea, and edema, either pulmonary and/or peripheral) in the presence of an above-normal cardiac index (> 3.0 L/min/m2) due to high flow shunting from AVF.^[1]

Typically, the AVF is quite large and is likely located in the upper arm, more proximal to the heart. When addressing the problem of HOHF, the nephrologist is faced with the dilemma of preventing progression of heart failure at the expense of loss of vascular access. Nevertheless, treatment should be directed at correcting the underlying problem by surgical banding or ligation of the fistula.^[3]

Case Presentation

A 36-years old female patient come to our hospital with chief complaint shortness of breath during light activity, orthopnea, paroxysmal nocturnal dyspnea, ascites and lower extremity oedema since 6 months ago. She also complaining her left brachiocephalic AVF was enlarged since 2 years ago. She had history of hypertension and chronic kidney disease due to chronic glomerulonephritis from 9 years ago, and routinely undergoing hemodialysis since 6 years ago.

On Physical head examination showed pale conjunctivae with scleral icterus, neck examination showed marked increased jugular venous pressure with positive Lancisi sign. Chest examinations showed enlargement of heart border and pansystolic murmur grade 4/6 at left lower sternal border. Abdominal examinations showed hepatic



enlargement with positive hepatojugular reflex, positive shifting dullness test. On Extremity examination we found a large ectatic left brachiocephalic AVF with positive Nicoladoni-Branham sign (Fig. 1) and pitting oedema at bilateral lower extremity.



Figure 1. Large aneurysmatic left brachiocephalic arteriovenous fistula

Electrocardiography (ECG) examination upon admission showed sinus rhythm with right axis deviation and incomplete right bundle branch block. Laboratory examination showed low level of haemoglobin 9,4 mg/dl, thrombocytopenia 141.000 mg/dl, abnormal renal function with creatinine 5,4 mg/dl and hyperbilirubinemia. Chest X-Ray showed enlargement of the heart. Transthoracic echocardiography showed dilated right atrium and ventricle, left ventricle D-shaped and paradoxical IVS movement, left ventricular ejection fraction 67% with increase right ventricle CO 10,54 L/minutes and cardiac index (CI) 7,8 L/minutes/m², moderate pulmonary regurgitation, severe tricuspid regurgitation (TR), high probability of pulmonary hypertension and decreased right ventricular contraction with tricuspid annular plane systolic excursion 1.5 cm. (Fig. 2). Arteriovenous fistula Doppler ultrasound revealed enlargement of draining vein with diameter up to 3 cm, no thrombus was seen in upper extremity draining vein and feeding artery. High AVF blood flow rate (Qa) 4.2 L/minutes and Qa/CO ratio 0.4 (Fig. 3).



Figure 2. Dilated right atrium and right ventricle with high probability of PH



After discussion with patient and multidiscipline team involving cardiologist, nephrologist and vascular surgeon, decision to do AVF ligation procedure and temporarily change of the hemodialysis vascular access into central venous catheter was made. After AVF ligation, there was a

significant improvement in patient's heart failure symptoms and quality of life including her daily physical activity and upon transthoracic echocardiography control examination revealed significant improvement of left ventricular diastolic D-shaped (Fig. 4).



Figure 3. High flow AVF in feeding artery and large dilated draining vein



Figure 4. Improvement of RV diastolic pressure before AVF ligation (A) and after AVF Ligation (B)



Discussion

The incidence of AVF-associated HOHF is poorly defined. Risk factors for AVF-associated HOHF include upper arm AVF, male sex, history of vascular access surgery, and vascular access blood flow (Qa) >2.0 L/ min The finding of Qa/CO ratios >0.3 has also been suggested as a risk factor for high-output heart failure and decompensation. Brachiocephalic fistulas can have double the flow of radiocephalic fistulas, given the former's proximity to the heart.^[4]

Patients with HOHF present with signs and symptoms of heart failure, such as dyspnea, orthopnea, paroxysmal nocturnal dyspnea, reduced exercise tolerance, peripheral edema, and fatigue. In contrast to low- or normal-output heart failure, manifestations of HOHF may include a wide pulse pressure, warm extremities, and a hyperdynamic precordium, along with a systolic murmur secondary to a high-flow state. Patients with HOHF from AVF will additionally have a history of end stage renal disease and an AVF on 1 of their extremities, usually the upper arm, that is large and aneurysmal. The Nicoladoni-Branham sign is the phenomenon in which temporary occlusion of the high-flow AVF may lead to a modest reduction in heart rate (~7 bpm) via the vagus nerve-mediated baroreceptor reflex.^[4,5]

The AVF should be examined at every visit. The presence of a large, distended fistula with very

strong thrill is suspicious for high blood flow and should prompt a quantitative evaluation, particularly in the presence of heart failure signs and symptoms. Patients also should be followed for signs and symptoms of heart failure as a routine part of every visit to determine whether HOHF is present. An echocardiogram should be obtained when any new symptoms or signs suggestive of dysfunction cardiac develop, and follow-up echocardiography 3-6 months after creation of the AVF is also recommended. Echocardiographic findings suggesting the development of HOHF include dilation of the inferior vena cava, new right ventricular dilation or dysfunction and increasing estimated pulmonary artery pressures.^[6,7]

In patients with HOHF secondary due to AVF, management begins with control of volume status with dialysis and diuretics, correction of anaemia, treatment of hypertension and pharmacological management of heart failure. If the patient has more than one arteriovenous access, one should be closed immediately if it is thought to be contributing, with preservation of the shunt with the best blood flow. The patient's clinical status and symptom should then be reassessed.^[6,8]

Reduce blood flow of the AVF as close as possible to minimum volume flow necessary for adequate dialysis (600 ml/min). Several different surgical techniques have been used to reduce AVF flow. The goal of surgery is to reduce fistula blood flow



while maintaining sufficient flow for adequate dialysis. These techniques have included access banding and distalisation of the anastomosis to a smaller artery. If refractory heart failure persists, the AVF should be occluded and replaced with a tunnelled catheter or small graft since the resistance is higher in grafts than greatly dilated fistulas. Peritoneal dialysis may also be an option among some patients.^[7,8]

Conclusion

The presence of AVF in ESRD patients carries a significant impact on cardiac functions. It can precipitate HF decompensation and lead into HOHF in the short term or long term. Routine screening of AVF blood flow rate and Echocardiogram, identifying high risk patients, and early management is very important to prevent irreversible myocardial damage.

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