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Case Report

The Killer Inside Me: A Large Arterio-Venous Fistula, Meant to Save My Life but Can Kill Me with High-Output Cardiac Failure

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Abstract

The author discussed a case of high-flow fistula in hemodialysis patient and its effect on the heart. High-flow AVF is a common clinical entity in hemodialysis patients, and no doubt contributes to late cardiovascular problems with escalation of pre-mature death. Optimization of flow in these vital conduits are important to prevent pre-mature death related to cardiovascular dysfunction. Prospective trials to investigate the relationship of volumetric flow in AVFs to cardiac dysfunction to mitigate and prevent these impediments are desperately needed.

Keywords: High-Output Cardiac Failure; High-Flow Fistula; Arterio-Venous Fistula; Pulmonary Hypertension; Left Ventricular Hypertrophy

Case History

The patient is 45-year-old African American female on maintenance hemodialysis through an arterial-venous fistula (AVF) in the left upper arm for the last 3 years. She came to Toledo Vascular Access Center (TVAC) with infiltration and low urea reduction ratio (URR) despite high blood flow through the fistula. On examination, she is well-nourished middle-aged female with blood pressure of 146/90 mm Hg, a heart rate of 102 beats per minute and S3 gallop. The ECG tracing showed trigeminy but otherwise sinus rhythm. Respiratory and abdominal examination were within normal limits. Doppler ultra-sound of the shunt showed that she has left upper arm brachial-cephalic AVF with a fistula diameter of 2.5 cm near its origin and located at 2 mm depth from the skin surface. The Doppler ultra-sound also showed that the flow through the fistula is 3300 ml/min. The Nicoladoni-Branham maneuver was positive (decrease in pulse rate from 104 to 77 beats per minute and rise in the blood pressure to 157/100 mmHg) on exerting sustained pressure on the arterial anastomosis of the AVF for 60 seconds. This sign indicates that the AVF is producing cardiac volume overload [1]. Echocardiography revealed left ventricular dilation and hypertrophy along with an estimated cardiac output of 7.3 L/min. She underwent a fistulogram at the TVAC which showed a mega-fistula with relative stenosis in the outflow vein at the cephalic arch levels and evidence of pulmonary hypertension manifested by reflux of the contrast into the internal jugular venous system on systole. She has cardiomegaly but clear lung fields.

Discussion

The best hemodialysis access is native arterio-venous fistula (AVF), which should have just enough blood flow to maintain dialysis efficiency and by the same token prevent access thrombosis. The arbitrary guidelines for a blood flow in a typical AVF are (600-1500ml/min) [2].

The proposed criteria for high-flow AVF are [3,4]:

- AVF blood flow of >2.2L/min
- Hypertrophy of the feeding artery with increased blood flow
- Cardio-pulmonary recirculation of >20%

- High-output cardiac failure with cardiac output >4-8L/min
- Cardiac index>3L/M2

High-flow AVF is plagued by many long-term complications including, steal syndrome affecting the limbs, aneurysms with inherited consequences of ruptures and bleeding, high-output cardiac failure, pulmonary hypertension and cardiomyopathy [2,5,6]. Within two weeks of AVF creation, blood volume increases, leading to greater venous return and increased right atrial, pulmonary artery, and left ventricular end-diastolic pressure [7]. Both plasma atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) concentrations increase after AVF creation, peaking 10 days after AVF placement [8].

The long-term outcome of autologous AVF remains poor, contributing to patient morbidity and immense economic burden. In a recent large series of AVF analysis, the primary failure rate was found to be 23%. And at one year 40% of patients either had a failed access or required at least one intervention to maintain AVF patency [9].

The failure rate in lower arm fistula are more marked than upper arm fistula [10]. Upper arm fistula especially brachial-cephalic AVF (BCAVF) have many long-term complications, including high-flow rates, and greater incidence of cephalic arch stenosis (CAS) compared with lower arm fistula [11,12].

The diagnosis of high-flow AVF is difficult and needs a high index of suspicion along with measurement of volumetric AVF flow and cardiac output [3]. The volumetric flow of the AVF should be carried out at the feeding brachial artery, as it is reproducible, easy to image the artery which has less blood turbulence compared to doppler flow measurement throughout the venous conduit [13].

High-output cardiac failure can be diagnosed with transthoracic echocardiography but may require right heart catheterization [14]. Nicoladoni-Branham maneuver is a simple bed-side test that can aid in the diagnosis of high-output cardiac failure. Nonetheless, the diagnosis must be confirmed by echocardiography or right heart catheterization to prevent cardiovascular complications of high-flow AVF [14,15].

Flows in the AVF as great as 2,000ml/min may predict CAS [16,17]. The consequence of which would be high pressure within the AVF with development of aneurysms and eventually access thrombosis. This is the results of the interplay of high flow exerting high wall shear stress resulting into development of neo-intimal hyperplasia with enhancement of inflammatory mediators and endothelial dysfunction. The final consequences of these interchanges are the progress and advancement of CAS [18-21]. Moreover, the interaction of biological factors and outward physical remodeling would end up in the emergence of the high-flow AVF [3]. The combination of decrease cardiac afterload secondary to decrease peripheral resistance and increased sympathetic activity cause increase in cardiac output [22-24].

Cardiovascular disease remains number one killer in patients with end-stage renal disease on hemodialysis (ESRD/HD). Patients with ESRD/HD have both traditional and non-traditional risk factors for cardio-vascular disease e.g. diabetes, peripheral vascular disease, hypertension, increased inflammatory markers, abnormal lipoprotein B, secondary hyperparathyroidism, anemia, homocystinemia, and endothelial dysfunction, that increase the risk of death and cardiovascular risks in these patients [25]. The peril of worsening heart failure is directly proportional to the flow of the arterio-venous access and is greater with worse pre-existing cardiac function. This relationship between high flow in the AVF and cardiac output leading to late complications like high-output cardiac failure, pulmonary hypertension and cardiomyopathy with increased left ventricular end-diastolic volume. These tangible cardio-vascular snags of high-output AVF and cardiac complications should be approached with great care [26-31].

Any patient with evidence of left ventricular hypertrophy or congestive heart failure should have their vascular access flow measured. Access flow can be measured monthly using the transonic hemodialysis monitor, HDO2 Transonic Systems Incorporated, Ithaca, NY, USA [31]. The transonic device provides an important information regarding AVF blood flow and cardiac output. If the flow through the AVF exceeds >2L/min with cardiac output of >5L/min, the ratio of access flow to cardiac output should be measured and if the ratio is >25%, this may indicate increased risk of high-output cardiac failure [27,28,31].

Banding or surgical intervention to decrease the inflow to outflow ratio have been shown to be an effective method to decrease flow and treat steal syndrome in high-flow AVF [32-34].

In conclusion: high-flow AVF is a common clinical entity in hemodialysis patients, and no doubt contributes to late cardiovascular problems with escalation of pre-mature death. Optimizing flow in these vital conduits to prevent pre-mature death related to cardiovascular dysfunction merit prospective trials to investigate the relationship of volumetric flow in AVFs to cardiac dysfunction and put forward measures to mitigate and prevent these impediments.

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