Challenges and Management of High-Flow Arteriovenous Fistulae

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Summary: The Kidney Dialysis Outcomes Quality Initiative and Fistula First Initiative have embraced the arteriovenous fistula as the gold standard for hemodialysis accesses. Despite this status, fistulae are fraught with complex issues ranging from a high primary failure rate to high flow resulting in increased cardiovascular morbidity. It is important not to overlook the insidious peril of a hyperfunctioning access that may actively promote cardiac overload, cardiopulmonary recirculation, rapid access growth with aneurysm enlargement, recurrent venous stenosis resulting in access failure, and inflow/outflow mismatch. Once recognized, flow can and should be reduced to mitigate these and other negative effects.
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The hemodialysis access has made it possible for chronic kidney failure patients to receive long-term outpatient treatment.[1,2] Although complications of clotted catheters and infected expanded polytetrafluoroethylene grafts overshadow the day-to-day workings of the hemodialysis unit, a high-flow access likely will go unnoticed. It is not until complaints of Dialysis Associated Steal Syndrome (DASS),[3] or an aneurysm has enlarged to the point of eventual rupture, that notice is taken. Too often, repeat episodes of congestive heart failure are considered to be cardiac and go unrecognized as a problem related to a hyperfunctioning access.

The ideal hemodialysis access functions with just enough flow to prevent thrombosis while maximizing dialysis efficiency. A useful, although arbitrary, guideline for ranges of blood flow within a typical dialysis access are the following: low (600 mL/min), normal (600-1500 mL/min), and high (1500-4000 mL/min) categories.[4] Flow-related problems are patient specific and mostly are unrecognized because there is very little correlation with symptoms. A low-flow access can cause both DASS and cardiac overload, depending on the degree of pre-existing systemic vascular disease and cardiac dysfunction. Conversely, a high-flow access causes neither DASS nor cardiac overload symptoms.[3,5]

Thus far, proposed treatments are based entirely on clinical symptoms rather than attempts to normalize access flow. A lack of prospective data to support flow reduction and the fear of access loss resulting from intervening on an otherwise, well-functioning access, compounds the resistance to address high-flow accesses. Retrospective data and numerous anecdotes suggest a significant benefit in proactive management. This review focuses on the negative effects of high-flow accesses and techniques to normalize flow.

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CARDIAC OVERLOAD

High-output cardiac failure secondary to an arteriovenous fistula (AVF) is a well-described phenomenon in both dialysis and nondialysis patients. In a young nondialysis patient with a symptomatic arteriovenous malformation there is urgency to repair the defect. In a young dialysis patient with an upper-arm AVF, we tend to tolerate an access with 2 to 4 L/min flow without much thought about its deleterious effects. In nearly all instances of access flow measurements, the results are obtained at rest. Cardiac output can double or even triple with exertion, the shunt could worsen to as much as 10 to 12 L/min while climbing a flight of stairs.[6,7]

After creation of an AVF, many patients experience an instantaneous decrease in peripheral vascular resistance that results in a compensatory increased cardiac output. Cardiac stress of the AVF causes a variety of changes including decreased subendocardial viability index, diastolic dysfunction, congestive heart failure, pulmonary hypertension, and, finally cardiomyopathy, with four-chamber cardiac enlargement. The hemodynamic effects of an AVF can result in the activation of the sympathetic nervous system, increasing pulse rate, stroke volume, and contractility. These conditions can lead to hypertrophy of the left ventricle (LV).[8] An enlarged LV consequently decreases the LV diastolic pressure and thus decreases the subendocardial viability index. This would indicate that the patient's coronary perfusion is not proportional to the work done by the heart.[5,9-11] More importantly, it is a sign of subendocardial ischemia and resultant fibrosis.[2]

Another concern regarding AVFs is the urea reduction ratio (URR), which all hemodialysis units monitor. A low URR in an otherwise well-functioning access is another simple method to determine if an access has pathologically high flow. To understand how to use this tool, one

must first understand cardiopulmonary recirculation; first described in 1992 by Schneiditz et al.[12] During a dialysis treatment, fistula flow returns a high percentage of cleaned blood to the right heart and pulmonary circulation. With the next stroke volume, a percentage of the freshly dialyzed blood will be returned immediately to the fistula. Freshly cleaned blood re-entering the fistula will be cleaned again, but this time without significant urea removal. Upper-arm fistulas flowing at nearly 2 L/min will return blood to the heart such that 30% of the next stroke volume and cardiac output represents freshly cleaned blood. Therefore, a higher fistula flow will result in lower dialysis efficiency and a low URR.[12-17] For this reason, as well as others, we must always try to explain why a patient has a low URR.

Skepticism surrounding the cardiac overload theory stems from a lack of readily measurable and tangible criteria. The aforementioned low URR in an otherwise high-flow AVF is a good test but there is limited knowledge of cardiopulmonary recirculation and its affect on URR. Nevertheless, a simple test can help determine if an AVF is a cardiac stressor. The Nicoladoni–Branham sign can be elicited by brief (30-60 se) manual compression of the fistula at the arterial anastomosis. The response to this diagnostic maneuver is a decrease in pulse rate and an increase in blood pressure, which occurs as a result of instantaneous normalization of circulating blood flow by occluding the fistula shunt. Release of the inflow will result immediately in an increase in heart rate within about 15 seconds.[18] This test is easily reproducible and requires no special equipment.

RAPID ACCESS AND ANEURYSM GROWTH

Cardiac output is the driving force maintaining both flow and pressure within a hemodialysis access. However, the pressure within the access is difficult to predict at any given point because it is dependent on variable compliance and cross-section. Static pressures within the access will increase as a venous stenosis impedes flow. Fistulae may be viewed as part cylinder, so the Law of Laplace can predict wall tension, as follows:

$$T = \frac{pr}{2t},$$

where T = wall tension, p = pressure, r = radius, and t = wall thickness.[19,20] For a given pressure, an increased radius requires an increased wall thickness to accommodate a stable wall tension. Furthermore, an increased pressure requires an increased thickness to maintain stable wall tension.

In some instances, high flow results in lumen sclerosis; in other instances, high flow results in lumen dilation. Extremes of high flow will cause rapid dilation and formation of the mega-fistula, as seen in this 32-year-old woman with a 4-year-old fistula (Fig. 1). In the case of an AVF, aneurysmal dilation occurs as the result of a complex interplay between biologic factors that induce outward wall remodeling and physical factors such as wall tension that is directly proportional to intra-access pressures. Segments of the vascular wall not exposed to needle injury tend to dilate uniformly and maintain wall thickness as the access develops. In most cases, wall thickness improves over time and makes the AVF durable. These areas represent true aneurysms of the venous system with intact layers of intima, media, and adventitia. Repeat needle injury to the cannulation zone of an AVF results in a cycle of tissue injury and healing, which, under hypervolemic pressure, result in dilation of the vascular wall. Frequent injury to the vascular wall replaces healthy tissue with scar formation and the effect of biologic factors contained within the normal tissue is diminished. These pseudoaneurysms will continue to grow, but frequently are unable to remodel properly and maintain wall thickness. In short, a pseudoaneurysm develops and becomes susceptible to rupture.[21] Moderation of a high-flow system should diminish the pressure sufficiently to reduce the wall stress. In a series of patients who underwent Minimally Invasive Limited Ligation Endoluminal-assisted Revision (MILLER) banding owing to rapid aneurysm growth, pressure within the mid-fistula was reduced by 60% to 80% and aneurysm growth was arrested.[22]

PERIPHERAL AND CENTRAL VENOUS STENOSIS

When the balance between biologic and physical factors favors lumen sclerosis rather than dilation, peripheral and central venous stenosis will develop and threaten access viability. Examples include brachiocephalic and brachiobasilic AVFs, which preferentially develop cephalic arch and central vein stenosis, respectively, but not exclusively. Although AVFs generally require fewer interventions per access year than arteriovenous grafts, a few sites of venous stenosis account for a large portion of repeat interventions.[23] A particularly troublesome site within brachiocephalic AVFs is the cephalic arch.[24,25] Of all brachiocephalic fistula failures, 19.5% to 77% of these cases are attributed to cephalic arch stenosis (CAS).[26-28] In a cohort of radiocephalic and brachiocephalic AVFs, the incidence of CAS was observed to be 15%. More importantly, brachiocephalic AVFs are 37 times more likely to contain CAS than radiocephalic AVFs.[27] This disparity in the prevalence of CAS between brachiocephalic and radiocephalic fistulae likely is accounted for by differing hemodynamics at the cephalic arch.[29]

The central veins are also very susceptible to flow-limiting stenosis after creation of an AVF. Brachiobasilic AVFs have a higher rate of central stenosis than radiocephalic AVFs.[30] Clinically asymptomatic before the integration of a vascular access, central venous stenosis can become symptomatic when high flow is introduced to the circulatory system. This eventually may result in significant arm swelling, cyanosis, and collateral vein formation on chest walls. Traditionally, central stenoses result

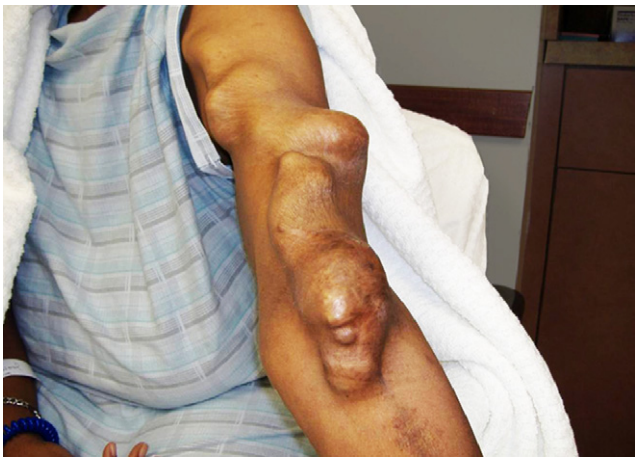

Figure 1. Example of a mega-fistula.

from previous subclavian catheters,[31-33] but central vein stenosis occurs in the absence of any previous central catheters. In a study that evaluated 133 dysfunctional accesses, 41% had evidence of a significant subclavian vein stenosis despite no previous subclavian vein catheterization.[34-36] From this finding one can conclude the stenosis forms as a result of the presence of fistula flow rather than trauma from catheterization injury.

High flow within the venous outflow of an AV access is correlated with intimal hyperplasia. Various reasons for venous intimal hyperplasia have been proposed; however, the most likely cause of intimal hyperplasia is intimal injury as a result of turbulence,[37] and has a direct correlation with hypervolemic flow.[29,38] Therefore, such a trigger for intimal hyperplasia should be mitigated by reducing flow within the AVF.[39] In the study by Miller et al,[36] a retrospective analysis of patients who had flow reduction using the MILLER banding procedure showed a clinically significant reduction in the need for interventions at the cephalic arch. A total of 33 patients requiring 2 or more cephalic arch interventions within 3 months and had flow reduction were followed up.

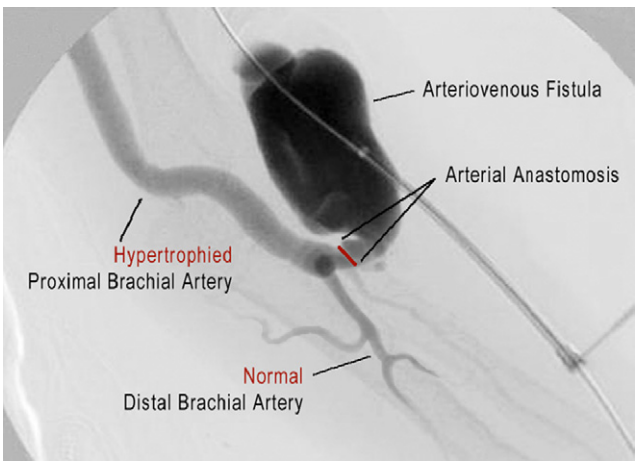

Figure 2. Large arterial anastomosis, low-resistance arteriovenous fistula, and a hypertrophied proximal brachial artery are characteristic of high-flow fistulae.

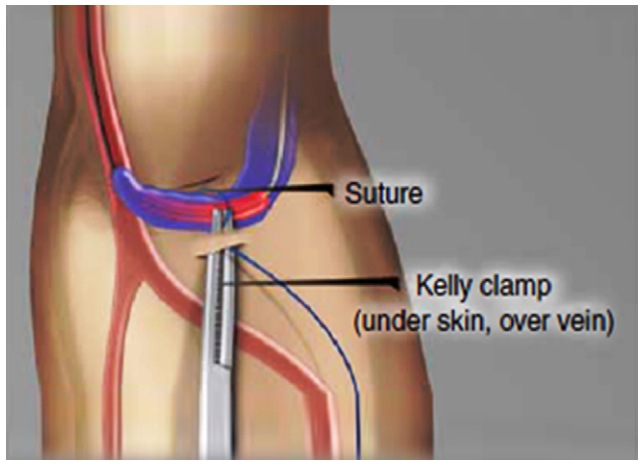

Figure 3. Tunneling a suture around a high-flow arteriovenous fistula using the MILLER banding technique.

Over an average follow-up period of 14 months, the intervention rate was reduced from 3.34 to 0.9 interventions per access year, after the flow reduction. In addition, the 3-, 6-, and 12-month cephalic arch primary patency rates were 91%, 76%, and 57%, respectively. The literature frequently reports the primary patency of the cephalic arch after angioplasty as 76%, 42%, and 23% at 3, 6, and 12 months, respectively.[40-44]

INFLOW/OUTFLOW MISMATCH

As previously mentioned, treatments must be individualized because there is little correlation between access flow and clinical symptoms of excessive flow. This mismatch between the inflow and outflow occurs when the carrying capacity of the outflow veins is insufficient to handle the inflow. The result is the development of backpressure, which causes significant access dysfunction and arm swelling. To resolve the backpressure, decreasing the inflow to less than 1 L/min can restore balance. This allows the collateral veins to handle the flow without expressing symptoms.[3,22]

In the case of occluded central veins, some patients will develop a swollen arm and others will be completely asymptomatic. When the central veins remain occluded despite attempted interventions, a flow-reducing band can alleviate symptoms. Jennings et al[45] applied the MILLER banding technique to 22 centrally occluded patients presenting with swollen extremities. The mean access flow of 1,640 mL/min before banding decreased to 820 mL/min after banding ($P < .01$). In 20 of the patients, swelling resolved promptly; in the other 2 patients, swelling markedly improved. Three patients had aneurysm repair with simultaneous inflow banding and decreased intra-access pressure after flow restriction. Two fistulas failed at 8 and 13 months. The mean follow-up period was 8 months. This study highlights that the ideal access is one that has just enough flow to maintain patency while providing enough flow for adequate clearance of solute and fluid.[45]

FLOW REDUCTION

High flow within a fistula system is the result of hypertrophy of the arterial system (Fig. 2) combined with a low-resistance venous outflow. The carrying capacity of an artery is dependent on the radius to the fourth power and, therefore, a 1-mm increase in lumen diameter will result in a doubling of the flow-carrying capacity. Once the arterial hypertrophy occurs, it is irreversible. Similarly, once the vein hypertrophies with aneurysmal dilation, it is irreversible unless surgically resected. Ultimately, short of access ligation, the only remaining treatment to reign in a pathologically shunting system is to apply resistance to the system.

BANDING

The introduction of a high-resistance band is a reasonable treatment for a low-resistance venous pathway, which transforms a functional access into a pathologic shunt. Banding physiology is best explained by Poiseuille's Law, which states that fluid flow (Q) is proportional to radius (r), pressure across a gradient (Delta Pressure, for example, arterial pressure-central venous pressure), and inversely proportional to resistances, length (l), and viscosity (η):

$$Q = \frac{\Delta P \pi r^4}{8 \eta l}.$$

Banding techniques decrease flow by decreasing the radius at a specific point and, as a result, access flow (Q_{access}), and pressure is directly sacrificed to increase distal arterial flow (Q_{distal}) and pressure.

Historically, banding of the access inflow has had limitations. Bands that are too tight cause poor dialysis efficiency or thrombosis of the access. Conversely, bands that are too loose do not alleviate symptoms.[46-49] The MILLER banding technique overcomes the inherent difficulties of sizing associated with banding by using a 3- or 4-mm diameter intraluminal balloon as a sizing dowel. Once the subcutaneous 2.0 Prolene suture (Ethicon, Menlo Park, CA) is tied around the outside of the vessel, the intraluminal balloon ensures the end lumen diameter is precise and predictable. Bands that are too tight can be stretched or even broken using angioplasty technique. If a band is too loose, the procedure easily is repeated because it is minimally invasive (Fig. 3). It can be performed easily and safely in an outpatient setting with good outcomes and minimal associated morbidity.

Miller et al[22] published a study of 183 patients treated for steal and high flow using the MILLER banding technique. A total of 114 patients presented with hand ischemia (steal) and 69 patients presented with clinical manifestations of pathologic high access flow such as congestive heart failure. Overall, 183 patients underwent a combined 229 bandings with technical success achieved in 225. Complete symptomatic relief (clinical success) was attained in 109 steal patients and in all high-flow patients. The average follow-up time was 11 months, with a 6-month primary band patency of 75% and 85% for steal and high-flow patients, respectively. At 24 months the secondary access patency was 90% and the thrombotic event rates for upper-arm fistulas, forearm fistulas, and grafts were 0.21, 0.10, and 0.92 per access year, respectively.[22]

The MILLER banding technique effectively treats high-flow AVFs showing both cardiac overload and steal symptoms because it adds resistance into the system and decreases total circuit blood flow. However, in patients who have low-flow accesses and show steal symptoms, slowing down the access would result in access thrombosis. Therefore, treatments that increase total circuit blood flow, such as the Distal Revascularization-Interval Ligation and proximalization,[3,46,50] are more appropriate procedures.

REVISION USING DISTAL INFLOW

In a few instances, banding may not be the best option, such as AVFs with a greater than 20-mm peri-anastomotic area. In these cases, a piece of vein, or expanded polytetrafluoroethylene, can be used to move the inflow to a more distal location. The revision using distal inflow (RUDI) technique involves ligation of the fistula at its origin followed by re-establishment of the fistula via bypass from a more distal arterial source to the venous limb of the AVF. Its design adds resistance to the system because it uses a smaller distal artery as inflow and lengthens the fistula with a smaller-diameter bypass. Nevertheless, revascularization surgeries are complex and met with various degrees of success.[3,51] RUDI is not commonly performed because of several reports of limited success.

CONCLUSIONS

Although the AVF has been a well-established access solution for end-stage renal disease patients on hemodialysis, high flow from overfunctioning AVFs can cause a variety of problems that often go unnoticed. Newly developed AVFs can increase stress on the circulatory system and result in heart chamber enlargement and cardiac overload. This high flow also can trigger a variety of biologic factors that cause uneven growth of the access, which can lead to the development of fragile aneurysms. Furthermore, AVF-related high flow has been shown to induce venous stenosis, especially of the cephalic arch. Finally, high-flow AVFs can display a significant inflow rate disproportional to their increased outflow rate. This inflow-outflow mismatch causes severe backpressure that becomes most notable in patients with central venous occlusions resulting in arm swelling.

Nevertheless, in hopes of preserving AVFs, two main techniques have been developed to alleviate high flow. The MILLER banding technique takes a less-rigorous approach by simply banding down a segment of the AVF to slow down its internal blood flow. This minimally

invasive procedure can be performed multiple times until the desired results are achieved. On the other hand, the RUDI technique favors larger AVFs with high flow. In this surgical procedure, the fistula is ligated next to the anastomosis and then reconnected to the venous outflow from a more distal arterial source. Unfortunately, this complex procedure usually is avoided because its success has been less predictable.

Both the MILLER banding and RUDI techniques treat high-flow AVFs by revising the anastomosis and introducing resistance into the circuit. The result is to decrease total extremity flow and cardiac output. With advancing knowledge of AVFs, we recognize that untreated high flow can lead to detrimental effects. Further prospective research should be directed toward resolving high-flow AVFs so we can proactively treat these accesses rather than wait for patients to have irreversible effects.

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