Challenges and Management of High-Flow AVFs

Percutaneous flow reduction to manage sequelae of high-flow arteriovenous fistulas.

BY GREGG A. MILLER, MD

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 is as follows: low (600 mL/min), normal (600–1,500 mL/min), and high (1,500–4,000 mL/min). Flow-related problems are patient-specific and are mostly unrecognized because there is very little correlation with symptoms. A low-flow access can cause both dialysis access steal syndrome and cardiac overload, depending on the degree of preexisting systemic vascular disease and cardiac dysfunction. Conversely, a high-flow access may cause neither dialysis-associated steal syndrome nor cardiac overload symptoms.[1]

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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 of 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 when climbing a flight of stairs.

CARDIOPULMONARY RECIRCULATION

A low urea reduction ratio in an otherwise "well-functioning access" is a simple measurement to determine if an access has pathologically high flow. To understand how to use this tool, one must understand cardiopulmonary recirculation, as first described in 1992 by Schneditz et al. During 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 immediately be returned to the fistula. Freshly cleaned blood reentering 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 urea reduction ratio.[2-4]

RAPID ACCESS AND ANEURYSM GROWTH

Cardiac output is the driving force maintaining both flow and pressure within a hemodialysis access. 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 a "mega-fistula" (Figure 1). In the case of an AVF, aneurysmal dilation occurs due to a complex interplay between biologic factors, which induce outward wall remodeling, and physical factors such as wall tension, which is directly proportional to intra-access pressures.

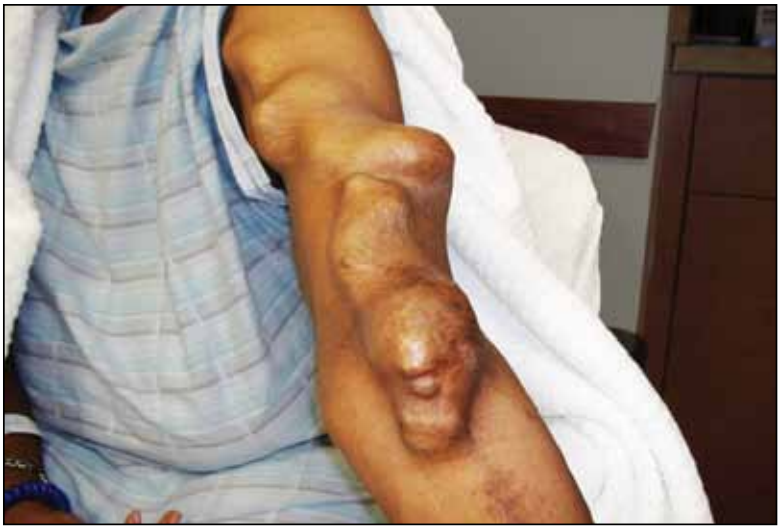

Figure 1. Example of a "mega-fistula" as seen in this 32-year-old woman with a 4-year-old fistula.

Segments of the vascular wall that are 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, results 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 are frequently unable to remodel properly and maintain wall thickness. In short, a pseudoaneurysm develops and becomes susceptible to rupture. Moderation of a high-flow system should diminish the pressure sufficiently to reduce wall stress. In a series of patients who underwent minimally invasive limited ligation endoluminal-assisted revision (MILLER) banding to manage rapid aneurysm growth, pressure within the midfistula was reduced by 60% to 80%, and aneurysm growth was arrested.[5,6]

PERIPHERAL AND CENTRAL VENOUS STENOSIS

Clinically asymptomatic before the integration of a vascular access, central and peripheral venous stenosis can become symptomatic when high flow is introduced to the circulatory system. This may eventually result in significant arm swelling and intra-access hypertension. Traditionally, central stenoses result from previous subclavian catheters, but central vein stenosis may occur 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. From this finding, one can conclude that the stenosis forms due to the presence of hypervolemic, turbulent fistula flow. Moreover, brachiocephalic AVFs are 37 times more likely to contain cephalic arch stenosis than radiocephalic AVFs. This disparity in the prevalence of cephalic arch stenosis between brachio- and radiocephalic fistulas is likely accounted for by differing hemodynamics at the cephalic arch.[7,8]

High flow within the venous outflow of an arteriovenous 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 due to turbulence, which has a direct correlation with hypervolemic flow. Such a trigger for intimal hyperplasia should be mitigated by reducing flow within the AVF.

In the study by Miller et al, a retrospective analysis of patients who had flow reduction using the MILLER banding procedure demonstrated a clinically significant reduction in the need for interventions at the cephalic arch. A total of 33 patients requiring two or more cephalic arch interventions within 3 months and had flow reduction were followed. During an average follow-up of 14 months, the intervention rate was reduced from 3.34 to 0.9 interventions per access-year following the flow reduction. Additionally, the 3-, 6-, and 12-month cephalic arch primary patency rates were 91%, 76%, and 57%, respectively. The literature reports the primary patency rate of the cephalic arch after angioplasty as 76%, 42%, and 23% at 3, 6, and 12 months, respectively.[9]

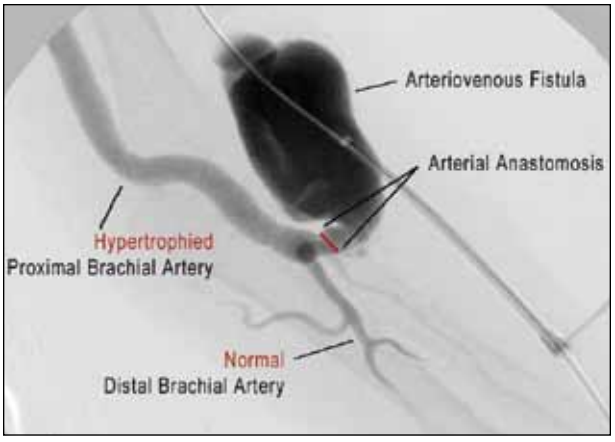

Figure 2. A large arterial anastomosis, low-resistance AVF, and a hypertrophied proximal brachial artery are characteristic of high-flow fistulas.

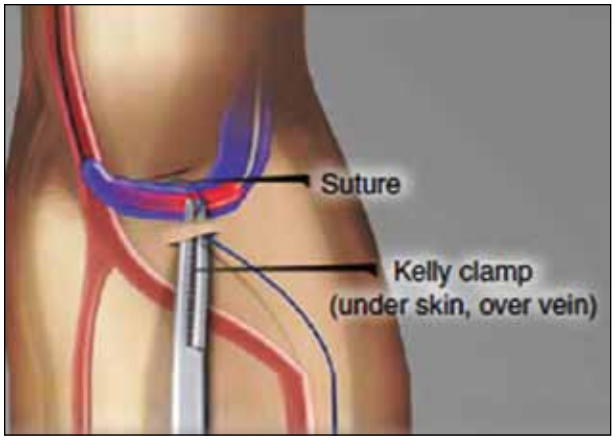

Figure 3. Tunneling a suture around a high-flow AVF using the MILLER banding technique.

INFLOW/OUTFLOW MISMATCH

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. In the setting of central vein stenosis or occlusion, the resultant backpressure causes significant access dysfunction, frequently manifesting as arm swelling. To resolve the backpressure, lowering the inflow below 1 L/min can restore balance. This also allows the collateral veins to handle the flow without expressing symptoms.

When the central veins remain occluded despite attempted interventions, a flow-reducing band can alleviate symptoms. Jennings et al applied the MILLER banding technique to 22 centrally occluded patients presenting with swollen extremities. Mean access flow of 1,640 mL/min before banding decreased to 820 mL/min after banding ($P < .01$). In 20 patients, swelling resolved completely; in the other two patients, swelling markedly improved.[10]

FLOW REDUCTION

High flow within a fistula system is the result of hypertrophy of the arterial system (Figure 2) combined with a low-resistance venous outflow. The carrying capacity of an artery depends 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 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 has transformed 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 (ΔP) (eg, arterial pressure-central venous pressure) and inversely proportional to resistances, length (L), and viscosity (μ): $\Delta P = \frac{8\mu LQ}{\pi r^4}$. Banding techniques decrease flow by decreasing the radius at a specific point, and as a result, access flow (Qaccess) and pressure are directly sacrificed to increase distal arterial flow (Qdistal) 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. 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 is tied around the outside of the vessel, the intraluminal balloon ensures that the end lumen diameter is precise and predictable. Bands that are too tight can be stretched or even broken using an angioplasty technique. If a band is too loose, the procedure is easily repeated because it is minimally invasive (Figure 3). It can be safely performed in an outpatient setting with good outcomes and minimal associated morbidity.

Miller et al published an article on 183 patients who were treated with the MILLER banding technique for steal syndrome and high flow. One hundred fourteen presented with hand ischemia (steal) and 69 with clini-

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cal 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 achieved in 109 steal patients and in all high-flow patients. The average follow-up time was 11 months, with 6-month primary band patency rates of 75% and 85% for steal and high-flow patients, respectively. At 24 months, the secondary access patency rate was 90%, and the thrombotic event rates for upper arm fistulas, forearm fistulas, and grafts were 0.21, 0.1, and 0.92 per access-year, respectively.[5]

The MILLER banding technique effectively treats patients with high-flow AVFs exhibiting 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 exhibit steal symptoms, slowing down the access would result in access thrombosis. Treatments that increase total circuit blood flow, such as distal revascularization and interval ligation and proximalization, are more appropriate procedures for these cases.

CONCLUSION

Although the AVF has been a well-established access solution for end-stage renal disease patients on hemodialysis, high flow from "over-functioning" 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. High flow can also trigger a variety of biologic factors that cause uneven growth of the access, which can lead to the development of fragile aneurysms. AVF-related high flow has been shown to induce venous stenosis, especially of the cephalic arch. High-flow AVFs can display a significant inflow rate that is disproportional to their outflow carrying capacity. This inflow-outflow "mismatch" causes severe backpressure that becomes most notable in patients with central venous occlusions, resulting in arm swelling.

In hopes of preserving AVFs, the MILLER banding technique will alleviate high flow 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. The MILLER banding technique treats high-flow AVFs by revising the juxta-anastomotic area and introducing resistance into the circuit. The result is a decrease in 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 that we can proactively treat these accesses rather than wait for patients to have irreversible effects. ■

TAKE-HOME POINTS

- Flow-related problems are patient-specific and mostly go unrecognized because there is very little correlation between access flow and clinical symptoms of excessive flow.
- Cardiac output is the driving force maintaining both flow and pressure within a hemodialysis access.
- The MILLER banding technique can be used to effectively treat high-flow AVFs.

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