

Understanding Steal Syndrome: Causes and Prevention

Strategies to prevent the development of significant VASS.

BY SCOTT S. BERMAN, MD, FACS

Vascular access–induced steal syndrome (VASS) is an uncommon but challenging complication that occurs due to a functioning arteriovenous (AV) fistula or graft in 6% of chronic kidney disease patients who require hemodialysis.¹ In the US, this represents approximately 20,000 patients, given that the current incidence of chronic kidney disease (CKD) patients requiring hemodialysis is 350,000.² The clinical presentation of VASS can range from mild symptoms that are limited to the actual hemodialysis session, such as hand pain and coldness, to more severe symptoms, such as digital gangrene and limb threat, which occur independent of the hemodialysis treatment. The timing of VASS also varies between acute presentations coincident with the creation of the AV access to a more indolent, chronic course that declares itself years after access construction. The significant challenge that the treatment of VASS creates comprises resolution of the extremity ischemia while trying to preserve the hemodialysis access for the patient. These two goals are often at hemodynamic odds with each other in any given extremity. Our relatively poor understanding of the pathophysiology of VASS has resulted in a diverse menu of treatment options depending on the etiology of the “steal.” This article reviews the current understanding of the causes and prevention of VASS.

CAUSES OF VASS

When patients present with VASS, they are manifesting an imbalance between the low-resistance outflow of the AV access and the high-resistance outflow in the distal, arterial vasculature of the forearm and hand. This imbalance results in preferential flow through the access at the expense of the forearm and hand. It is generally accepted that after an AV access is created, the normal response is a significant, com-

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pensatory increase in blood flow through the inflow artery. This can amount to a tenfold increase in magnitude and is usually accompanied by dilation of the donor artery (as well as the outflow vein, if the access is autogenous). However, this is somewhat intuitive because the normal mean blood flow through the brachial artery is 73 mL/min, and the minimum blood flow that is required to maintain an AV graft is 600 mL/min.^{3,4} This compensatory arterial dilation is thought to be mediated with the release of nitric oxide from the vascular endothelium of the donor artery in response to high blood flow velocity.⁵ Without this compensatory response from the donor vessel, acceptable access flow would never be realized. Understanding this basic mechanism of VASS allows one to understand some of the underlying causes and risk factors that predispose patients to the complications of limb ischemia.

ARTERIAL INFLOW DISEASE

One of the causes of VASS that is both simple to diagnose and to treat is pathology within the arterial inflow to the AV fistula or graft. In my own experience with a prospective series of patients evaluated for significant VASS, an arterial inflow lesion occurred in a minority of patients (<5%).⁶ The current majority of patients with CKD also harbor risk factors for atherosclerosis, particularly diabetes mellitus.

Obstructive lesions in the subclavian, axillary, or brachial arteries can predispose patients to VASS, simply due to a lack of adequate compensatory flow to the extremity to accommodate both the low-resistance outflow of the access and the high-resistance vascular bed of the forearm and hand. Most arterial stenoses (and even some short segment occlusions of the subclavian, axillary, and brachial artery proximal to an AV access) are readily diagnosed with arterial duplex imaging, computed tomographic angiography, or catheter-directed angiography. Moreover, these lesions are usually amenable to percutaneous revascularization with either angioplasty alone or angioplasty and adjunctive stenting. In the unusual case when percutaneous revascularization fails, surgical options include in-line or extra-anatomic bypass, using either autogenous or prosthetic material. Because these lesions are usually short, durable results are generally seen, regardless of the revascularization technique.

HIGH-ACCESS FLOW

Unlike VASS, which is caused by inflow arterial lesions, ischemia is often caused by high-access flow and has a much more complicated etiology to understand and treat. As alluded to earlier, access flow is determined by the interaction of the resistances associated with the access outflow, distal vascular bed, and collaterals. This interaction has been compared with the electrical resistance array seen in a Whetstone bridge, although actual measurement *in vivo* has not been accomplished to validate the inference.⁷ Many factors inherent to the CKD population contribute to these resistance factors and are hypothesized to play a role in VASS. Just as diabetes is a common factor in CKD, diabetic atherosclerosis (in the form of medial calcinosis) is thought to predispose patients to VASS. The specific mechanism that allows diabetes to function in VASS is unknown; however, it seems reasonable to hypothesize that a lack of vascular adaptation, both in the inflow artery and the collateral beds, as a result of diabetic vascular disease, would cause this. In my published experience with the distal revascularization-interval ligation technique for treating VASS, I have only encountered one patient with significant, treatment-requiring ischemia who did not also harbor diabetes mellitus.⁸ Not only is diabetic vascular disease suspected to predispose patients to VASS, but diabetic neuropathy may somehow play a role in a predilection for significant ischemic symptoms, particularly with pain and motor dysfunction that are often seen in patients manifesting significant ischemia after access construction.

A subgroup of patients with VASS have too much flow going through the AV access. This may be related to an artery-to-vein or artery-to-graft anastomosis that is too large, in which the collateral flow in the extremity is inadequate

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to achieve tissue perfusion distal to the access site. This may also be the result of arterial occlusive disease within the radial and ulnar arteries, affecting compensatory flow around the palmar arch. The resultant preferential diversion of flow through the low-resistance AV access comes at the expense of the high-resistance, diseased, distal arterial bed. Some patients only manifest VASS during the hemodialysis session. In these patients, the etiology is usually related to a drop in systemic blood pressure (and cardiac output) and, therefore, extremity perfusion pressure. Management of this subgroup is often limited to adjustment and/or avoidance of antihypertensive medications on the days the patient receives hemodialysis treatments. Unfortunately, there is a paucity of data published that distinguishes among the various types of VASS; thus, a clear understanding of the effect of each etiology on individual patients is not well understood. Therefore, each patient presenting with VASS needs a thorough evaluation of extremity anatomy and access hemodynamics before a treatment regimen can be instituted.

PREVENTING VASS

Prevention strategies for VASS rely predominantly on an understanding of the various etiologies. First and foremost, excluding arterial inflow disease is a process that should arguably take place before the placement of an AV access, during the preoperative evaluation. As part of the initial physical examination of a CKD patient (before access placement), a careful extremity-arterial evaluation should be conducted, including an examination for the presence and caliber of pulses. In the common scenario of an upper extremity access, this examination comprises palpation of carotid, axillary, brachial, radial, and ulnar pulses. Bilateral brachial blood pressures should also be measured. A gradient of >15 mm Hg among the extremities suggests a significant stenosis or occlusion in the artery proximal to the brachial in the extremity with the lower reading. In some patients, well-developed collateral circulation around a chronic subclavian artery stenosis or occlusion will not be found in the physical examination, therefore masking the arterial inflow pathology without more intensive testing. Thanks to the Fistula First Initiative and the most recent American Kidney Foundation Quality Outcome Initiative, a significant emphasis has been applied to creating autoge-

nous AV fistulae in the majority of CKD patients. This has brought the role of ultrasound-based duplex imaging of extremity arteries and veins to the forefront, which identifies the adequate anatomy that will maximize fistula construction.⁴ Duplex examination of the extremity arterial system provides a cost-effective, noninvasive evaluation of the presence of arterial pathology or variant anatomy that may predispose patients to VASS. The combination of a careful physical examination and duplex imaging should reveal arterial pathology in the majority of patients before access construction. VASS can therefore be avoided by either choosing the unaffected extremity, or by treating the inflow disease before or coincident with the creation of the access. Whether or not arterial duplex imaging should be performed on a routine basis before all AV access construction remains to be determined. However, once VASS has manifested, there is no question that arterial duplex imaging can help determine the inciting mechanism.

If the preoperative assessment fails to identify factors in the extremity arterial system that would predispose the patient to VASS, prevention becomes a more complicated problem. A number of intraoperative approaches have been put forth, either to prevent VASS or to identify patients at risk. One such strategy is to avoid the use of the brachial artery as the inflow vessel for the AV access. By originating the access on the radial artery, even for antecubital accesses, the risk of VASS has been shown to be less than that with brachial artery inflow. It is likely that this contributes to the relatively lower flow through the access provided by the radial artery and, therefore, higher flow in the collateral vessels (ie, ulnar artery) feeding the distal extremity. Another intraoperative approach to limiting the incidence of VASS is keeping the size of the artery-to-vein or artery-to-graft anastomosis <75% of the inflow artery. Interestingly, this rationale was applied to the development of the tapered polytetrafluoroethylene grafts introduced in the late 1980s. Practical application of this theory, however, failed in the routine use of tapered grafts, which have not reliably reduced the incidence of VASS. Intraoperative measurements of digital pressures have been shown in two prospective studies to reliably identify patients at risk for VASS.^{1,9} Using the digital brachial index (DBI), Goff et al identified patients with a DBI of <0.45 as having a significant risk for VASS.¹ Other risk factors identified in both studies included diabetes, female gender, and brachial-based access sites. Intraoperative DBI measurements are somewhat cumbersome and, despite these data, have not been widely applied. A more recent attempt to reduce the incidence of VASS involves the measurement of access flow. With contemporary duplex imaging, access flow can be measured noninvasively with values that correlate with ultrasound dilution methods. Access fistulae and grafts

with flows >2,000 mL/min are certainly at risk for hemodynamic complications. Current technology also allows the measurement of access flow during construction using intraoperative Doppler-based flow probes.¹⁰ My limited experience with this technology has failed to demonstrate a correlation with high-flow fistulae and the subsequent development of VASS in the immediate postoperative period.

CONCLUSION

VASS is an uncommon yet devastating complication of AV access fistulae and grafts for hemodialysis. Significant VASS develops in 6% of patients undergoing upper extremity hemodialysis access surgery and requires intervention to reverse the ischemia, often at the expense of the access site. Risk factors for the development of significant VASS include use of the brachial artery as inflow, diabetes mellitus, female gender, and underlying upper extremity arterial insufficiency. Intraoperative measurement of digital-brachial indices at the time of access surgery has been shown to further identify patients at risk for significant VASS but is cumbersome to perform and has not been widely adapted. Strategies to prevent the development of significant VASS include use of the radial artery to access inflow in forearm fistulae and grafts, use of the high-axillary artery as inflow for upper arm access grafts, and detailed arterial imaging prior to access surgery to identify and treat significant occlusive disease in the upper extremity arterial vasculature. Despite these efforts, VASS remains a challenging clinical problem to overcome in the hemodialysis population because its occurrence cannot be uniformly predicted either before or at the time of fistula or graft construction. ■

Scott S. Berman, MD, FACS, is from the Southern Arizona Vascular Institute, in Tucson, Arizona. Dr. Berman may be reached at (520) 628-1400; sberman@azvasc.com.

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