Treating CVD in the Dialysis Patient

Central venous stenosis or obstruction is a common yet difficult problem to manage in hemodialysis patients and can lead to disabling symptoms and potential access dysfunction.

BY SANJOY KUNDU, MD, FRCPC, FCIRSE, FSIR, RPVI

Central venous occlusive disease (CVD), composed of venous stenosis and obstruction, is a prevalent and clinically significant problem in managing access for hemodialysis patients. CVD affects the hemodialysis access circuit by causing symptomatic venous hypertension and access flow dysfunction. The incidence of CVD has been reported in the literature to be in the range of 25% to 40%.[1,2]

PATIENT CONSIDERATIONS

Possible Causes of CVD

There has been a strong association of CVD with the previous placement of central venous catheters and pacemaker wires. One study demonstrated that 27% of patients with CVD had a history of previous central venous catheter placement.[3] In addition, there is a very high incidence of CVD (42%–50%) in patients with a history of subclavian catheters compared with central venous catheters placed via the internal jugular vein route.[4-7] One suggested mechanism for the development of CVD includes central venous catheter–induced trauma to the venous endothelium and secondary inflammatory damage within the vessel wall at the time of insertion. Other proposed mechanisms include the presence of a foreign body in the vein along with increased flow and turbulence from the creation of an arteriovenous (AV) access.[7-11]

Potential Risk Factors

A history of venous access placement or central vein procedures in hemodialysis patients is the most common risk factor for developing CVD.[3,7,12] The access site location for central venous catheter placement is also an important risk factor for CVD; catheters placed by a subclavian access have a particularly high risk, with a 42% incidence of CVD compared to a 10% rate via an internal jugular vein access.[4-7] There is also an increased predilection for CVD to occur with left-sided venous access catheter placement, which may be related to the more tortuous course that catheters traverse from a left-sided access.[6,13-15] The high incidence of CVD with hemodialysis catheters may be related to the large caliber of these catheters or the high flow rates required for hemodialysis.

> "Endovascular intervention is the first line of treatment in hemodialysis patients with CVD."

Peripherally inserted central catheters and central venous port catheters are also becoming an increasingly prevalent risk factor for CVD. Most patients with CVD secondary to peripherally inserted catheters and central venous port catheters are most commonly asymptomatic, and the disease clinically declares itself after a hemodynamic challenge such as the placement of an

ipsilateral AV access for hemodialysis.[16,17] Pacemaker and defibrillator wires can also potentially lead to CVD.[18-21]

Clinical Presentations

CVD may be asymptomatic but can be detected on a preaccess assessment mapping venogram or diagnostic fistulagram for an immature fistula, which is delayed in maturation beyond 90 days.[22,23] Most occult CVD becomes clinically apparent after the development of a functioning AV access. Symptom development secondary to CVD depends on the anatomical site of the stenosis or occlusion. A narrowing or occlusion of the subclavian vein most commonly presents with AV access dysfunction or ipsilateral edema of the extremity and breast. Brachiocephalic vein stenosis or occlusion affects blood flow from the same side of the face as well as the upper extremity and breast, leading to ipsilateral extremity and possible facial and neck edema.

Approximately 50% of patients with significant CVD will develop upper extremity edema.[22] Significant edema is much more common once a functional ipsilateral upper extremity AV access has been created and has matured.[24] Ongoing cannulation of a matured AV access for hemodialysis can lead to further exacerbation of the edema, with acute swelling, tenderness, pain, and associated erythema after hemodialysis. Associated edema of the breast on the same side and pleural effusions may develop.[25,26]

CVD may lead to aneurysmal dilation and tortuosity of an AV access. Progression may be prevented with prompt treatment of the causative central venous occlusive lesion. Marked aneurysmal dilatation may have to be treated with surgical revision, ligation, or endovascular management of the AV access. CVD leads to the development of collaterals, which divert blood centrally via enlarged collateral veins. The collateral veins are often evident on physical examination on the neck, chest, and ipsilateral extremity.

Superior vena cava syndrome is an uncommon but potentially catastrophic complication of superior vena cava stenosis or obstruction or bilateral brachiocephalic vein narrowing or occlusion.[27,28] This clinical entity is composed of edema of both upper extremities, face, and neck, along with multiple dilated collateral veins over the chest and neck. Acute emergent treatment of superior vena cava syndrome is required, most commonly using an endovascular approach.

CVD may also decrease access blood flow, leading to access recirculation and inadequate or prolonged hemodialysis sessions. AV access dysfunction may also present as elevated venous pressure during hemodialysis and prolonged bleeding from needle sites after dialysis. If there is a hemodynamically significant decline in access blood flow, the AV access may become thrombosed. Thrombolysis techniques will be temporarily effective or lead to recurrent thrombosis unless the CVD is also properly managed.

Diagnosing CVD

The diagnosis of CVD is made based on a combination of clinical and imaging findings. A significant subgroup of patients will have a history of previous central venous catheter placement or central venous procedures and will present with ipsilateral arm, breast, face, or neck swelling. Depending on the location of the access, a proportion of patients will have evidence of AV access dysfunction with decreased access flows or aneurysmal dilation of the fistula. On physical examination, there may be numerous dilated collateral veins in the neck or chest and arm edema on the side of the CVD. In the cases of bilateral brachiocephalic vein or superior vena cava stenosis or occlusion, patients may present with a combination of findings suggestive of superior vena cava syndrome. CVD can sometimes be diagnosed by duplex ultrasound with an absence of normal respiratory variation in the diameter of central veins and polyphasic atrial waves.[29] It is difficult to visualize the central veins with duplex ultrasound in patients with an elevated body mass index or significant chest musculature.

Digital subtraction venography is the current standard for the diagnosis of CVD due to its increased sensitivity and specificity compared with duplex ultrasound.[1] All patients undergoing diagnostic fistulagrams for AV access dysfunction or fistulas delayed in maturation should undergo a complete assessment of the complete access circuit from anastomosis to the right atrium with contrast venography to rule out CVD. Magnetic resonance venography or CO_2 venography are infrequently used as alternatives to conventional digital subtraction venography, but there is no significant literature to date on its usefulness in diagnosing CVD. However, it should be noted that patients with decreased glomerular filtration rate are at risk of developing nephrogenic systemic fibrosis with the intravenous administration of gadolinium for magnetic resonance imaging.[30]

TREATMENT OPTIONS FOR SYMPTOMATIC CVD PATIENTS

Endovascular intervention is the first line of treatment in hemodialysis patients with CVD. The treatment options include percutaneous transluminal angioplasty (PTA), placement of bare-metal stents (BMS), and

recently, covered stents. The Kidney Disease Outcomes Quality Initiative guidelines recommend endovascular treatment with PTA with second-line stent placement as the preferred treatment approach to CVD.[31]

PTA

PTA for CVD was first reported in the literature by Glanz et al in 1984, with a 100% technical success rate and no reported patency rates.[32] A subsequent study by Trerotola et al in 1986 demonstrated similar technical and clinical success rates.[33] Conventional plain balloon angioplasty is the first-generation endovascular technology and the first line of treatment for CVD. Unfortunately, at the time of the preliminary PTA studies, there were no clearly defined reporting standards in place, leading to variable study methodologies, endpoints, and results of treatment. There are no large randomized control or cohort studies to assess PTA for CVD, making it difficult to draw conclusions on the outcomes of PTA and make comparisons to newer alternative technologies.

> "Stent structure and composition may be a factor in the initial technical success rate and long-term patency . . . "

PTA has demonstrated a variable technical success rate that ranges from 70% to 90%.[2,18,34-38] A study by Kovalik et al in 1994 made some interesting observations, including a technical failure rate of 7%, with > 50% improvement in 70% of CVD patients with nonelastic lesions and < 50% improvement in 23% of CVD patients with elastic lesions. The study concluded that there were two types of central venous lesions: nonelastic lesions that responded well to PTA and elastic lesions that were unresponsive or poorly responsive to PTA. It was believed that the histology of the two types of lesions were different based on observations on intravascular ultrasound, with the nonelastic lesions predominantly composed of fibrosis.[34]

Overall, the PTA patency results for CVD demonstrate a wide range of variability. There is a 6-month primary patency range of 23% to 63% and a cumulative patency range of 29% to 100%. There is a 12-month primary patency range of 12% to 50% and a cumulative patency range of 13% to 100%.[2,18,34-38] One of the largest studies to date (composed of 47 patients) on PTA for CVD was done by Bakken et al in 2007 and demonstrated a technical success rate of 77%. Primary patency rates at 3, 6, and 12 months were 58%, 45%, and 29%, respectively; cumulative patency rates at 3, 6, and 12 months were 76%, 62%, and 53%, respectively.[38] In summary, technical failures will occur in a minority of patients when treating CVD with PTA in the range of 10% to 30%. There is clearly a subgroup of CVD patients with lesions unresponsive to PTA. It is also apparent that multiple repeated interventions with close surveillance are required to maintain patency and prevent occlusion over the long-term.

BMS

BMS were first placed in the dialysis access circuit for refractory stenoses unresponsive to PTA by Gunther et al in 1989.[39] BMS are second-generation endovascular technology and the second line of treatment for CVD. BMS provide fixed mechanical support to a site of stenosis that is resistant or unresponsive to PTA, secondary to elastic recoil or neointimal hyperplasia. BMS are potentially useful in CVD in the setting of kinked stenoses, collapsing or elastic stenoses post-PTA, sealing dissections or circumscribed perforations post-PTA, establishing and maintaining patency of chronic central vein occlusions, early recurrent stenoses after PTA, and after PTA of highly resistant unresponsive stenoses.

However, there are significant limitations to BMS. During deployment, BMS may migrate or shorten and fracture in the short- or long-term.[40-43] BMS placement may preclude future endovascular procedures, central venous line placement, surgical treatment options, or future AV access creation. It is also clearly evident that BMS incite intimal hyperplasia, leading to recurrent stenoses and/or occlusions requiring multiple repeat interventions to maintain patency.[42]

According to the United States Renal Data System, the use of BMS in hemodialysis access interventions has significantly increased from 0% in 1991 to over 12% in 2008.[44] The exponential increase in BMS usage in hemodialysis access procedures has led to the development of guidelines for its use. The Society of Interventional Radiology Quality Improvement guidelines recommend that BMS be reserved for central vein lesions in which PTA has failed or that recur within 3 months after an initially successful PTA or rupture after PTA.[45] Similarly, the consensus guidelines of the National Kidney Foundation Dialysis Outcomes Quality Initiative recommend that the use of stents be reserved for surgically inaccessible stenoses in which PTA fails.[31,46,47]

The results for BMS demonstrate a wide range of variability. The majority of the literature shows a very high technical success rate (approximately 100%).[34-38]

Stent structure and composition may be a factor in the initial technical success rate and long-term patency, although this has not been demonstrated in the litera-

ture to date. As a general rule, self-expanding stents have been used for CVD. The first-generation self-expanding stent used for endovascular intervention was the Wallstent (Boston Scientific Corporation, Natick, MA). The Wallstent is constructed of 18 filaments of Elgiloy woven into a mesh. The advantages of this stent include radiopacity, the ability to reconstrain the stent when partially deployed, and wide availability. The disadvantages of this stent include foreshortening at the time of placement, eccentric loading (stenosis) that can lead to concentric narrowing and decreased radial strength, rigidity or lack of flexibility, and rarely, delayed shortening and migration.[40,41,48-50]

Nitinol stents are the second-generation self-expanding stents. Nitinol is an alloy of nickel and titanium and has a crystalline structure that exists in two types of temperature-dependent forms. Nitinol undergoes a reversible shape transformation (martensitic transformation) that is preset by the ratio of nickel and titanium and high-temperature heating. When nitinol transforms to its higher-temperature crystalline form (28º–33º C), it will expand to its preset size and become relatively more rigid. Nitinol also has the characteristic of superelasticity, which will cause an applied external force to deform it, but it will attempt return to its original shape over time or if the external force is removed.[41,50-52]

The advantages of nitinol stents include flexibility, ease of deployment, a wide range of diameters and lengths, a lack of foreshortening, and low-profile delivery systems. The disadvantages of nitinol stents include the relative lack of radiopacity (unless there are radiopaque markers at the ends of the stents), sharp edges to the ends of the stents that can damage the recipient vein, and a lack of ability to reconstrain the stent once it is partially deployed. Nitinol stents are presently manufactured by numerous proprietors.

The results for BMS in the setting of CVD have been quite variable. There is a 3-month primary patency range of 63% to 100% and a cumulative patency range of 72% to 100%, a 6-month primary patency range of 42% to 89% and a cumulative patency range of 55% and 100%, and a 12-month primary patency range of 14% to 73% and a cumulative patency range of 31% to 91%.[34-38,42,43,53-58] One of the largest retrospective studies to date on BMS with the Wallstent for CVD was performed by Haage et al in 50 patients and was published in 1999.[55] This study demonstrated 3-, 6-, and 12-month primary patency rates of 92%, 84%, and 56%, respectively. There was a cumulative patency rate at 6 and 12 months of 97%. Unfortunately, these results have not been replicated elsewhere in the literature to date.

A more recent retrospective study on nitinol BMS for CVD was by Vogel et al in 2004 with 16 patients and demonstrated 3-, 6-, and 12-month primary patency rates of 81%, 74%, and 67%, respectively. Cumulative patencies were not reported in this study.[53] There are no randomized controlled trials to date comparing PTA and BMS in the setting of CVD. A recent retrospective study by Bakken et al published in 2007 comparing PTA and BMS for CVD demonstrated 3-, 6-, and 12-month primary patencies with PTA of 58%, 25%, and 29%, respectively, in comparison with 3-, 6-, and 12-month primary patencies with BMS of 65%, 54%, and 45%, respectively. There were 3-, 6-, and 12-month cumulative patency rates with PTA of 76%, 62%, and 53%, respectively, in comparison with 3-, 6-, and 12-month cumulative patencies with BMS of 72%, 55%, and 46%, respectively. There was no significant difference in patency results between the PTA or BMS groups.

In summary, it appears that BMS for CVD show a high initial technical and clinical success rate. There is clearly a subgroup of CVD patients who are unresponsive to PTA immediately or on a short-term basis and will require BMS to achieve technical and clinical success. However, there is no literature to date demonstrating the superiority of BMS over PTA in the setting of CVD. Future large, prospective, randomized controlled trials will be required to determine the appropriate role of BMS for CVD.

Covered Stents

Covered stents, also known as endografts, have been proposed as a new treatment option for CVD. A potential benefit of a covered stent is that it may provide a relatively inert and stable intravascular matrix for endothelialization while providing the mechanical advantages of a BMS. This could potentially reduce neointimal hyperplasia, which causes restenosis after PTA or BMS placement. A disadvantage of covered stents may be the cost because covered stents are typically three times the cost of a BMS. Other disadvantages include the large profile of the delivery systems (7–10 F) and the limited availability of large-diameter covered stents. A major disadvantage is that there is nothing published in the literature to support using them in the central veins.

Covered stents are available in balloon-expandable or self-expanding platforms. In practical terms, a self-expanding platform would be preferred, given the rigidity of the balloon-expandable platforms, angulations in the central venous system, and long length of lesion coverage sometimes required in CVD. There is a small amount of literature on covered stent usage in the hemodialysis access circuit. Most of the literature to date has been on the treatment of graft or outflow vein aneurysms and refractory venous outflow stenoses.[59-65]

Covered stents for CVD have only been mentioned in two publications to date. In 1996, Sapoval et al mentioned the use of a nitinol plus Dacron covered stent (Cragg Endopro, Mintec, La Ciotat, France) for an instent restenosis of a Wallstent, with asymptomatic recurrent restenosis after 6 months.[63] In a study by Quinn et al in 2003, six covered stents were placed for CVD and 11 covered stents for venous outflow stenoses.[66] There were combined primary patency rates at 2, 6, and 12 months of 40%, 32%, and 32%, respectively, and secondary patency rates at 2, 6, and 12 months of 70%, 55%, and 39%, respectively. They used a Palmaz stent (Cordis Corporation, Bridgewater, NJ) with an expanded polytetrafluoroethylene graft material manually sewn on.

Further randomized controlled trials comparing PTA, BMS, and covered stents with long-term follow-up will be required to determine the potential role of covered stents in the treatment algorithm for CVD.

Surgical Options for CVD

Percutaneous endovascular intervention is the accepted first line of treatment for CVD. However, in patients refractory to endovascular options, surgical options must be evaluated. If there is a functioning hemodialysis access in the ipsilateral extremity to the site of CVD, a simple reduction procedure may bring the volume down to something that can be accommodated by collateral circulation and continue to provide adequate flow for dialysis with resolution of symptoms. If not, then the CVD can be addressed by extra-anatomic bypass, including jugular vein turn-down procedures, subclavian vein to external or internal jugular vein bypass, or axillary to femoral vein bypass.[67-69]

Surgical options for CVD are associated with significant morbidity in patients with CVD due to poor short- and long-term patency and are a last-resort treatment alternative in patients refractory to percutaneous endovascular treatment options.

FUTURE DIRECTIONS FOR MANAGING CVD

Future treatments may include coated drug-eluting stents with bioactive coating to improve endothelial healing inside the stent and limit neointimal hyperplasia. Larger-diameter Cutting balloons (Boston Scientific Corporation) or cryoplasty are other potential PTA advancements. Another alternative may be brachytherapy with beta radiation, which has shown some benefit in coronary intervention.

Further characterization of biologic, molecular, histologic, and pathologic mechanisms of CVD, as well as development of preventive strategies, are the keys to improving long-term patency of the hemodialysis access circuit.

CONCLUSION

Prevention of CVD in hemodialysis access patients is of the utmost importance. Central venous catheter placement or central venous procedures are the most important risk factors for CVD. In patients with renal dysfunction, central and peripheral venous access placement should be avoided if at all possible. The use of peripheral venous access lines should be minimized to preserve future peripheral and central venous capital as potential access sites.

All of the current treatment options for CVD will lead to recurrent stenosis or occlusion, requiring close surveillance with multiple repeat interventions to maintain patency. Further prospective, randomized controlled trials with long-term follow-up for all of the currently available treatment options will be necessary to develop appropriate treatment algorithms. Further advancements in endovascular technique and technology, with rigorous scientific evaluation, will be required to continue to improve the long-term results for this difficult patient management problem. ■

Sanjoy Kundu, MD, FRCPC, FCIRSE, FSIR, RPVI, is Medical Director at The Vein Institute of Toronto & Scarborough Vascular Ultrasound in Toronto, Ontario, Canada. Dr. Kundu has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Kundu may be reached at (416) 929-0834; sanjoy_kundu40@hotmail.com.

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