Review Article

Review of Central Venous Disease in Hemodialysis Patients

Sanjoy Kundu, MD, FRCPC, FSIR

A common problem in the management of patients who are undergoing hemodialysis is central venous occlusive disease. There has been extensive literature on the treatment of this important and prevalent problem. Treatment options to date include percutaneous balloon angioplasty, bare metal stents, and surgical bypass. Unfortunately, all the available treatment options have poor long-term patency, requiring repeated interventions. More recently, covered stents have been mentioned in the literature for the treatment of central venous stenosis and obstruction. There are very few data to date on this technology, and further randomized controlled trials will be needed to compare the efficacy of percutaneous balloon angioplasty, bare metal stents, and covered stents. It appears that it is of paramount importance to prevent this difficult problem by limiting access to, or intervention in, the central venous system.

BACKGROUND

CENTRAL venous stenosis and obstruction, or central venous disease (CVD), is a common and significant problem in the long-term management of the access circuit for patients undergoing hemodialysis. CVD disrupts the hemodialysis access circuit by causing venous hypertension and access flow dysfunction with or without debilitating symptoms. This can result in loss of the access site as a result of access malfunction or surgical ligation for symptom relief. The incidence of CVD has been reported in the literature to be in the range of 25%–40% (1,2).

ETIOLOGY

There has been a strong association of CVD with previous placement of

From the Department of Medical Imaging, Scarborough Hospital, 217 Davenport Road, Toronto, ON, Canada M5R 1J3. Received January 23, 2010; final revision received and accepted January 28, 2010. **Address correspondence to** S.K.; E-mail: sanjoy_kundu40@hotmail.com

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central venous catheters and pacemaker wires. One study (3) demonstrated that 27% of patients with CVD had a history of previous central venous catheter placement. In addition, there is a very high incidence of CVD in patients with a history of subclavian catheters (42%–50%) compared with central venous catheters placed via an 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).

Risk Factors

A history of central venous access placement or central vein intervention in patients undergoing hemodialysis is the most common risk factor for CVD. Placement of multiple central venous catheters, with increased duration of catheter dwell times, have been associated with an increased risk of

CVD (3,7,12). The access site for central venous catheter placement is also an important risk factor for CVD. Central venous catheters placed by a subclavian access are associated with a particularly high risk, with a 42% incidence of CVD compared with a 10% rate with catheters placed via an internal jugular vein access (4–7). CVD also tends to frequently occur with left-sided access for catheter placement, which may be related to the more tortuous course catheters have to 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 possibly to the high flow rates required for hemodialysis. To the author's knowledge there is no literature available to date to assess the impact of catheter caliber on CVD.

Peripherally inserted central catheters and central venous port catheters are also becoming increasingly important risk factors for CVD. Most patients with CVD secondary to peripherally inserted catheters and central venous port catheters are usually asymptomatic and present clinically after a hemodynamic challenge such as placement of an ipsilateral AV ac-

cess (16,17). Pacemaker and defibrillator wires can also lead to CVD, with development of clinical symptoms after the placement of an AV access in the ipsilateral extremity (18–21).

CLINICAL FEATURES

CVD can be asymptomatic and detected on a diagnostic venogram or fistulogram before access placement for an immature fistula (22,23). Most occult CVD cases become clinically apparent after development of a functioning AV access in the ipsilateral extremity. Symptomatology secondary to CVD depends on the anatomic location of the stenosis or obstruction. Narrowing or occlusion of the subclavian vein most commonly presents with edema and/or venous hypertension of the corresponding extremity and/or breast. Brachiocephalic vein stenosis or occlusion affects blood flow from the same side of the face and the upper extremity and breast, leading to edema of the ipsilateral extremity and possible facial edema.

Approximately only 50% of patients with significant CVD will develop ipsilateral upper-extremity edema (22). Edema is much more common when a functional ipsilateral upper extremity AV access has been created (24). Use of this access for hemodialysis, can lead to further exacerbation of the edema, with acute swelling, tenderness, pain, and associated erythema, which can mimic cellulitis. Associated edema of the breast on the ipsilateral side along with 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 inciting central lesion. Marked aneurysmal dilation may have to be treated with surgical revision or ligation of the AV access. CVD leads to the development of collateral vessels, 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 (SVC) syndrome is a very uncommon but feared complication of SVC stenosis or obstruction or bilateral brachiocephalic vein narrowing or occlusion (27,28). This clinical syndrome comprises edema of both upper extremities, face, and neck, along with multiple dilated collateral veins over the chest and neck. Acute emergent treatment of SVC syndrome is required.

CVD may also decrease access blood flow, leading to access recirculation and inadequate dialysis. This may also present as elevated venous pressure during hemodialysis and prolonged bleeding from needle sites after dialysis. If there is a significant decline in access blood flow, the AV access may become occluded secondary to thrombosis. Thrombolysis techniques will be ineffective or lead to recurrent thrombosis unless the CVD is also treated.

DIAGNOSIS

The diagnosis of CVD is based on clinical and imaging findings. A subgroup of patients will have a history of central venous catheter placement or intervention 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 flow rates. On physical examination, there may be numerous dilated collaterals vessels in the neck or chest and arm edema on the side of the CVD. In cases of bilateral brachiocephalic vein or SVC stenosis or occlusion, patients may present with a constellation of findings suggestive of SVC syndrome. CVD can at times be diagnosed by duplex ultrasound (US), 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 US in patients with a high body mass index or significant chest musculature.

Digital subtraction central contrast venography is the current gold standard for the diagnosis of CVD, due to its increased sensitivity, compared with duplex US (1). All patients undergoing diagnostic fistulography for AV access dysfunction should undergo a complete assessment of the entire access circuit with contrast venography to rule out CVD. Magnetic resonance venography or CO_2 venography are alternatives to conventional venography, but there is no significant literature to date on their use in the assessment of CVD. However, it should be noted that patients with decreased glomerular filtration rate are at risk of developing nephrogenic systemic fibrosis after the administration of intravenous gadolinium (30).

TREATMENT OPTIONS

Endovascular intervention is the present mainstay of treatment in the hemodialysis of patients with CVD. The treatment options include percutaneous transluminal angioplasty (PTA), placement of bare metal stents (BMSs), and recently, placement of covered stents. The National Kidney Foundation Disease Outcomes Quality Initiative guidelines (31) recommend PTA with or without stent placement as the preferred treatment approach to CVD.

PTA

PTA for CVD was first reported by Glanz et al (32) in 1984, with a 100% technical success rate. A subsequent study by Trerotola et al (33) in 1986 demonstrated similar technical and clinical success rates (33). PTA represents first-generation technology and is 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, a situation resulting in variable study methodology and endpoints. There are no large randomized controlled studies that have provided level 1 evidence in the assessment of PTA for CVD, making it difficult to draw conclusions on the outcomes of PTA and draw comparisons versus alternative technologies.

PTA has demonstrated a variable technical success rate ranging from 70% to 90% (2,18,34–38). A PTA study by Kovalik et al (34) in 1994 made some interesting observations, including a technical failure rate of 7%, with greater than 50% improvement (ie, nonelastic lesions) in 70% of the patients with CVD and less than 50% improvement (ie, elastic lesions) in 23% of the patients with CVD. The study concluded that there were two types of central venous lesion: nonelastic lesions, which responded well to PTA, and elastic lesions, which were unresponsive or poorly responsive to PTA. It was believed that the histologies of the two types of lesions

were different based on observations on intravascular US (34).

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

Bare Metal Stents

BMSs were first placed in the dialysis access circuit, for refractory stenoses, by Günther et al in 1989 (39). BMSs are the second-generation technology and second line of treatment for CVD; they provide mechanical support to a site of stenosis that is resistant or unresponsive to PTA. BMSs are potentially useful in CVD in the setting of kinked stenoses or collapsing or elastic stenoses after PTA; and for sealing dissections or circumscribed perforations after PTA, for establishing and maintaining patency of chronic central vein occlusions, and after PTA of highly resistant stenoses.

However, there are significant limitations to BMSs. After deployment, BMSs may migrate, shorten, or fracture on a subacute or delayed basis (40–43). BMS placement may preclude future endovascular procedures or surgical revision. It is also clearly evident that BMSs can incite intimal hyperplasia, leading to recurrent stenoses and multiple repeat interventions to maintain patency (42). The use of BMSs in hemodialysis access PTA interventions has significantly increased

from 0% in 1991 to more than 9% in 2001 according to the United States Renal Data System (44). The exponential increase in BMS use in hemodialysis access procedures has led to the development of guidelines for their applications. The Society of Interventional Radiology Quality Improvement Guidelines (45) recommend BMSs be reserved for central vein lesions in which PTA has failed or that recur within 3 months after initially successful PTA or in cases of rupture after PTA. Similarly, the consensus guidelines of the National Kidney Foundation Dialysis Outcomes Quality Initiative (31,46,47) recommend that the use of stents be reserved for surgically inaccessible stenoses in which PTA fails.

The results for BMS use demonstrate a wide range of variability. The vast majority of the literature demonstrates a very high technical success rate, as high as 100%.

Stent structure and composition may be a factor in the initial technical success rate and long-term patency, although this has not been clearly demonstrated in the literature to date. As a general rule, self-expanding stents have been used for CVD. The first generation self-expanding stent is the Wallstent (Boston Scientific, Natick, Massachusetts). The Wallstent is constructed of 18 filaments of Elgiloy woven into a mesh. The advantages of this stent include its low profile, flexibility, and radiopacity. The disadvantages of this stent include foreshortening at the time of placement, the fact that eccentric loading (stenosis) can lead to concentric narrowing and decreased radial strength, and rare delayed shortening and migration (40,41,48–50). The second-generation self-expanding stents are made of nitinol, an alloy of nickel and titanium. It has a crystalline structure, which exists in two types of temperature-dependent forms. Nitinol undergoes a reversible shape transformation (ie, martensitic transformation), which is preset by the ratio of nickel and titanium and high temperature heating. When nitinol transforms to its higher temperature crystalline form (28°C–33°C), it will expand to its preset size and become relatively more rigid. Nitinol also has the characteristic of superelasticity, which will allow an applied external force to deform it but

attempt to return to its original shape over time or if the external force is removed (41,50–52).

The results of BMS use in the setting of CVD have been quite variable. At 3 months, primary patency rates are 63%–100% and cumulative patency rates are 72%–100%; the respective rates are 42%–89% and 55%–100% at 6 months and 14%–73% and 31%–91% at 12 months (34–38,42,43,53–58). One of the largest retrospective BMS studies to date, with Wallstent treatment of CVD, was that of Haage et al (55) in 1999. In 50 patients, primary patency rates were 92% at 3 months, 84% at 6 months, and 56% at 12 months. There was a cumulative patency rate of 97% at 6 and 12 months (55). Unfortunately, these results have not been replicated elsewhere in the literature. A more recent retrospective study on nitinol BMSs for CVD in 16 patients by Vogel et al (53) in 2004 demonstrated 3-, 6-, and 12-month primary patency rates of 81%, 74%, and 67%, respectively. Cumulative patency rates were not reported in this study (53). There are no randomized control trials to date comparing PTA and BMS use in the setting of CVD. A recent retrospective study by Bakken et al (38) comparing PTA versus BMS placement for CVD demonstrated 3-, 6-, and 12-month primary patency rates of 58%, 25%, and 29%, respectively, with PTA, compared with 65%, 54%, and 45%, respectively, with BMSs. Cumulative patency rates at 3, 6, and 12 months were 76%, 62%, and 53%, respectively, with PTA, compared with 72%, 55%, and 46%, respectively, with BMSs. There was no significant difference in patency results between the PTA and BMS groups.

In summary, it appears BMS placement has a high technical success rate in CVD. There is clearly a group of CVD cases that are unresponsive to PTA and will require BMSs to achieve technical success. However, there is no literature to date demonstrating the superiority of BMSs versus PTA in the setting of CVD. Future randomized control trials will be needed to determine the appropriate role of BMSs for CVD.

Covered Stents

Covered stents, also known as peripheral endografts, have been pro-

posed as a new treatment option for CVD. The potential advantages of covered stents would include providing a relatively inert and stable intravascular matrix for endothelialization while providing the mechanical advantages of a BMS. This could potentially reduce the intimal hyperplastic response that causes restenosis after PTA or BMS placement. 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. There is minimal literature on covered stent use 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). To the author's knowledge, covered stents for CVD have been mentioned in only two publications to date. Sapoval et al (63), in 1996, mentioned the use of a nitinol plus Dacron covered stent (Craig Endopro; Mintec, La Ciotat, France) for in-stent restenosis of a Wallstent, with asymptomatic recurrent restenosis after 6 months. In a 2003 study, Quinn et al (66) placed six covered stents for CVD and 11 covered stents for venous outflow stenoses. Combined primary patency rates were 40%, 32%, and 32%, respectively, at 2, 6, and 12 months; the respective secondary patency rates were 70%, 55%, and 39%. The investigators used Palmaz stents (P308; Johnson & Johnson, Warren, New Jersey) with expanded polytetrafluoroethylene graft material manually sewn on (66). Further randomized controlled trials with long-term follow-up will be necessary to determine the role of covered stents in CVD.

Surgical Options for Central Venous Disease

Percutaneous endovascular therapy is the first line of treatment for CVD. However, in patients whose disease is refractory to endovascular options, surgical possibilities 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 sufficiently to be accommodated by collateral circulation and continue to provide adequate flow for

dialysis with resolution of symptoms. If not, the CVD can be addressed by extraanatomic 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 and are a last-resort treatment alternative in patients whose disease is refractory to percutaneous endovascular treatment options.

FUTURE DIRECTIONS

Future treatments may include coated drug-eluting stents with a rapamycin or paclitaxel coating to improve endothelial healing inside the stent, larger-diameter cutting balloons, or cryoplasty. Other alternatives may include brachytherapy with β-radiation.

Further characterization of hemodynamic, molecular, and pathologic mechanisms of CVD, and development of treatments and preventative strategies, are critical to improve long-term patency of the hemodialysis access circuit.

SUMMARY

Prevention of CVD in patients undergoing hemodialysis is critical. Central venous catheter placement or intervention is the most important risk factor for CVD. In patients with renal dysfunction, central venous catheter placement should be avoided if at all possible, particularly in the subclavian vein. The use of other peripheral catheters should be minimized to preserve future peripheral and central venous capital as potential access sites.

All the current treatment options for CVD will lead to recurrent stenosis or occlusion requiring multiple repeat interventions to maintain patency. Further randomized controlled trials of currently available treatment options with long-term follow-up are essential in the future to develop appropriate treatment algorithms. Further advancements in treatment technique, technology, and mechanisms of CVD with proper scientific evaluation will be required to continue to improve the long-term results in this difficult problem.

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