Central Vein Stenosis

18

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18.1 Epidemiology

True incidence and prevalence of central vein stenosis are not known as most of CVS is asymptomatic and therefore fails detection. A study done by Schwab et al. estimates prevalence of subclavian stenosis at 25 % [1]. Similarly, another study from the 1980s reported venographic evidence of subclavian stenosis in 18 of 36 (50 %) patients [2, 3]. In a retrospective investigation of symptomatic HD patients undergoing angiography, 19 % of all patients and 27 % of those with a previous history of CVC placement were found to have CVS, similar to the finding of 16 % in a duplex and angiographic study [4, 5].

18.2 Risk Factors Associated with CVS

18.2.1 Prior History of CVC

It is uncommon for CVS to occur in HD patients, without prior history of central venous access placement or intervention. Placement of multiple central venous catheters, with increased duration of catheter dwell times, has been associated with a higher risk of CVS [4, 6, 7].

Study by Hernandez et al. examined 42 consecutive chronic renal failure patients in whom subclavian catheters had been placed as the initial vascular access for hemodialysis. All patients underwent sequential venography studies: at baseline (24–48 h after removal of the catheter) and 1, 3, and 6 months thereafter. Venograms were considered abnormal when there was evidence of unequivocal strictures (more than 30 % narrowing), with or without collateral circulation. At baseline, 52.4 % ($n=22$) of patients showed stenotic vein lesions ($n=19$) or total thrombosis ($n=3$), and identical lesions were also observed after 1 month. Surprisingly, 10 of 22 patients with initial CVS (45.4 %) showed spontaneous recanalization of venous lesions in the venographies performed 3 months after removal. Patients with definitive stenosis at 6 months had a higher number of inserted catheters (1.58 versus 1.2; $p<0.05$), longer time in place (49 versus 29 days; $p<0.05$) than those without CVS or with spontaneous recanalization of venous lesions during follow-up. Furthermore, a higher number of catheter-related infections were observed in patients with definitive CVS (66.6 % versus 33.3 %; $p<0.05$) [8]. Similarly, a study by Macrae et al. found that 55 out of 133 patients (41 %) had evidence of significant CVS on venogram. Patients with CVS had a longer duration on HD and a history of a previous HD catheter insertion [7].

In a prospective study by Oguzkurt et al., 57 patients with temporary dialysis catheters had catheter venography by pulling back the catheter just before removal. This study showed that even short-term catheters result in significantly high rates of pericatheter sleeve and thrombus formation, which are two of the important causes of catheter malfunction. These findings remind us again that we should avoid unnecessary catheter insertion even for short term in these chronically ill patients [9].

18.2.2 Site of CVC Placement

Central venous catheters placed by a subclavian access have a particularly high risk, with a 42 % incidence of CVS compared to a 10 % rate with catheters placed via an internal jugular vein access [2, 6, 10]. This has been reported by Schillinger et al. angiographically, when they compared the subclavian–brachiocephalic vein of 50 patients dialyzed by subclavian catheter to those of 50 patients dialyzed by internal jugular catheters [11].

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There is also an increased predilection for CVS to occur with left-sided access for catheter placement, which may be related to the more tortuous course catheters that have to traverse from a left-sided access [11–14].

18.2.3 PICC and Ports

Study by Grove et al. showed that the overall thrombosis rate was 3.9 % after PICC placement. Multivariate analysis of the results indicated that only catheter diameter remained significant. There was no thrombosis in catheters 3 F or smaller. The thrombosis rate was 1 % for 4-F catheters, 6.6 % for 5-F catheters, and 9.8 % for 6-F catheters [15]. The smallest acceptable catheter diameter should be used to decrease the incidence of venous thrombosis. New central vein stenosis or occlusion occurred in 7 % of patients following upper arm placement of venous access devices. Patients with longer catheter dwell time were more likely to develop central vein abnormalities. With an increasingly prevalent use of PICC lines, the complication of CVS is likely to become more prevalent. Not only the awareness of this complication needs to be improved, the use of alternative means of intravenous access, such as single-lumen central venous infusion catheters, should be seriously considered to avoid the loss of an arm vein for the future creation of AVF [15–19].

18.2.4 Pacemaker/Defibrillator Wires

CKD/ESRD shares risk factors with cardiovascular disease, and it is not uncommon for these patients to undergo implantation of pacemakers or defibrillators. Thirty consecutive patients with a transvenous defibrillator lead underwent bilateral contrast venography of the cephalic, axillary, subclavian, and brachiocephalic veins as well as the superior vena cava before an elective defibrillator battery replacement. The mean time between transvenous defibrillator lead implantation and venography was 45 ± 21 months. Sixteen patients (>50 %) had more than one lead in the same subclavian vein. No patient had clinical signs of venous occlusion. Subclavian stenosis (defined as more than 50 % stenosis) was found in 50 % of patients, and 13 % of patients had more than 75 % stenosis [20–22]. Total or partial obstruction of the access veins occurs relatively frequently after pacemaker or ICD implantation. Multiple pacing or ICD leads are associated with an increased risk of venous obstruction, whereas antiplatelet/anticoagulant therapy appears to have a preventive effect on the development of access vein thrombosis [23]. If a device is to be placed in a dialysis patient, it should not be placed on the side of the AV access because of the high incidence of CVS. Another study by Bulur et al. studied 86 patients who had undergone biventricular device implantation. Subclavian vein stenosis was present in 39 % of all participants. Among the patients with subclavian obstruction ($n = 33$), 8 had mild obstruction, 15 had severe obstruction, and 10 had total occlusion [24].

18.2.5 Catheter Composition

A variety of plastic materials including polyvinyl chloride, polyethylene, polyurethane, and silicone has been used in the production of CVC for hemodialysis. In animal experiments, silicone was shown to be less thrombogenic than other materials [25–27]. Trials systematically comparing CVC made from different materials to assess their respective rates of infection or thrombosis in hemodialysis patients have not been published.

In a rabbit model, polyethylene and polytetrafluoroethylene catheters caused more inflammation than silicone and polyurethane. Obstructions of the venous lumen were significantly more frequent with the rigid catheters than with the soft catheters [26].

18.2.6 Idiopathic

A study by Oguzkurt et al. showed that 10 % of hemodialysis patients had stenosis of a central vein without a previous central catheter placement. Central venous stenosis in hemodialysis patients without a history of central venous catheterization tends to occur or be manifested in patients with a proximal permanent vascular access with high flow rates [28].

18.2.7 Catheter Infections

A study by Hernandez et al. retrospectively analyzed 80 catheterizations in a total of 54 chronic HD patients from a single center. Sixteen catheters had to be removed because of a well-documented catheter-related infection. For comparison they matched 14 concurrent catheters, which were electively removed without evidence of infection and with a negative culture of the catheter tip. A venogram of the ipsilateral arm was performed in all the cases after more than 6 months of catheter removal. CVS was three times more common among patients with previous catheter-related infection (75 % versus 28 %; $p < 0.01$) [8, 14, 29, 30].

A group of 479 jugular vein catheterizations, 403 RJVC, and 77 LJVC done in 294 prevalent hemodialysis patients were analyzed. Of the RJVC, 44 (10.9 %) of 403 were removed because of infection compared with 16 (20.8 %) of 77 LJVC ($p < 0.02$). The overall incidence of infections was 1.58 episodes of infection per 1,000 catheter days, 1.57 for RJVC, and 3.72 for LJVC, respectively. Catheter dwell times were not different in this study [14].

18.3 Pathogenesis

The precise mechanism of CVC-associated CVS remains largely undefined. Plausible mechanisms are linked to the CVC-induced trauma to the venous endothelium and the resultant inflammatory response within the vessel wall. Aside from the initial trauma at the time of CVC placement, many factors, including the presence of foreign body in the vein, sliding movement of the catheter with respiration, postural and head movements, and increased flow and turbulence from creation of AVF, alone or in combination, stimulate various processes within the vessel wall. The high blood flow associated with dialysis, turbulence, and vibrations has shown to cause platelet aggregation and deposition and endothelial hyperplasia [31–33].

Histologic examination of specimens from subclavian vein stenosis has corroborated this endothelial hyperplasia hypothesis by demonstrating the presence of fibrous tissue [34]. In addition, the uremic milieu hypothesis has been supported by recent findings of intimal changes in the cephalic vein of renal failure patients even prior to AV fistula construction [35]. Intravascular thrombosis can result from the release of profibrotic cytokines that are associated with platelet–platelet aggregates in this scenario [36]. Furthermore, direct physical damage from the movement of the catheter tip or body against a vessel wall can potentially result in thrombin generation, platelet activation, expression of P-selectin, and an inflammatory response [37, 38].

18.4 Clinical Features

CVS can be asymptomatic, detected on a pre-access placement diagnostic venogram or fistulogram for an immature fistula [1, 39]. One hundred ninety patients, 61 with acute renal failure and 129 with chronic renal failure, underwent hemodialysis using a total of 302 subclavian vein catheters. Local hematomas and sepsis (seven events) were the only acute complications. Subclavian vein stenosis and/or thrombosis had occurred and were shown in 5 of 44 patients who had AV access created distal to the venous outlet obstruction, resulting in the loss of 3 of 5 of these accesses.

In view of the fact that subclavian vein stenosis or occlusion is not associated with any clinical findings and we were unable to identify any predisposing factors associated with the use of the catheters, all patients who have had previous subclavian vein catheters probably should be evaluated to determine the patency of the subclavian vein before creation of a permanent access in that arm [39]. Most occult CVS becomes clinically apparent after development of a functioning AV access in the ipsilateral extremity.

Symptomatology secondary to CVS depends on the anatomical 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 breast. Innominate 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 edema. Approximately, only 50 % of patients with significant CVS will develop ipsilateral upper extremity edema. In study by Schwab et al., 47 patients underwent upper arm venography to evaluate fistula dysfunction. Subclavian vein stenosis was documented in 12. Eleven of twelve had elevated venous dialysis pressure (196 ± 8.9 mmHg), and six had arm edema [1].

Edema is much more common once a functional ipsilateral upper extremity AV access is created [40]. Use of this access for HD 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 [41, 42]. See Fig. 18.1 below.

CVS 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. CVS leads to the development of collaterals, which divert blood centrally via enlarged collateral veins. The collateral veins are often evident on physical examination of the neck, chest, and ipsilateral extremity. Rarely, the collaterals can bypass sufficient blood flow centrally, leading to improvement or stabilization of the CVS symptoms. See Fig. 18.2 below.

Superior vena cava syndrome is a very uncommon but feared complication of superior vena cava stenosis or

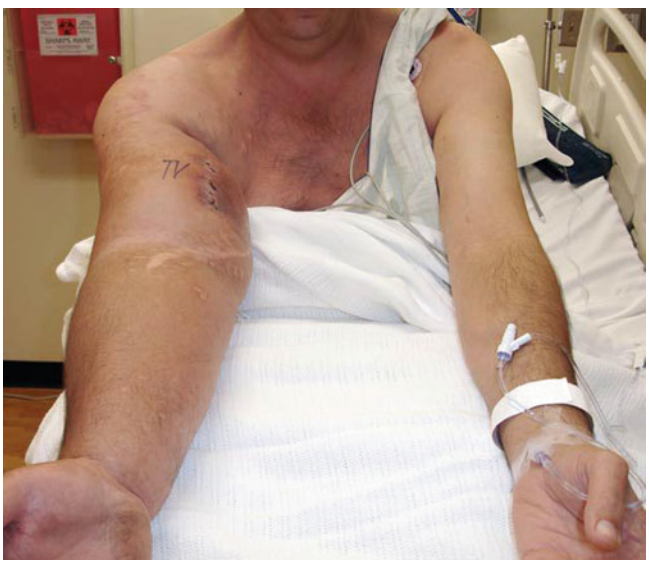

Fig. 18.1 R arm swelling due to CVS (Image courtesy of Dr. Vachhrajani)

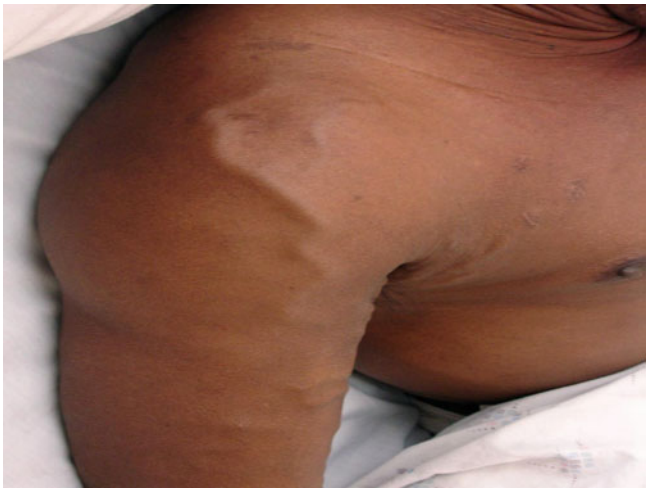

Fig. 18.2 Visible distended veins on arm and shoulder (Image courtesy of Dr. Vachhrajani)

obstruction or bilateral innominate vein narrowing or occlusion [43, 44]. This clinical syndrome is comprised of edema of the 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 sometimes required.

CVS may also decrease access blood flow, leading to access recirculation and inadequate dialysis. This may also present as elevated venous pressure during HD 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 CVS is also treated.

Positioning of the central venous catheter tip low down in the superior vena cava or in the right atrium has been advocated to improve dialysis adequacy and to reduce the incidence of catheter thrombosis. However, placement of the catheter tip within the right atrium may be associated with an increased risk of right atrial thrombus.

18.5 Diagnosis

An asymptomatic CVC is usually detected by angiography performed either in preparation of access placement or after the placement of AV access.

The diagnosis of the CVS can most often be made or suspected based upon a careful history and clinical examination. History of previous CVC placement, especially if multiple, should alert one to the possibility of CVS. Presence of pacemakers or automatic cardioverter defibrillators should prompt careful investigation to look for the presence of CVS and its resolution prior to placing a vascular access on the ipsilateral side. Extrinsic causes of CVS should also be considered and investigated.

Examination revealing numerous dilated collaterals in the neck or chest and arm edema on the ipsilateral side indicates obstruction to outflow. In case of bilateral CVS, a clinical picture of SVC syndrome can be seen, with facial edema. The direction of blood flow in collateral veins can be ascertained by careful examination. Central vein stenosis can often be confirmed by color-flow duplex venous ultrasound. A normal respiratory variation in the diameter of central veins and polyphasic atrial waves are present in most patients with patent central veins [45]. The presence of numerous collaterals in the neck is usually indicative of CVS. However, Doppler may mistake a dilated collateral vein as a patent central vein, unless attention is paid to the absence of respiratory variation [46]. It may be difficult to visualize central veins with ultrasound in those with significant muscle mass or obesity.

Central venography is the gold standard for the diagnosis of CVS. In a series of 141 patients, 54 stenoses were diagnosed in 41 patients by color-flow duplex and 64 stenoses were diagnosed by angiography [47]. There were 13 CVS (20.3 %), with 9 of the 13 CVS diagnosed by angiography only. Digital subtraction angiography is more sensitive than color duplex sonography in the evaluation of dialysis access. The DOQI guidelines recommend venography prior to placement of a permanent access in patients with previous subclavian catheterization [48]. See Figs. 18.3 and 18.4 below.

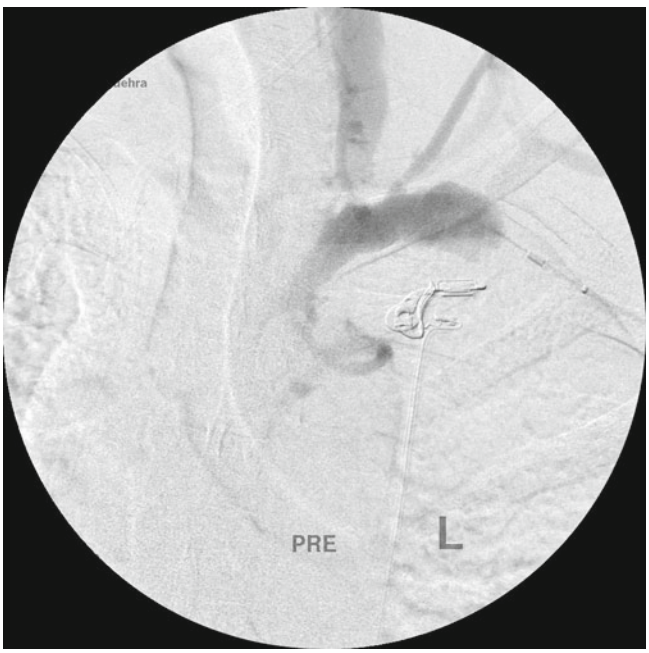

Fig. 18.3 L-sided central angiogram showing complete occlusion of L brachiocephalic vein with back flow into L IJV and presence of collaterals

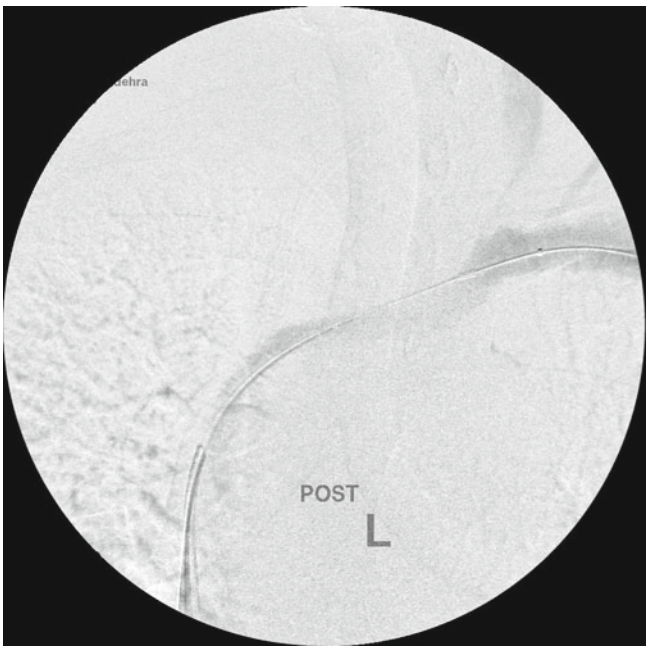

Fig. 18.4 Post-PTA disappearance of collaterals with forward flow

Magnetic resonance venogram permits avoidance of radiocontrast in a patient with advanced chronic kidney disease (CKD), where preservation of residual renal function is important [49]. This may also be useful in those with radiocontrast allergy. However, it should be noted that patients with decreased glomerular filtration rate (GFR) are at risk of developing nephrogenic systemic fibrosis [50].

18.6 Treatment

The treatment of CVS is indicated when symptoms are present. Some patients with asymptomatic CVS have adequate development of collaterals, which allow continuation of dialysis from the access without the development of symptoms or signs. These individuals need close monitoring and intervention if there is deterioration.

18.6.1 Conservative

The conservative treatment of CVS that is non-emergent involves elevation of the extremity and anticoagulation to prevent thrombosis associated with CVS. This strategy may be effective as a bridge to more definitive therapy and relies on the development of relatively adequate collaterals. In one study of high-grade (>50 %) CVS in 35 asymptomatic HD patients with 38 AVGs, 86 venograms were reviewed [51]. No intervention was done in 28 %, and none of these patients deteriorated or need further interventions. In contrast, 72 % of the patients who underwent percutaneous angioplasty (PTA) had escalation of CVS after PTA in 8 %, which required further interventions. PTA of asymptomatic CVS greater than 50 % in the setting of hemodialysis access maintenance procedures was associated with more rapid stenosis progression and escalation of lesions, compared with a conservative approach. These observations are consistent with the empiric observation that stenosis often accelerates after PTA [52].

The appropriate use of prophylactic PTA can reduce thrombosis rates and possibly prolong access life [53–55], but injudicious use of the same technique may accelerate stenosis formation and access failure. This is not surprising as the mechanism of venous PTA is endothelial disruption and intimal stretching. Damage to these sensitive vessel layers can trigger immune reactions, myointimal proliferation, and fibromuscular hyperplasia, processes that together may ultimately accelerate stenosis formation [56].

In their study, not only did CVS progress at a greater rate in treated individuals, but PTA also may have triggered adverse events such as new stenosis formation, stent requirement, and progression to symptomatic arm swelling. Along with being detrimental to the long-term patency of the central veins, the treatment of asymptomatic CVS with PTA had a low technical success rate in this cohort. A mean of 40 % residual stenosis was left after treatment with PTA despite aggressive use of large high-pressure balloons. While this degree of residual stenosis following PTA is higher than that in other studies in which PTA and/or stent placement for CVS has been described, restenosis is always the rule when this treatment modality is implemented.

On the other hand, it can be argued that the 40 % residual stenosis in the aforementioned study left the lesion essentially untreated, so there is no surprise that the outcomes in the intervention group were worse. This observation is especially meaningful in light of the Society of Interventional Radiology (SIR) guidelines' definition of a "refractory lesion" as postintervention stenosis of>30 % [52]. Some authors have even suggested that central venous lesions represent a primary indication for stent placement due to the poor outcome usually found with balloon dilation alone and the relatively small diameters that can be achieved with PTA [57, 58].

18.6.2 Percutaneous Transluminal Angioplasty

Glanz et al. first reported PTA for CVS in 1984, with 100 % technical success rate [57]. A subsequent study by Trerotola et al. in 1986 demonstrated similar technical and clinical success rates [60]. PTA is the first-generation technology and the first-line treatment for CVS. Unfortunately,

at the time of the preliminary PTA studies, there were no clear defined reporting standards in place, leading to variable study methodology and endpoints. There are no large randomized control level one studies to assess PTA for CVS, making it difficult to draw conclusions on the outcomes of PTA, and make comparisons to alternative technologies.

The work of Gerald Beathard set the tone for the debate in the early 1990s. In his pivotal study, stenoses were identified by venography in patients who met a set of clinical criteria indicating the need for evaluation. The lesions were classified by location and type. Central lesions had the worst secondary patency with only 28.9 % of all lesions remaining patent at 180 days, compared with a secondary patency 61.3 % for peripheral lesions treated with PTA alone ($p<0.01$) [61]. PTA has demonstrated a variable technical success rate ranging from 70 to 90 % [31, 61–66]. A PTA study by Kovalik et al. in 1994 [60] made some interesting observations, including a technical failure rate of 7 %, with greater than 50 % improvement (nonelastic lesions) in 70 % of patients with CVS, and less than 50 % improvement (elastic lesions) in 23 % of patients with CVS. 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 felt the histology of the two types of lesions were different based on observations on intravascular ultrasound [62].

Overall, the PTA patency results for CVS demonstrate a wide range of variability. There is a 6-month primary patency range of 23–63 % and a cumulative patency range of 29–100 %. There is a 12-month primary patency range of 12–50 % and a cumulative patency range of 13–100 % [31, 61–66]. One of the largest studies to date on PTA for CVS by Bakken et al. in 2007, comprising of 47 patients, demonstrated a technical success rate of 77 %. There was a primary patency rate at 3 months of 58 %, 6 months of 45 %, and 12 months of 29 %. There was a cumulative patency rate at 3 months of 76 %, 6 months of 62 %, and 12 months of 53 % [66].

Technical failures will occur in a minority of patients when treating CVS with PTA in the range of 10–30 %. There is clearly a subgroup of CVS patients with elastic lesions, unresponsive to PTA. It is also apparent that multiple repeated interventions with close surveillance are required with PTA for CVS, to maintain patency and prevent occlusion over the long term.

Guideline 20 of K/DOQI recommends percutaneous transluminal balloon angioplasty (PTA), with or without stent placement and is considered the preferred approach to CVS [67]. PTA usually provides excellent initial results, but the long-term primary patency is not optimal. Among 50 CVS in a series of 862 venous stenoses, an initial success rate of 89 % was followed by primary 6-month patency of only 25 % [61]. In contrast, peripheral venous angioplasty had an initial success rate of 94 % and a 6-month primary patency of 77 % indicating a different response of central veins to angioplasty. This is probably due to their greater elasticity and recoil than peripheral veins. Postoperative surveillance, either by clinical examination or by angiography, is necessary to detect recurrence of the lesion. Multiple procedures are usually needed.

It is important to note that in patients with a pacemaker, angioplasty can be successfully performed with pacemaker wires in place [20–22]. In a study by Asif et al. 28 consecutive patients underwent PTA procedure. Technical success was 95 %. Postprocedure clinical success was achieved in 100 % of the cases where the procedure was successful. The primary patency rates were 18 and 9 % at 6 and 12 months, respectively. The secondary patency rates were 95, 86, and 73 % at 6, 12, and 24 months, respectively. On average, 2.1 procedures/year were required to maintain secondary patency. There were no procedure-related complications. This study finds PTA to be a viable option in the management of PM/ICD lead-induced CVS.

The histologic basis for recurrent stenosis after PTA has been studied in stenotic AVF but not in CVS. Immunohistochemical measurement of proliferating cell nuclear antigen showed a very high proliferative index in 20 restenotic AVF, when compared with 10 primary stenotic AVF [68]. The process was even more significant in diabetic individuals. However, the process of neointimal hyperplasia seen in AVF stenosis may not be applicable to the process of smooth muscle hyperplasia in CVS (Table 18.1).

Table 18.1 Comparing patency rates of PTA

Angioplasty			Primary patency		Secondary patency	
Study	Year	Number	6 month	12 month	6 month	12 month
Beathard [59]	1992	27	29	–	–	–
Quinn et al. [61]	1995	28	81	23	100	100
Surowiec et al. [63]	2004	35	55	43	–	80
Bakken et al. [64]	2007	49	–	29	77	73

18.6.3 Bare-Metal Stents

Bare-metal stents (BMS) were first placed in the dialysis access circuit, for refractory stenoses by Gunther et al. in 1989 [67]. BMS are the second-generation technology and second-line treatment for CVS. BMS provide mechanical support to a site of stenosis which is resistant or unresponsive to PTA. BMS are potentially useful in CVS in the setting of the following: kinked stenoses, elastic stenosis post-PTA, sealing dissections or circumscribed perforations post-PTA, establishing and maintaining patency of chronic central vein occlusions, and post-PTA of highly resistant stenoses.

However, there are significant limitations to BMS. Post-deployment, BMS may migrate, shorten, or fracture on a subacute or delayed basis. BMS placement may preclude future endovascular procedures or surgical revision. It is also clearly evident that all BMSs incite intimal hyperplasia, leading to recurrent stenoses and multiple repeat interventions to maintain patency.

The use of BMS in HD access PTA interventions has significantly increased from 0 % in 1991 to over 9 % in 2001 according to the United States Renal Data System [69, 70]. The exponential increase in BMS usage in HD access procedures has led to the development of guidelines for its applications. The Society of Interventional Radiology Quality Improvement Guidelines recommend BMS be reserved for central vein lesions in which PTA has failed or that recur within 3 months after initially successful PTA or rupture after PTA [52]. 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 [71–73].

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 utilized for CVS. The first-generation self-expanding stent is the Wallstent™ (Boston Scientific). The Wallstent™ is constructed of 18 filaments of Elgiloy woven into a mesh. The advantages of this stent include low profile, flexibility, and radiopacity. The disadvantages of this stent include foreshortening at the time of placement, eccentric loading (stenosis) which can lead to concentric narrowing and decreased radial strength, and rare delayed shortening and migration [74–78].

The second-generation self-expanding stents are the nitinol stents. Nitinol is 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, which is preset by the ratio of nickel and titanium and high-temperature heating. When nitinol transforms to its higher temperature crystalline form (28–33 °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 attempt to return to its original shape over time, or if the external force is removed [77–80].

The results for BMS demonstrate a wide range of variability. The vast majority of the literature demonstrates a very high technical success rate, in the range of 100 %. There is a 3-month primary patency range of 63–100 % and a secondary patency range of 72–100 %. There is a 6-month primary patency range of 42–89 % and a secondary patency range of 55–100 %. There is a 12-month primary patency range of 14–73 % and a secondary patency range of 31–91 % [57, 58, 62–66, 81–86]. One of the largest retrospective studies to date on BMS with Wallstent™ for CVS by Haage et al. published in 1999 with 50 patients demonstrated a 3-month primary patency rate of 92 % and 6- and 12-month primary patency rates of 84 and 56 %, respectively. There was a secondary patency rate at 6 and 12 months of 97 % [84]. Unfortunately, these results have not been replicated elsewhere in the literature. Another retrospective study on nitinol BMS for CVS by Vogel et al. in 2004 [79] with 16 patients demonstrated 3-, 6-, and 12-month primary patency rates of 81, 74 and 67 %, respectively. Secondary patency rates were not reported in this study [81].

There are no randomized control trials to date, comparing PTA and BMS in the setting of CVS. Another retrospective study by Bakken et al. [64] published in 2007 comparing PTA and BMS for CVS demonstrated 3-, 6-, and 12-month primary patency rates with PTA of 58, 25, and 29 % in comparison with 3-, 6-, and 12-month primary patency rates with BMS of 65, 54, and 45 %. There were 3-, 6-, and 12-month secondary patency rates with PTA of 76, 62, and 53 % in comparison with 3-, 6-, and 12-month secondary patency rates with BMS of 72, 55, and 46 %. There was no significant difference in patency results between the PTA and BMS group.

In summary, it appears BMS for CVS demonstrate a high technical success rate. There is clearly a group of CVS patients, who are unresponsive to PTA and will require BMS to achieve technical success. However, there is no literature to date demonstrating the superiority of BMS over PTA in the setting of CVS. Future randomized control trials will be needed to determine the appropriate role of BMS for CVS. See Figs. 18.5, 18.6, and 18.7 below.

18.6.4 Covered Stents

Covered stents (CS) also known as peripheral endografts have been proposed as a new treatment option for CVS. The potential advantages of a CS would include providing a relatively inert and stable intravascular matrix for endothelialization while providing the mechanical advantages of a

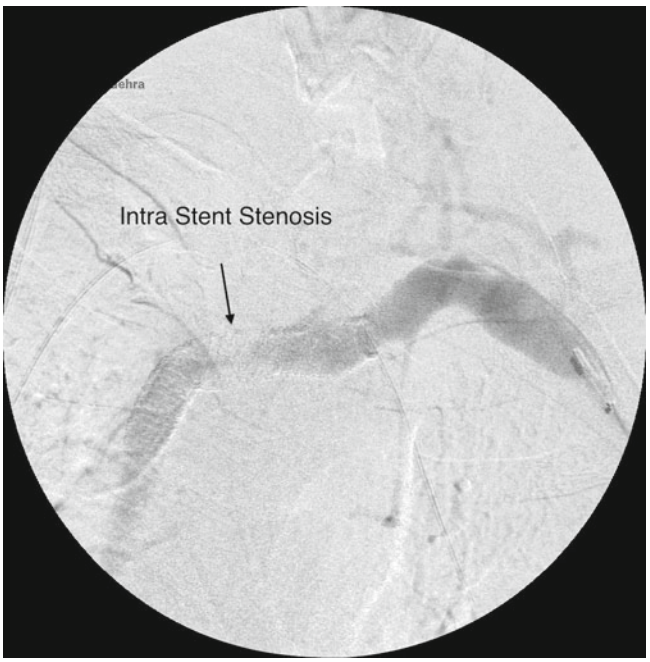

Fig. 18.5 Intrastent stenosis in L brachiocephalic vein

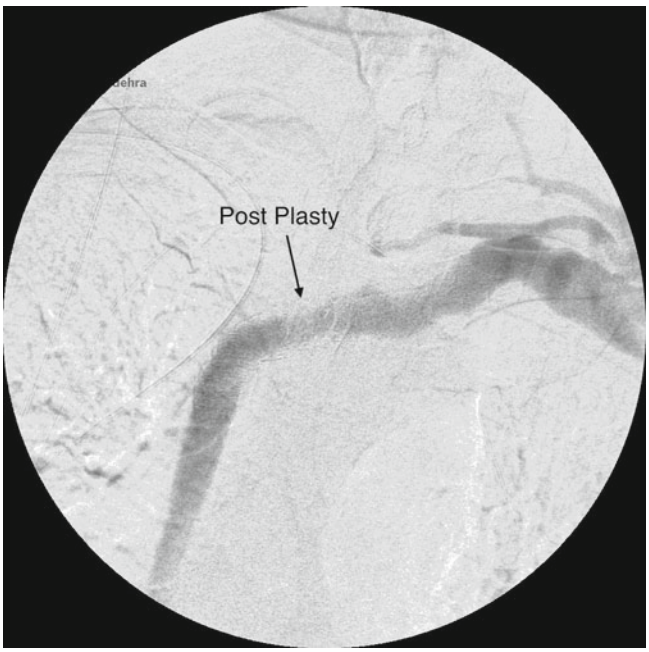

Fig. 18.7 Post-PTA result of intrastent stenosis

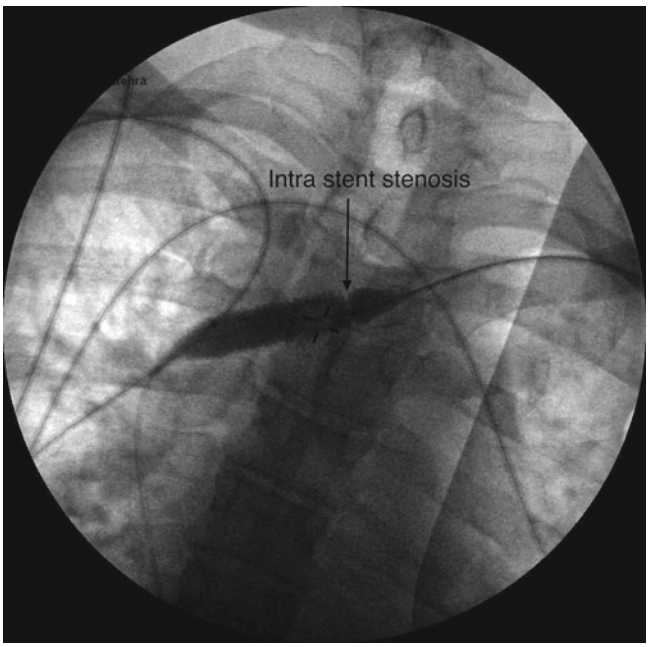

Fig. 18.6 PTA of intrastent stenosis

BMS. This could potentially reduce the intimal hyperplastic response, causing restenosis post-PTA or BMS placement. CSs 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 CS usage in the HD access circuit. Most of the literature to date has been on the treatment of graft or outflow vein aneurysms and refractory venous outflow stenosis. A recent study by Jones et al. looked into the role of CS in CVS. Their results indicated primary patency of 67 and 45 % at 12 and 24 months, respectively. Secondary patency rates reported were 80 and 75 % at 12 and 24 months [14]. Another recent study by Anaya-Ayala et al. also reported 100 % technical success like Jones study. Their 12-month primary and secondary patency rates were 56 and 100 %, respectively [39]. These studies have shown superiority over BMS and PTA and are the steps in the right direction. Further randomized studies are needed to confirm these findings. One of the concerns with covered stents is the risk of jailing other central veins (Table 18.2).

18.6.5 Surgery

Access Abandonment

The simplest surgical solution for access-associated CVS is ligation of the access, which results in immediate relief of symptoms [87–90]. At the same time ligation is the most frustrating option as the vascular pathology causing the patient's problem is not corrected and the respective extremity is rendered unsuitable for further access procedures. Access abandonment requires placement of further catheter, while new access is being planned resulting in increased morbidity of these patients. This may result in development of CVS on contralateral side as well, which may preclude further access creation on contralateral side. Therefore

Table 18.2 Comparing patency rates of stents

			Primary patency		Secondary patency		
Study	Year	Number	6 month	12 month	6 month	12 month	Stent type
Stent after angioplasty failure							
Quinn et al. [61]	1995	28	67	11	100	89	Gianturco
Haage et al. [82]	1999	50	84	56	97	97	Wall
Vogel et al. [79]	2004	15	74	67	–	–	Smart
Jones et al. [85]	2011	30	81	67	100	80	Viabahn
Anaya-Ayala et al. [86]	2011	25	–	56	–	100	Viabahn
Primary stent placement							
Bakken et al. [64]	2007	26	–	21	–	46	Wall

access ligation should be considered as the last resort, only when interventional or other surgical therapy of CVS is unreasonable or has failed.

Surgical Reconstruction

Based on data obtained from the few reports in the literature [91–96], the results of surgical reconstruction of mediastinal veins in ESRD patients are better than those of interventional procedures with primary patency rates of 80–90 % at 12 months. These procedures, however, always mean major surgery. Patch angioplasty of a subclavian or brachiocephalic veins or orthotropic bypass surgery [93–96] requires clavicular division or sternotomy (and general anesthesia) and is associated with high rates of postoperative morbidity and mortality. Extra-anatomical bypass (such as axillary-to-internal jugular vein) [91, 92, 96] is less distressing to the patient, but this results in the loss of another central vein for further access.

Advanced Procedures

When the central venous drainage of all four extremities is compromised, construction or maintenance of AV access can be difficult or impossible. In low-risk patients fit for median sternotomy, a subclavian artery-to-right atrial appendix bridge graft [97] can be constructed, or an axillary vein-to-right atrial bypass [94] be performed. In patients unfit for major surgery, fashioning an arterio-arterial loop graft [98, 99] can be considered as an alternative to the insertion of a translumbar, transhepatic, or transthoracic, cuffed tunneled catheter [69, 70, 100].

Future Directions

Future treatments may include coated drug-eluting stents with rapamycin or paclitaxel to prevent development of neointimal hyperplasia inside stent. Other alternatives may include brachytherapy with beta radiation, which has shown some benefit in coronary circulation.

The greatest impact will be achieved as we evolve our understanding of various hemodynamic, molecular, pathologic, and genetic factors of CVS. This might result in the development of newer treatments and preventative strategies that may be critical to improve the patency of CVS lesions.

Conclusion

Prevention of CVS in HD patients is paramount like any other condition. Central venous catheter placement is the most important risk factor for CVS. Central venous catheter placement should be avoided if at all possible, particularly in the subclavian vein. The use of other peripheral lines should be minimized to preserve future peripheral and central venous capital as potential access sites. This would need close collaboration between nephrologists and internists. Policies need to be created for CKD patients specifically dealing with this issue.

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

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