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Chapter

Hemodialysis Vascular Access with Central Venous Disease

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Abstract

Vascular access (VA) for hemodialysis (HD) is the lifeline of a patient. Arteriovenous fistula (AVF) is the gold standard of VA, but there are challenging situations when providing long-term VA becomes challenging, in the presence of central vein stenosis (CVS), which is common in patients on hemodialysis, but its exact prevalence is not known. It would be ideal to have proper venous mapping with imaging modality to be able to plan central venous access. This prior venous mapping will help to plan the target vein and delineate venous path to be able to place HD catheter in the best position or resolve the VA-related problems. However, digital subtraction angiography remains the gold standard of the procedure, during which the target vein is accessed via ultrasound guidance, and subsequent passage of wire is done under fluoroscopic guidance. Venous angiography and, if indicated, angioplasty are performed. For complete chronically occluded thrombotic veins, recanalization needs to be attempted. Stenting is reserved for a select group of patients. There are advances in endovascular techniques to deal with CVS, and it needs a multidisciplinary team approach to tackle the complex issues of VA-related central venous disease (CVD).

Keywords: hemodialysis vascular access, central venous disease, central venous stenosis, central vein angioplasty, central vein stenting, complete thrombotic occlusion of central veins

1. Introduction

Long-term hemodialysis (HD) is dependent on reliable vascular access (VA), which is the lifeline of a patient. We have come a long way in the chronic HD treatment due to advances in VA. Starting from Scribbner's shunt to single-lumen HD catheters in the femoral vessels to double-lumen non-tunneled non-cuff HD catheters, to tunneled cuff catheters (TCC), and to early stick arteriovenous grafts; all for urgent start HD. Over the years, we also moved the catheters from subclavian veins (SCV) to internal jugular veins (IJV), for VA in the upper part of the body, basically for access draining to the superior vena cava (SVC). Unfortunately, the non-cuffed HD catheters became a tool to continue HD for a prolonged period (a practice very commonly encountered in the part of the world where the author is working, mainly due to nonavailability of VA expertise) in the absence of matured arteriovenous fistula (AVF) or graft. This led to injury to the vessel wall, leading to thrombosis and central vein occlusion and compromised VA for HD due to central venous stenosis (CVS). Even the TCCs are associated with CVS. Two major factors

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implicated in development of CVS are venous trauma resulting from cannulation of central veins and hemodynamic stress secondary to high flow due to access site AVF, causing central venous disease (CVD).

AVF is the gold standard of VA. Ideally, all patients starting HD should have AVF in place, but that is not possible. In 2015, 80% of patients were using a catheter at HD initiation, a rate that has changed only marginally since 2005, and at 90 days after the initiation of HD, 68.5% of patients were still using catheters [1, 2]. Late referral by nephrologists to surgeons has been an underappreciated cause of initiation of HD with central catheters. [3]. There are situations when a patient has multiple AVF failures, in the upper limbs. It then becomes a challenging situation to provide dialysis to these patients with a reliable access. The problem is further compounded in patients with prior central vein HD catheters, resulting in CVD. CVS is considered to be common in patients on hemodialysis, but its exact prevalence is not known. The CVS may have occurred due to insertion of central catheters, PICC, or pacemaker leads. Due to direct contact of these devices with the wall of the central veins, and the constant movement, both lateral (like a pendulum) and cephalocaudal direction associated with breathing and the cardiac cycle cause endothelial injury. Pathological examinations of central veins obtained at autopsy have shown that even short-term catheters are associated with foci of local intimal injury with endothelial denudation and adherent thrombus [4].

In patients with AVF, development of CVS is partly related to turbulent blood flow and neointimal hyperplasia (NIH). Infection related to prior catheter insertion may also be responsible for CVS. Extrinsic compression, either musculoskeletal or arterial, can be contributing to CVS.

However, in an otherwise healthy person, the CVS hardly, if ever, causes problems. The problem comes to light when an AVF or graft is placed on the ipsilateral side where there is presence of CVS. It also gets recognized when a fresh attempt is made to insert cuffed or non-cuffed tunnel catheter in the central vein. We should realize that CVS leading to CVD is difficult to treat and often resistant to treatment. In CVD, VA for HD sometimes need to be abandoned, or in serious cases, the patient's life may be threatened. Therefore, one should strive for the ideal situation of catheter avoidance and central vein preservation and remember that prevention is ideal and better than cure.

2. Clinical case study

This is a 52-year-old diabetic female, who was on regular hemodialysis 3 times a week with right internal jugular vein (IJV) TCC as her vascular access for 7 months and a non-maturing left brachiocephalic vein AVF. She presented with progressively increasing orthopnea of 1-week duration and was presumed to have coronary artery disease. She was referred to our hospital cardiology department for coronary angiography (CAG), who subsequently asked nephrology services to give her a post CAG dialysis. Her CAG was normal. When on dialysis and on attempting to ultrafilter her, she had intradialytic hypotension, with persistent and increasing breathlessness. She was examined by a consultant and a clinical diagnosis of CVS was made. The patient was clinically not in fluid overload state. HD was terminated, and her blood pressure and symptoms settled. The next day, she underwent contrast-enhanced tomography of central vein, which proved stenosis at the cavo-atrial junction, at the site of the tip of the dialysis catheter. She was subjected to central vein angioplasty and her symptoms resolved (**Figure 1**) (author's personal work).

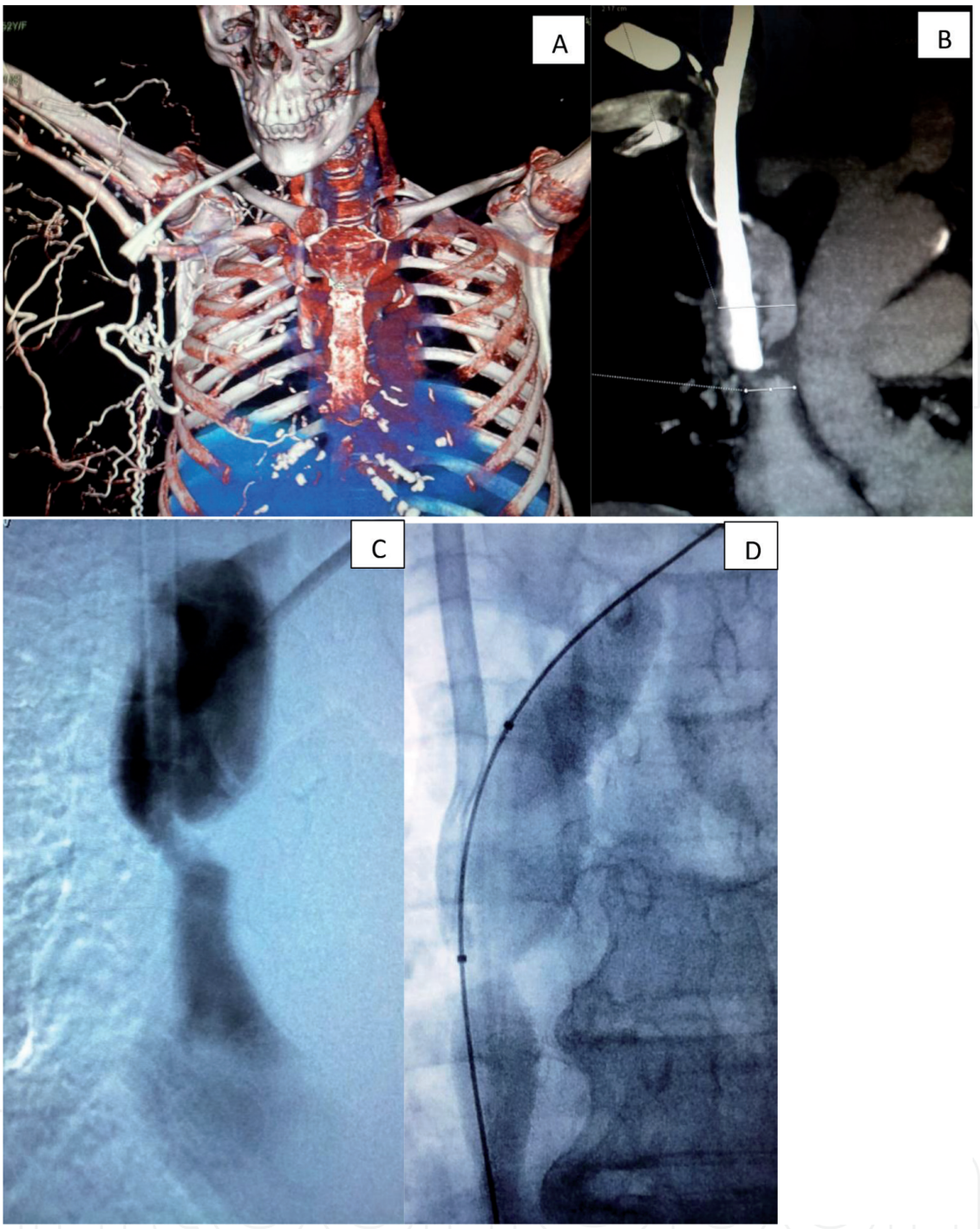


Figure 1.
CT scan and angiography images: (a) 3D reconstructed image showing clots around the TCC tip and also multiple collateral veins in the thoracic cage, suggestive of CTO; (b) stenosis at cavo-atrial junction at the site of TCC tip; (c) findings of (b) on CT scan are confirmed on angiography; and (d) angioplasty of the CVS with TCC in situ.

3. What are central veins?

In the neck, SVC, bilateral brachiocephalic vein (BCV), IJV, external jugular vein (EJV), and SCVs would constitute the central veins for upper limb access, whereas femoral, common iliac, and external iliac veins and the infrarenal inferior vena cava could constitute the lower limb central veins.

4. Preservation of central veins

Since CVS precludes to a creation of successful VA for long-term HD, it is essential to spread the awareness about preservation of central veins. It is a common practice to preserve peripheral veins (especially in nondominant hand) in a patient with chronic kidney disease (CKD, G III or higher). This is known to residents, fellows, nursing staff, etc. However, the concept of preservation of central veins in CKD is not widespread. CKD patients do get repeated central catheters at pre-HD stage due to medical problems, or PICC line, or the cardiologists requiring to put cardiac rhythm devices. There needs to be a dialog with cardiology colleagues to try and avoid insertion of SCV leads in CKD patients who are going to need HD in future. They can be requested to go for epicardial lead pacemakers (**Figure 2**) (author's personal work). Also, stiff non-cuffed HD catheters should not be kept in situ in jugular veins for more than 15 days. Even if patient has acute kidney injury and likely to need prolonged HD beyond 15 days, it is essential to change them to TCC, which has lesser chances of CVS than non-cuffed catheters. SCV catheterization should not be performed in CKD patients, although it still occurs frequently. Many intensivists prefer SCVs for central line insertion, and they should be convinced to avoid it in CKD patients.

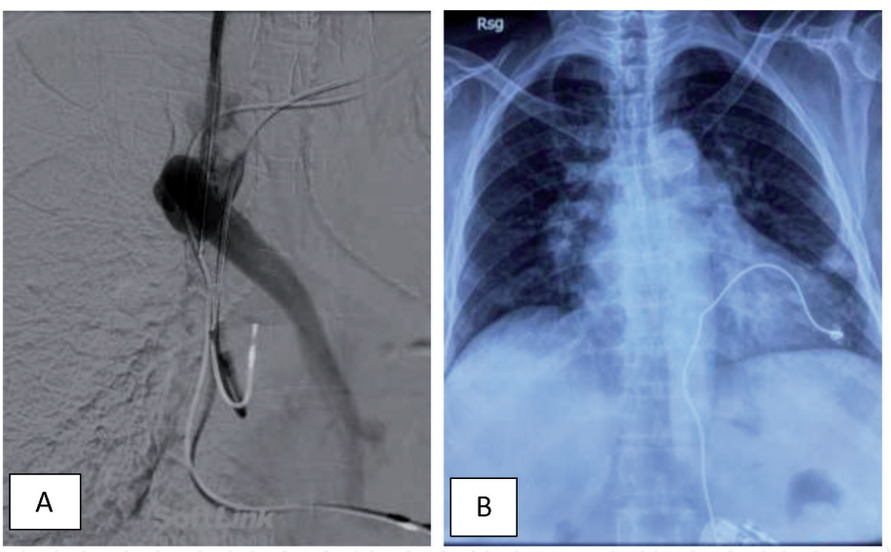


Figure 2.
(A) Pacemaker lead, with TCC. Pull back angiograph shows SVC occlusion and filling of azygous vein. (B) Pacemaker with epicardial leads in a patient with CKD GIIIb due to DKD.

5. Pathophysiology

CVS can be either related or unrelated to current or previous catheters. Catheter-associated CVS are related to type of catheter, i.e., stiff non-tunneled or softer tunneled catheter, duration, number of catheters in the same site, vein site, and tip position are all related to CVS. The material of catheter can be responsible for platelet aggregation and thrombosis. This can lead to episodes of catheter-related bloodstream infections (CRBSI). Also, the blood turbulence produced due to tip design can be responsible for CVS. Traditionally, catheters inserted in SCVs are known to cause CVS, as the SCV is located between the clavicle and first rib. However, left IJV or EJV catheters passing through the left brachiocephalic veins are responsible for left BCV stenosis. In the left BCV, stenosis occurs at two points, at the junction of the

left IJV to the left SCV and junction of the left BCV to the right BCV continuing as the SVC. So, the stenosis occurs at the contact points of catheters with vessel wall in this situation. Also, left BCV is situated between aorta and sternum and can contribute to stenosis. Right IJV catheters were not considered to cause CVS; however, prolong catheters in the right IJV, passing through the right BCV, can also cause right BCV CVS. Catheter tips in the SVC or SVC-right atrium junction are also known to cause thrombosis and/or CVS at the tip position site, (as illustrated in the clinical case above). Catheters on the ipsilateral side of cardiac rhythm device lines or ipsilateral AVF are also known to cause CVS. In the later situation, it is due to turbulent blood flow at high flow generated by AVF. This is more prominently seen in cephalic arch with proximal brachiocephalic AVF and rarely with distal radio-cephalic AVF.

6. Asymptomatic CVS

Patients with prior history of central catheters or pacemaker leads may have CVS but are usually asymptomatic. The CVS comes to notice only when a VA is placed on ipsilateral side of CVS or a patient undergoes imaging for some other indications. Even in the presence of VA, asymptomatic CVS (<50%) are sometimes detected during other radiological procedures. These are usually found in cephalic arch or brachiocephalic veins. If these lesions are asymptomatic, one need not do intervention, even if stenosis is >50%. These require only observation for development of any symptoms or signs. This is because central veins are more elastic and prone to recoil after angioplasty. Also, the intimal damage to the veins caused by angioplasty balloon (cracking and fissuring of the vessel intima) may accelerate further stenosis, due to aggregation of thrombocytes and occurrence of thrombi leading to intimal hyperplasia and fibrosis at the site of the original stenosis [5, 6].

Prophylactic treatment of a stenosis that fulfills the anatomic criteria (>50% diameter reduction) but is not associated with a hemodynamic, functional, or clinical abnormality is not warranted and should not be performed. This is especially important for central venous lesions [7]. Two studies in one cohort were performed to address the issue of likelihood of developing CVS. Among 2811 patients, central venous stenosis was diagnosed in 120 (4.3%), at a median dialysis vintage of 2.9 (interquartile range, 1.8–4.6) years. Among a subset of 500 patients, all with a history of catheter use, 34 (6.8%) developed central venous stenosis, at a rate of 2.2 per 100 patient-years. The incidence of central venous stenosis was higher with a larger number of previous catheters [relative risk (RR), 2.2; 95% confidence interval (95% CI), 1.6–2.9]and pacemaker insertion (RR, 3.9; 95% CI, 1.7–8.9) and was lower with older age (RR, 0.7 per decade; 95% CI, 0.6–0.8) [8].

7. Diagnosis of CVS in symptomatic patients

The diagnosis of CVS is made from clinical and imaging findings. Most patients will have a history of previous central venous catheter placement and will present with ipsilateral arm, breast, face, or neck swelling. Many patients will have evidence of AV access dysfunction, with decreased access flows, increased venous pressures during dialysis, and a history of excessive bleeding from the puncture site after removal of needle. CVS leading to venous hypertension (VH) in the ipsilateral extremity and chest wall is a frequently encountered problem affecting 17 to 40% patients on HD. On physical examination, there may be numerous dilated collaterals in the neck or chest and arm edema or dilated tortuous draining veins of fistula on the side of the CVS. In the cases of bilateral innominate vein or SVC

stenosis or occlusion, patients may present with SVC syndrome. CVS can often be diagnosed by duplex ultrasound, with an absence of normal respiratory variation in the diameter of central veins and polyphasic atrial waves. It is difficult to visualize the central veins with duplex ultrasound in obese and muscular patients [9]. The author has seen patients with severe CVS presenting with not only ipsilateral limb edema but hemifacial swelling, diminished vision, and hearing loss on the right side and throbbing headache. We have also encountered a patient in whom there was no prior history of any catheters or lines but had narrowed fibrosed right IJV. This was detected at the time of insertion of TCC for initiation of chronic dialysis. In this particular patient, right EJV was patent (indirect evidence that the right IJV was blocked for a long time, but patient was asymptomatic). We had a patient with right upper limb oedema and history of multiple right IJV non-cuffed catheters for dialysis but never had any SCV catheters. During angiography, right SCV was found to be thrombosed. As per the European Best Practice Guidelines on VA [10], if symptomatic CVS is suspected, digital subtraction angiography (DSA) of the access and the complete venous outflow tract should be performed. In certain cases, ultrasonography with Doppler, computerized tomography (CT) of central veins or magnetic resonance imaging (MRI) plain study with time of flight (TOF) technique (**Figure 3**) may be deployed prior to DSA for venous mapping.

The main regions of central vein which are affected are the right BCV or SCV, right IJV, left BCV or SCV, and left IJV or SVC. The problem of cephalic arch stenosis in proximal brachiocephalic AVF (prevalence ~30%) or sometimes due to radio-cephalic AVF should also be kept in mind. Percutaneous transluminal angioplasty (PTA) with balloon dilatation is the primary basis for endovascular therapy. However, balloon dilation should be performed only if there is a clinical indication such as arm or face swelling. Several studies have reported that balloon dilation for a narrowed lesion found incidentally on angiogram and without symptoms accelerates lesion growth [11–13].

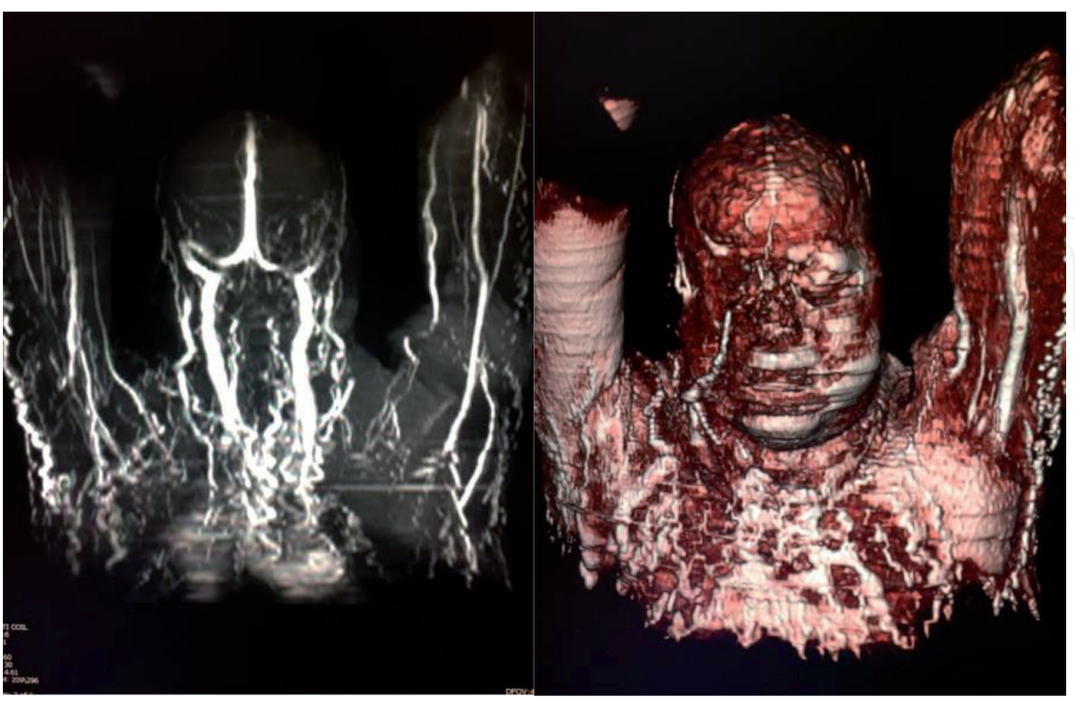


Figure 3.
MRI using TOF protocol, without contrast, to have venous mapping prior to intervention, in a patient who was grossly edematous and had no veins available to perform CT venography. It showed multiple levels of CVS, including bilateral IJVs, EJVs, and bilateral BCVs.

A major problem with lesions in the central veins is that many are elastic. It has been postulated that there are actually two types of lesions based upon their response to angioplasty, elastic and inelastic [14].

It is reasonable to perform PTA for central venous lesion if this intervention is required to maintain stable dialysis therapy. However, the presence of large fresh thrombus at the lesion site represents a contraindication to PTA unless the thrombus can first be removed by thrombectomy or another method. The left brachiocephalic lesions can be due to not only compressive stenosis but also organic stenosis. In particular, the left brachiocephalic vein may be compressed between the sternum and the right brachiocephalic artery [15].

8. CVD and similar conditions

The lesions can be partial but significant (>60% stenosis, for intervention purpose, however, venography demonstrating more than 50% stenosis of the subclavian, brachiocephalic vein, and superior vena cava in the upper extremity or the iliac vein or inferior vena cava in the lower extremity is considered central venous occlusion) or complete thrombotic occlusion [or chronic total occlusion (CTO)]. In addition, patients with TCC present with catheter malfunction and may have catheter-related sheath (CRS) (old term: fibrin sheath), with or without clot at the catheter tip (called catheter-related atrial thrombus, CRAT).

9. Options for patients with CVS needing VA

They can change to peritoneal dialysis if feasible. One can plan for thigh AVF or graft. But if both these are not the options, one is left with exploring central venous access with angioplasty and insertion of HD catheters, with TCC or Hemodialysis Reliable Outflow (HeRO®) device. For the purpose of this chapter, we would focus on upper limb CVS. Not to forget the fact that in the presence of SVC stenosis, no AVF or upper limb grafts will be feasible if the SVC stenosis is tight and has a chance for recurrence in spite of SVC angioplasty with or without stent. With improved dialysis survival, there are increasing numbers of patients who have exhausted definitive access options due to central venous stenosis and are maintaining dialysis on a central venous catheter. The HeRO allows an alternative by providing a definitive access solution [16].

10. The procedure: approaching and crossing stenosis

The approach to central vein could be from femoral veins, internal jugular veins, and brachial or cephalic veins. One needs to have thorough history, clinical examination, and Doppler study to arrive at a conclusion about suspected site of CVS. When these are unable to point the site of likely lesion, one may perform contrast-enhanced computer tomography (CT) venogram. The problem with CT venography is the need to inject iodinated contrast. This can be a problem in HD patients at times, who have no peripheral access to inject contrast. In such cases, magnetic resonance (MR) venography without gadolinium, using time-of-flight protocol, may yield equally good results and help plan the procedure. Gadolinium should be avoided in patients with eGFR of <30 ml/min due to risk of nephrogenic systemic fibrosis.

Once the procedure of angiography is decided, one needs to be ready with proper hardware, including various types of wires (0.35 and 0.14 regular and double length wires, hydrophilic wires, and stiff wires), various types of diagnostic and guiding catheters, larger-size high-pressure balloons, stents, or stent grafts. One may need large length vascular sheaths to provide stability to the catheter and guide wire, especially when femoral venous access is used.

10.1 How are the target lesion approached?

The indications to perform central vein angioplasty are either the patient is symptomatic with functioning vascular access in the upper limb, or the patient has had multiple vascular access failures in upper limb, and no reliable long-term vascular access is available. In such cases, the aim of central vein angioplasty is to create a passage to insert central dialysis catheter, which can be either TCC or hybrid graft if a reliable arterial inflow is available.

If the target lesion is right brachiocephalic vein or SVC, the right IJV is punctured, lesion attempted to be crossed with 0.014′ hydrophilic wire. If that is not successful, the right femoral vein is punctured and the lesion approached. The combination of this approach from either side helps take simultaneous angiographic shoots to define site and extent of lesion in terms of its length and diameter. We have encountered several cases, in which the thinnest of wires, sometimes even the coronary wires, or CTO wires (chronic thrombotic occlusion) fail to cross the lesion. In such cases, recanalization is attempted, which is described as sharp needle recanalization. When traditional endovascular methods fail, experienced interventionalists may utilize sharp or radiofrequency (RF)-assisted recanalization techniques. Sharp recanalization techniques require the use of the back end of a wire, 20- to 22-gauge (15–20 cm) percutaneous or transseptal needles, or transjugular intrahepatic portosystemic shunt needles [17]. In the absence of any other alternatives, we have been doing it with a long Chiba needle. One has to have simultaneous projections in anterior-posterior and lateral views to be sure that the puncture is in the proper venous plain and perforation is avoided. There is no true vessel wall, and a stent is placed to reconstitute the vessel wall if this is being done for upper extremity vascular access preservation. Otherwise, a catheter is inserted after balloon angioplasty of target lesion and creating enough venous diameters to allow smooth passage of the dialysis catheter. Recently, a new device called Surfacer® Inside-Out® access catheter system is available. The Surfacer Device offers a new approach for repeated venous access in patients with thoracic central venous occlusion (TCVOs) that enables the avoidance of left-sided catheter placement in individuals awaiting creation or maturation of permanent AV access or in patients who have exhausted all thoracic venous access options [18]. The author has no personal experience of using it; however, it is meant to restore and preserve access in the chronically occluded veins. Its advantages include restoration and sustenance of access, reliable and repeatable central venous access to the right IJV, preserving viability of secondary central veins, and optimization of the placement and maturation of permanent AV access.

TCVO [19], the authors divided TCVO as type 1, defined as any unilateral obstruction affecting either the internal jugular or subclavian vein. Type 2 includes all cases with ipsilateral occlusion of the brachiocephalic vein or ipsilateral obstruction of both internal jugular and subclavian veins. In TCVO type 3, both brachiocephalic veins are obstructed. Type 4 is characterized by central obstruction of the SVC. Thirty-six patients with TCVO treated in Vienna, Austria; Oxford, England; or Cologne, Germany, who required hemodialysis access between July 2016 and June 2018, with TCVO and history of multiple CVCs and AVF, were referred to the participating centers for vascular access. Thirty-two (89%) patients were eligible for the inside-out

approach (IOA) approach. Thirty-nine treatments were performed, with seven patients undergoing the IOA procedure for the second time more than 3 months after initial CVC placement. Dialysis access was established successfully in 38 of 39 (97%) implementations of the IOA procedure. Median intervention time was 43 minutes. No complications occurred. This appears to be a promising method, although this was an observational study and no comparison was made to any other methods.

If CVS is bypassed, and adequate inflow with axillary artery is available, the HeRO® device can be implanted, which will avoid the need for external TCC and minimize risk of CRBSI.

a. If the target lesion is in the subclavian vein or cephalic arch or left brachiocephalic vein, approach from arm cephalic, basilic, or rarely brachial vein is required, usually combined with femoral vein approach. In cases of tight CTO, to provide adequate support and stability to the procedure of angioplasty, the wire from the arm vein is passed all the way down to the femoral vein and snared out. The hydrophilic wire is then changed over to a stiff wire, and the procedure of angioplasty is completed.

b. If attempting to put femoral vein or inferior vena cava (IVC) TCC and dealing with iliac vein or IVC stenosis, approach will be from the femoral vein, and again recanalization is attempted with thin wire or sharp needle.

PTA with balloon dilatation is the primary basis for endovascular therapy. If symptoms are present, PTA is performed; however, patency is poor, so repeat procedures are often required. If it is a first PTA and there is no elastic recoil of the vein on table, one should not consider stenting. Recent studies using intravascular ultrasound (IVUS) for CVS showed that, although the lesion was sufficiently enlarged on angiography after PTA, IVUS demonstrated insufficient dilation or extrusion by the balloon catheter [20]. In such cases, repeat angioplasty with larger-size and higher-pressure balloons can be tried, keeping in mind the risk of central venous rupture. Insertion of stents in such situation can be tried. However, numerous studies showed failure of stent use, especially in HD VA, because of neointimal hyperplasia within the stent, leading to a lower patency rate than that of PTA [21, 22].

The stent graft (SG) is a structure that applies graft material to the inside or outside of the stent to create a physical barrier to NIH. Various studies and author's experience have been that even the SGs do not provide long-term venous patency. There have been reports of using drug-coated balloons for CVS; however, at present, the benefit seems limited.

Stenting for TCVO is appropriate in the following situations, provided there are associated hemodynamic or clinical abnormalities: acute elastic recoil (>50%) following PTA and recurrent stenosis within a 3-month period of PTA. If balloon angioplasty achieves insufficient dilation (e.g., severe recoil) or leads to dissection or acute occlusion of the affected vein, bailout stenting is performed. [15]. Balloon angioplasty is a basic treatment for central venous lesion, but stent implantation is sometimes required. The self-expandable or balloon-expandable stent is chosen by the lesion location and characteristics. The lesion in SCV is generally treated by self-expandable stent, and right BCV is treated by balloon-expandable stent. The organic lesion of innominate vein with plaque is treated by self-expandable stent. Note that the innominate venous stenosis is sometimes caused by compression between the right brachiocephalic artery and the sternum and this lesion is treated by balloon-expandable stent because the radial force of balloon-expandable stent is stronger than self-expandable stent. It is important to understand the indication and stent selection for central venous PTA [15].

It is also important to remember to choose a proper-sized stent. This is sometimes not possible to judge from the angiographic images, and what appears to be adequate angioplasty could be inadequate in terms of adequate vein diameter. IVUS is sometimes useful to guide the choice of proper balloon or stent size, as what appears to be an adequately treated vein could be having significant underlying stenosis, which is not possible to realize on conventional two-dimensional angiography. IVUS is an invasive modality that provides cross-sectional imaging of the veins but without the need for ionizing radiation or contrast administration. In addition to being a diagnostic tool that is easy and repeatable, it aids treatment decision-making. Though IVUS may be better suited than traditional venography to identify intraluminal narrowing and pre-/post-intervention outcomes, additional study is warranted to better characterize the value of IVUS in the VA-related CVS patient population. In a patient with CVS and prior allergy to iodinated contrast, angiography was performed using 1 cc of contrast, CVS confirmed, and the rest of the procedure of angioplasty and confirmation of adequate result was completed using IVUS (personal communication, Dr. Daniel Patel, Interventional Nephrologist, Volusia-Flagler Vascular Centre, Daytona Beach, Florida, USA).

If CRS is the cause for catheter malfunction, the same can be confirmed during catheter exchange procedure by doing pullback angiography prior to full removal of TCC. There can be only CRS, which can be tackled by balloon angioplasty and fibrin sheath disruption. Rarely (and in the authors' own experience), the CRS can be snared out. However, the CRS can be associated with CRAT. And it can become tricky to perform angioplasty, as the risk of pulmonary thromboembolism due to large CRAT, attached to TCC tip, is high. Sometimes, CRAT is attached to the wall of the right atrium. In such a case, if TCC is functioning well, the patient can be anticoagulated and kept under close observation. For a large CRAT or infected CRAT, attached to TCC tip, and where it is risky and not advisable to remove the CRAT, AngioVac thrombectomy device can be used. If these patients are poor candidates for conventional therapy of thrombolytics or surgical thromboembolectomy because of bleeding, failure of thrombolytics, or hemodynamic instability, they may benefit from percutaneous mechanical thrombectomy by AngioVac, which is proved to be effective for complete evacuation in most patients [23].

Surgical options may be considered for CVS, when the stenosis persists, and there are no alternative avenues to provide long-term reliable VA. One must assess the AVF for flow velocity, as this may be the cause of CVD. In such cases, inflow reduction either surgically or percutaneously can be performed. Another surgical option is unusual bypass including reconstruction surgery and claviculectomy or first rib resection if there is thoracic outlet syndrome. If an experienced vascular surgeon is available, the bypass surgery can be performed using graft (PTFE) from the brachial artery to ipsilateral or contralateral internal jugular veins or axillary or femoral veins. Direct connection to SVC or right atrium have also been attempted. One must remember that such surgeries are complicated and carries its own risks and complications.

There are occasions when there is coexistence of CVS and stenosis in the draining vein, either juxta-anastomotic or away from the site of anastomosis. In such situation, one must attempt and achieve patency of CVS lesion. Failure to do so can lead to severe swelling of the ipsilateral arm. The best way to relieve symptomatic CVS is closure of AVF. But it is not possible in patients, who have the last surviving access and shortage of vascular estate.

But, sometimes, there are more than one possible approach to a problem, and the opinion varies as per the specialist handling the case. For example, refer to **Figure 4**, a middle-aged lady on hemodialysis with CVS who had undergone angioplasty with stenting and resolution of right upper limb painful edema. She

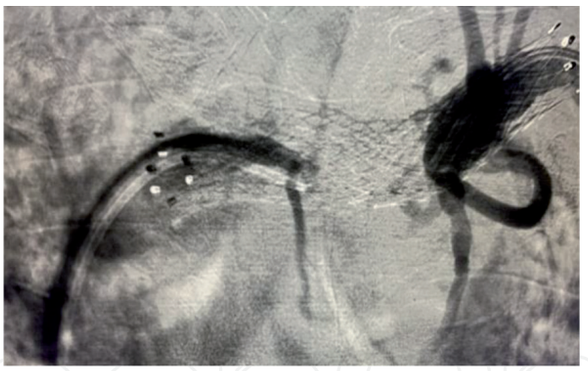


Figure 4.
Right SCV in-stent thrombosis.

presented with the same symptoms 6 months later. The angiography image shows in-stent stenosis. It was difficult to negotiate the stenosis. Various opinions were sought. The interventional nephrologist found that it was the high-flow fistula (flow volume 2600 ml/min, very high) which was responsible for her symptoms, and the patient should undergo flow reduction procedure. The interventional radiologist said that attempt to do in-stent angioplasty, followed by stent placement within the stent, should be the first option. The vascular access surgeon said that the stenosis is due to external compression and the patient should undergo middle 1/3rd claviculectomy to relieve her symptoms.

11. Cardiovascular consequences of AVF

Sometimes very-high-flow AVFs can lead to VH, progressive aneurysmal dilatation of vein with skin ulceration, hemodialysis access-induced distal ischemia, and high-output heart failure with or without pulmonary hypertension. AVFs have effects on cardiac functions related to the increase in preload and cardiac output (CO). It is difficult to define cardiovascular consequences due to AVF in a precise manner. This is due to the fact that patients requiring long-term hemodialysis tend to have volume overload due to water and salt retention. There could also be pressure load due to arterial sclerosis and hypertension and increased CO secondary to chronic anemia. In addition, many hemodialysis patients have significant pre-existing myocardial, valvular, or coronary heart disease, as the vascular calcification due to CKD-MBD (chronic kidney disease—mineral and bone disorder). Congestive heart failure (CHF) is highly prevalent among patients with ESRD. Approximately 35–40% of patients with ESRD have an established CHF diagnosis at initiation of hemodialysis. Worsening in cardiac functions soon after AVF creation has also been observed favoring a causative effect of the AVF on certain cardiac functions [24].

12. Conclusion

CVD is a common occurrence in HD patients, either due to mechanical or hemodynamic factors. It could be related to catheters or AVF. It can be symptomatic or asymptomatic. Interventions will be required in symptomatic patients and in those who have VA malfunction. There are various options, and one needs to choose the modality based on the patient's need. The outcomes cannot be predicted as each vein behaves differently. Proper knowledge of anatomical lesion, pathophysiology of the lesion, patient needs, and expertise available will determine the intervention

modality and outcomes. There is a need for multidisciplinary approach to tackle the situation. We, at our institute, work with a motto which is slightly modified from the old proverb, and we proudly say that "if there's a will and a vein, there's a way." Prevention of CVS by catheter avoidance is an ideal situation; however CVD due to hemodynamic issues not related to catheters cannot be avoided, and VA surveillance is the best way for early diagnosis and management of such lesions.

Acknowledgements

The author acknowledges the support of department of interventional radiology, cardiac catheterization lab, and its staff. Special thanks go to Dr. Gireesh Warawadekar, interventional radiologist and past president of the Indian Society of Interventional Radiology, and Dr. Nikhil Karnik, interventional radiologist. Special thanks to all the fellows, clinical associates, and junior consultants in the department of nephrology, Lilavati Hospital, for patient care.

Conflict of interest

The author declares no conflict of interest.

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