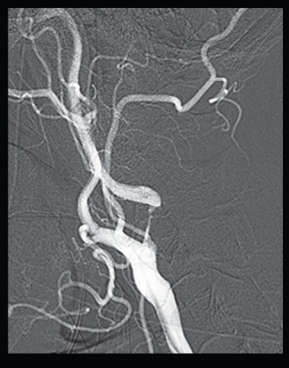
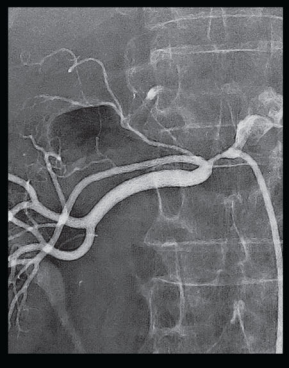
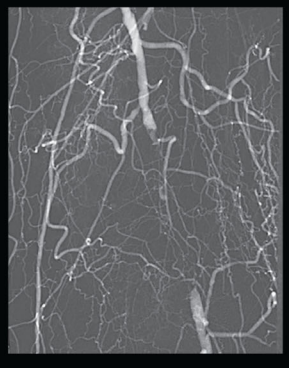
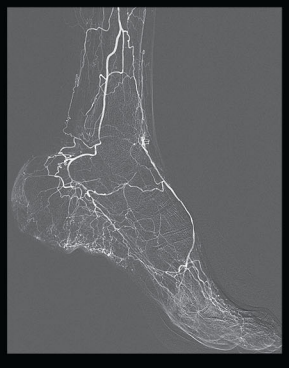
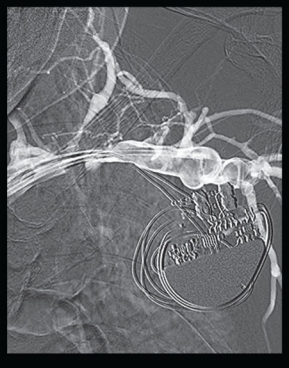
Carotid and Peripheral Vascular Interventions Textbook

Step-by-step technique

Thosaphol Limpijankit

CHAPTER 14

Endovascular Treatment of Central Venous Disease

Thosaphol Limpijankit

INTRODUCTION

Central venous disease (CVD) is narrowing or complete blockage of the large veins in the chest [i.e., superior vena cava (SVC)], brachiocephalic, and subclavian vein. In the past, if deep venous thrombosis presented in the upper body, the main location of involvement was the SVC (1,2). The thrombosis generally resulted from tumors or their treatment such as radiation therapy (3). With the rising use of indwelling subclavian catheters, subclavian and brachiocephalic venous occlusion commonly occurs nowadays. The majority of patients used to have previous hemodialysis catheter or pacemaker wire placed in the subclavian vein (4,5). Subclavian and brachiocephalic occlusion is often asymptomatic, but usually develops sudden edema of ipsilateral upper extremity, face, neck or chest when there is increased blood-flow from an arteriovenous (AV) graft or AV fistula (AVF). Furthermore, CVD might result in diminished vascular access flow, elevated venous pressure, and consequently AV graft or AVF thrombosis.

Surgical management of central vein obstruction has demonstrated durability, with 1-year primary patency of 80-86% (6,7), but it predisposes to be challenging since the blood vessels are deep within the chest. The morbidity rate also has been reported as high as 30% (6). Currently, endovascular intervention is the mainstay treatment of CVD and has shown its efficacy comparable to bypass surgery (8). This procedure using local anaesthesia is well-tolerated by the patients, and corresponds with shorter hospitalization. Percutaneous transluminal angioplasty (PTA) using stent implantation for elastic and recoiled stenotic lesions provides excellent initial results with low technical failure and is able to recover the function of the vascular access (9-11). Nevertheless, frequent or multiple procedures are often necessary to extend long-term stent patency (9,12).

Prevention of CVD is the key to prevent AV access failure as well as other CVD complications. These measures are avoidance of catheter placement in CVD and timely placement of AV fistulas in prospective dialysis patients. In addition, more study of mechanisms of development of CVD and invention of an effective device therapy will probably result in better ways of treating CVD.

ANATOMICAL CONSIDERATIONS

Veins of the upper extremity drain via the basilic or cephalic systems. The basilic vein is located in the upper arm (course along the medial aspect) and the cephalic vein (course along the lateral aspect) connect at the lower border of the teres major muscle to become the axillary vein. The axillary vein proceeds to the lateral border of the first rib, at which point it becomes the subclavian vein, which enters the thoracic inlet posterior to the clavicle and anterior to the first rib and scalenus anticus muscle (costoclavicular space) and connects with the internal jugular vein to form the brachiocephalic vein (13). The most proximal valve is near the venous angle, where the subclavian and jugular veins connect to become the brachiocephalic vein (Fig. 14-1). The left brachiocephalic vein traverses crossways downward, and the right passes steeply downward behind the manubrium. They join together to become the SVC. The left brachiocephalic vein is longer than the right, while neither has a valve. Moreover, the left brachiocephalic vein obstruction may be caused by organic stenosis, or alternatively compression between the right brachiocephalic artery and the sternum (14). This pathology requires balloon-expandable stent to scaffold and maintain blood-flow.

There are additional significant anatomical variations in the paths of left and right central veins. The right internal jugular vein crosses the neck nearly straight into the SVC

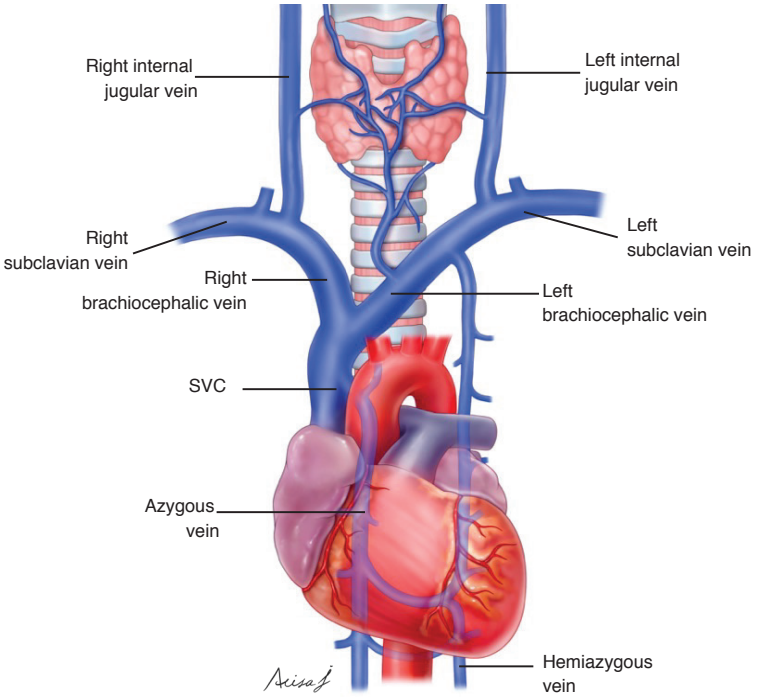


Figure 14-1. Central venous anatomy. Note the anatomical difference in the path of right and left central veins. The route from the right internal jugular vein into the brachiocephalic and superior vena cava (SVC) is almost vertically down. The vein on the left has to take a longer, tortuous course to reach the SVC.

and brachiocephalic vein. The left internal jugular vein merges with the left subclavian vein, and then takes a longer tortuous path to connect with the cavo-atrial junction. The left internal jugular vein has smaller cross-sectional area compared with the right internal jugular vein. These anatomic factors can result in greater contact from an implanted foreign body to the wall of the vessel wall when it is located from the left-handed side. Left-sided internal jugular catheters additionally have more movement than right-sided catheters with rotation of the neck and head, resulting in more endothelial injury and stenosis.

The azygos vein is a vein which drain the intercostal venous system into the lower SVC. The azygos system can potentially connect to the whole venous body supply. Occlusions in one portion of the thorax contribute to divert minimally some of the flow into the azygos system where it is consequently redirected to bypass the occlusion. Any occlusion above the azygos vein can be redirected into the SVC via the azygos system. However, if the SVC is occluded at the location of the azygos, blood can only access the heart via the inferior vena cava (IVC).

COLLATERAL CIRCULATION

For a healthy patient, blood returns to the heart through conventional venous pathways. Blockage of any one of these pathways will cause blood-flow to find new collateral pathways to return the heart (15,16). There are a lot of anastomotic collateral pathways that may potentially bypass the subclavian vein, brachiocephalic vein, and SVC (Fig. 14-2).

With subclavian vein occlusion, superficial and muscular veins around the shoulder, neck and thorax are recruited as collateral pathways, which empty into the azygous veins, jugular veins, or brachiocephalic veins. Shoulder collaterals involve the intercostal veins, suprascapular vein, subscapular vein, and lateral thoracic vein. Vessels involved in the neck collaterals include the inferior thyroid vein, jugular venous arch, external jugular vein, internal jugular vein, and vertebral vein.

With brachiocephalic vein blockage, the principal collateral pathway is up the ipsilateral jugular vein to the brachiocephalic or contralateral jugular veins through multiple head and neck collaterals. With SVC occlusion, collaterals forms as a consequence of the occlusion's position relative to the azygos vein. The azygos vein supplies venous return to the inferior SVC and is the single major vein to supply into the SVC apart from the left and right brachiocephalic veins. As the azygos system functionally connects in some way to the whole venous system, that collateral is an important bypass channel when SVC develops obstruction.

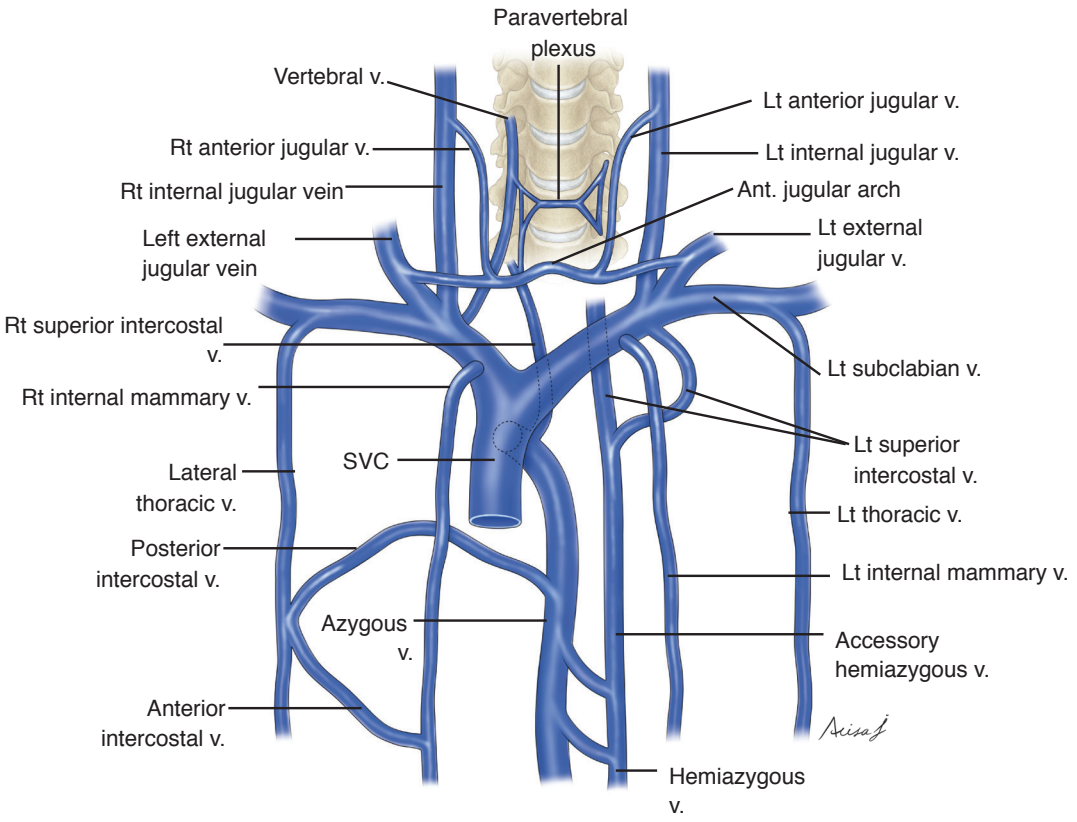


Figure 14-2. Schematic illustration of major collateral pathways that compensate for the presence of central venous obstruction. (Redrawn from Chasen MH, Charnsangavej C. Venous chest anatomy: clinical implications. *Eur J Radiol.* 1998;27:2-14, with permission from Elsevier.) Lt, left; SVC, superior vena cava; V, vein.

PATHOGENESIS AND ETIOLOGIES

Causes of occlusion are varied, and may be categorized into three pathology: intraluminal thrombosis/blockage, narrowing/stenosis, and extrinsic mass effect. Clinically, patients with venous occlusions frequently experience swelling prior to the occlusion location.

For the chest, mass effect from pulmonary or mediastinal malignancy may anatomically occlude venous flow. SVC is the common example of mass effect causing venous blockage. Stenosis or luminal narrowing may also develop secondary to chronic exposure to indwelling catheter, sequela of previous thrombosis, or radiation. Lastly, intraluminal blockage and thrombosis may occur from an indwelling catheter, tumor, hypercoagulable state (leading to acute thrombus), or trauma.

The most common cause of CVD is indwelling catheters including dialysis catheters, pacemaker or defibrillator wires. There has been a strong correlation of CVD, with prior implantation of central venous catheters for dialysis and pacemaker wires (5,17). In one study, 27% of CVD patients had a history of prior central venous catheter implantation (18). Moreover, there is an extraordinary high prevalence of CVD for patients who have a history of subclavian catheters of 42 to 50% in contrast to internal jugular vein catheters (4,19). A suggested mechanisms of CVD development includes central venous catheter-induced injury to the venous endothelium together with secondary inflammatory damage to the vessel wall which occurred during insertion. Other proposed mechanisms include the occurrence of a foreign body within the vein, sliding dislocation of the catheter with respiration, postural and head motility, along with increased turbulence and flow from the creation of an AV access. Turbulent blood-flow has been demonstrated to induce a provocative inflammatory response, platelet deposition, venous wall thickening, and stimulate intimal hyperplasia (20-22). Placement on the left-hand side of neck, location in subclavian vein, longer duration, and placement of multiple catheters, appear to predispose to the progression of CVD.

Venous thoracic outlet syndrome is an extremely rare disorder (1-2 persons in 100,000 population) (23), which can cause progressively axillosubclavian vein thrombosis (Paget-Schroetter syndrome) or effort thrombosis. The subclavian vein is very predisposed to trauma as it

traverses the junction of the clavicle and first rib and within the anterior-most part of the thoracic outlet. Additional to extrinsic compression, repeated forces in that region often result in fixed intrinsic trauma and extrinsic scar tissue development. Once primary thrombosis is identified, catheter-directed thrombolytic treatment is normally successful if initiated within 10-14 days of clot development, but frequently unmasks an underlying lesion. Decompression of the venous thoracic outlet, using thorough external venolysis, resection of the costoclavicular ligament, partial anterior scalenectomy, and first rib excision is essential. When bare metal stent (BMS) implantation is conducted on central venous lesions, care should be given to thoracic outlet syndrome, since they may contribute to complications such as stent distortion and occlusion (24). Since the occurrence of this syndrome is very seldom and is more frequent in young men in their 20_S, so thoracic outlet syndrome is unlikely to contribute the pathogenesis of CVD in dialysis patients.

PREVALENCE

The incidence of CVD is undefined and is probably underestimated because CVD can be asymptomatic. Serial or regular venograms also are not normally conducted following central venous catheter implantation or removal. Most dialysis patients usually become symptomatic in a short time following an ipsilateral AV access is formed as the blood-flow through the developing dialysis access rises. The currently available prevalence is limited to the studies of symptomatic dialysis patients requiring imaging studies. According to several studies, the occurrence of CVD has been published to range between 25% to 40 % (25,26). Previous implantation of pacemaker wires and central venous catheters has been robustly correlated with CVD (18,19), with one study finding that 27 % of CVD patients already had central venous catheters implanted (18). In asymptomatic patients, available studies have shown a relatively high occurrence of CVD in those patients with subclavian catheters (42-50%) in comparison with those with internal jugular catheter (4,19,27). Since CVD or occlusions are not correlated with any clinical findings and unable to identify any predisposing factors, all patients who already have had previous subclavian vein catheters should be assessed to verify the subclavian vein's patency prior to creation of a permanent AV access (28).

RISK FACTORS FOR CVS ASSOCIATED WITH CENTRAL VENOUS CATHETER

CVD is correlated with indwelling intravascular devices including long-term hemodialysis catheter, pacemaker or defibrillator wires, as well as peripherally inserted central catheter (PICC lines). Risk factors for CVD correlated with central venous catheter include:

- Multiple central venous catheter implantations and longer catheter dwell times (4,19)
- Subclavian location (4,19,27)
- Left sided catheterization (29,30)
- Catheter infection (29,31)
- Larger caliber of central venous catheter (12-14 Fr) (32)
- Catheter tip position in the proximal part of SVC (33)
- Catheter composition induced inflammation (e.g., polyethylene and Teflon>polyurethane> silicone) (17,34)

CLINICAL MANIFESTATION

Central venous catheter implantation is the most important CVD risk factor. CVD can be totally asymptomatic and can only be discovered by a venogram taken to prepare for AV access implantation (7,15). Following an ipsilateral AV access is created, CVD will probably become symptomatic abruptly because of increased flow. The symptoms rely upon the particular location of stenosis. While subclavian vein blockage is correlated with edema and venous hypertension of the related upper extremity and chest, brachiocephalic vein stenosis impedes blood-flow from the same side of the face as well as the upper extremity. Bilateral brachiocephalic vein blockage or SVC blockage symptoms are described in Table 14-1.

Table 14-1. Clinical manifestation of central venous disease (CVD) (35)

- Upper extremity edema
- Aneurysmal dilatation of the upper extremity veins and AVF
- Progression of collaterals
- Thrombosis of access
- Venous thrombosis
- Inadequate dialysis
- Recurrent infection
- SVC syndrome

Ipsilateral arm edema usually occurs after a high flow AV fistula or graft is created in that extremity (Fig. 14-3). Use of that access for dialysis frequently aggravates the edema more. Swelling, pain and tenderness in the extremity can resemble cellulitis. Development of tortuous, aneurysmal dilatation of an AVF may exacerbate CVD. Prompt AV fistulogram and correction of stenosis can stop progressive deterioration and rupture of the aneurysm. In chronic CVD, visible, palpable and tortuous veins across the extremity, neck and chest are developed to divert blood-flow centrally. Sometimes, the collaterals are sufficiently large enough to divert blood-flow to alleviate the symptoms and signs of CVD, although in most cases intervention is necessitated.

Significant decline in access blood-flow, episodes of prolonged bleeding from needle sites following dialysis, and raised venous pressures during hemodialysis are the early signs of CVD. Consequently, CVD may reduce access blood-flow and cause insufficient dialysis. An AVF generally stays patent even with low blood-flow, but an AVG is more likely to thrombose. Thrombectomy of these accesses without attempting to diagnose and treat occult CVD can be complicated by worsening of symptoms and recurrent thrombosis.

While infection may be a causative factor for CVD, CVD can also predispose to infection. In condition of venous congestion, access thrombosis with excessive bleeding during repeated needle cannulation for dialysis, can raise the infection risk.

SVC syndrome is the severe manifestation of CVD. It is distinguished by edema of both upper extremities, the neck and face with many dilated collaterals across the chest and neck. Sometimes, the blood-flow can be maintained via a dilated azygous vein. However, if unrelieved with angioplasty or stenting, it may be life threatening and may result in soft tissue edema of the neck with airway compression.

DIAGNOSIS

The diagnosis of CVD can frequently be made or suspicious from a thorough history and physical examination. Prior central venous catheter implantation history, particularly if of multiple and long duration should warn about the potential for CVD. Presence of pacemaker or defibrillator wires should warrant thorough assessment for the presence of CVD and its resolution before placing an AV fistula or graft on the ipsilateral side. Examination revealing swelling of arm on the ipsilateral side and many dilated collaterals in the chest or neck indicates obstruction to outflow.

In patients who have not received central venous catheterization, other etiologies, such as pacemaker wires, thoracic outlet syndrome, hypercoagulopathy, or

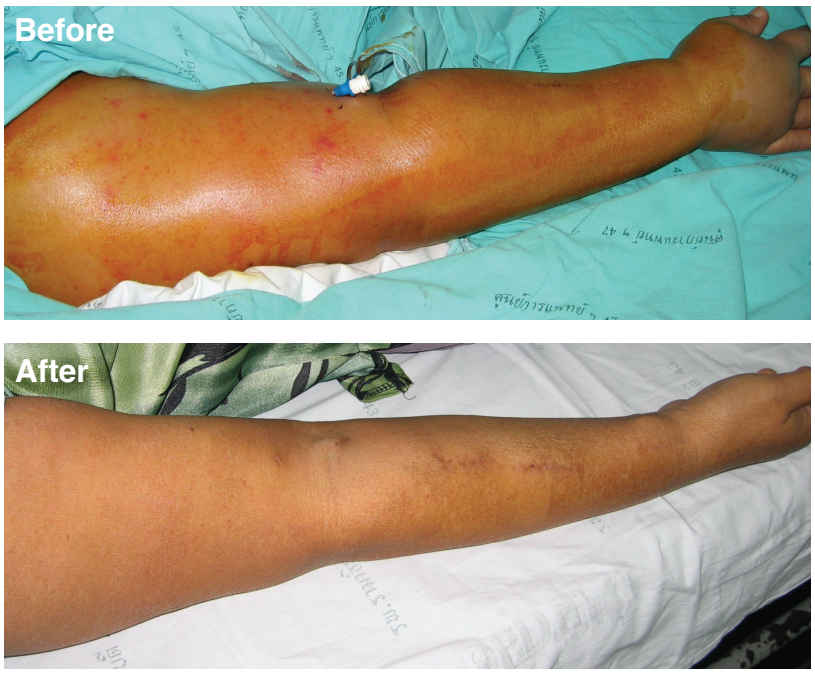


Figure 14-3. Right arm swelling secondary to right subclavian vein occlusion. A: Before endovascular intervention with a 6-Fr sheath in the brachiobasilic AV graft at the forearm. B: After successful balloon angiopalsty 2 weeks, the right arm swelling subsided.

extrinsic compression of mediastinal veins (e.g., mediastinal fibrosis, lymphoma, thoracic aortic aneurysm or goitre), should be considered.

The initial diagnostic study for CVD is color-flow duplex venous ultrasound as this technique is noninvasive with a high sensitivity and specificity (36). A normal vein is completely compressible on ultrasound. Non compressible vein with loss of respiratory variations, polyphasic atrial waves, and no doppler flow augmentation during interrogation indicate obstruction downstream of the probe. In addition, the existence of many neck collaterals generally indicates CVD. A limitation of this technique is potential acoustic shadowing from the clavicle, which may impair visualization of a short segment of the subclavian vein. It can also be problematic to visualize central veins using ultrasound in significant muscle mass or obese patients.

Computed tomography venography (CTV) and magnetic resonance venography (MRV) are increasingly utilized alternatives to x-ray contrast venography (37,38), which is still the gold standard for diagnosing CVD. MRV is useful in patients with advanced chronic kidney disease to avoid radiocontrast and to preserve renal function or in those with radiocontrast allergy. CTV is equal to digital subtraction angiography (DSA) (Fig. 14-4) while more capable for assessing central veins proximal to long segmental obstruction at the level of the first rib or to detect a position-dependent blockage (37).

Catheter-based central venography is typically done as an initial procedure to an endovascular intervention. DSA has more sensitivity than color duplex venous ultrasound in the assessment of dialysis access with exceptional ability to discriminate central venous anatomy from collateral veins. The 2019 Kidney Diease Outcomes Quality Initiative (DOQI) guidelines also recommend venography before implantation of a permanent AV access in previous subclavian catheterization patients (39).

TREATMENT

Treatment options applied for CVD rely upon the etiology of the disease. Current treatment options include medical therapy, endovascular intervention and open surgery (i.e., vein bypass). Raising the upper limb and adjunctive anticoagulant treatment may often mitigate edema correlated with CVD, particularly when it is involved with acute thrombus. However, those measures, are not applicable in chronic blockage. The use of anticoagulants alone has no function in the recanalizing process as the problem remains with the progression of scar tissue. Open surgical techniques are limited to a few highly morbid operations, including open endovenectomy, and venous bypass of the blocked central veins via sternotomy.

Endovascular intervention is the gold standard for treating CVD hemodialysis patients. The treatment

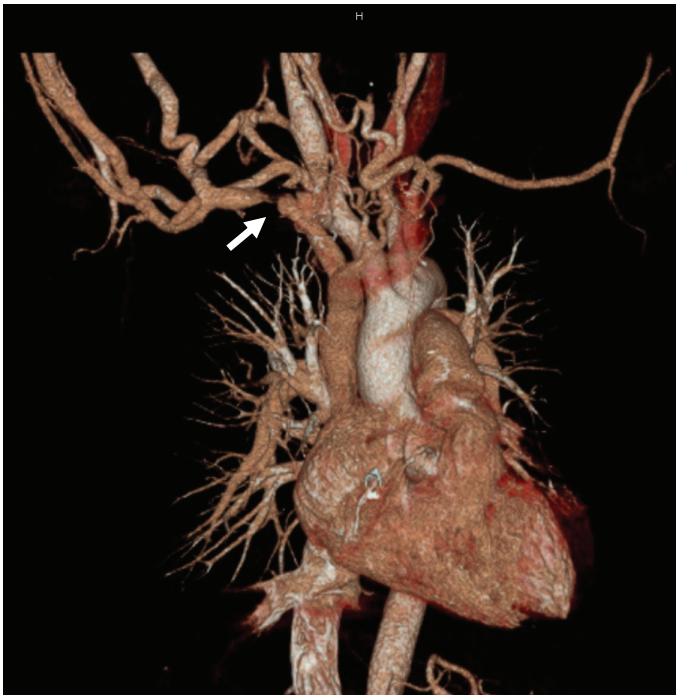


Figure 14-4. Computed tomography venography (CTV) of patients in figure 14-3 showing right subclavian vein occlusion (white arrow).

choices include PTA, implantation of bare metal stents (BMSs), and lately placement of covered stents (CSs). The KDOQI guidelines recommended PTA, without or with stent implantation as the favored CVD management approach (39). However, for patients intractable to endovascular treatment, surgical correction must be considered. Similarly, in patients without a history of central venous catheterization, when treatment of the underlying disease is not possible or cannot resolve arm swelling, then PTA without or with additional stent implantation is indicated.

When interventional management of CVD fails or is impossible, surgical evaluation is necessitated to establish the most effective surgical method, along with the procedural risk, and life expectancy. Initially, all venograms have to be re-assessed. If there is a functional arteriovenous AV access within the ipsilateral extremity to the site of CVD, a 'simple reduction' process can reduce the volume down to some levels that may be satisfied by collateral circulation while continuing to supply sufficient blood-flow for dialysis with alleviation of symptoms. If not, then manage the CVD with extra-anatomic bypass, incorporating axillary to femoral vein bypass, subclavian vein to external or internal jugular vein bypass, or jugular vein turn down process. If an ipsilateral surgical bypass is impossible, further venography of the contralateral arm should be performed to evaluate if a new access can be formed in another arm. After this, an ultimate treatment access ligation may be considered, to alleviate local symptoms.

ENDOVASCULAR INTERVENTION

Endovascular intervention is frequently used to relieve symptoms and to improve the function of the hemodialysis access. The 2019 update KDOQI clinical practice guideline for vascular access recommends that PTA, without or with stent implantation is considered the preferential CVD approach (39).

Indications for endovascular intervention

1. Symptomatic CVD with edema of upper extremity, face or chest wall
2. Inadequate dialysis secondary to CVD
3. CVD with thrombosis of AV access
4. Cardiac device-related subclavian stenosis (40,41)

The contraindication of endovascular intervention of CVD is the existence of large thrombus at the lesion except when the thrombus is first be removed by thrombectomy or other means (14,42). Other contraindications are surgical revision<30 days before referral, infected access, pulmonary hypertension, or the existence of a right-to-left intracardiac shunt (43). Since progression of collaterals can be correlated with symptom resolution, endovascular treatment can be postponed in asymptomatic patients capable of accomplishing adequate dialysis. Currently, the natural history of stent implantation and angioplasty in those elastic lesions are jeopardized by rapid and frequent recurrence. Potentially an asymptomatic lesion may become symptomatic following the intervention. Moreover, stenosis has been demonstrated faster progress following intervention (44,45). Therefore, more elasticity with potentially for severe recurrent neointimal hyperplasia should deter intervention in mildly symptomatic or asymptomatic CVD. Those patients need careful follow-up as deteriorating symptoms will necessitate intervention.

PATIENT SELECTION

When considering management of CVD, the clinical presentation, location and type of AV access, previous AV accesses, future potential AV accesses, potential for kidney transplantation, and life expectancy should be considered. CTV for the chest can help in assessing the feasibility of intervention and delineate the CVD. When the access has many other lesions, in older age with many other potential access sites, perhaps treatment is not needed, because a new AV access may be made and ligate the old access. Conversely, when a patient has one remaining access weak flows without possibility of transplant, more aggressive treatment can be considered. Central venous occlusions seldom result in access thrombosis since most patients grow collateral circulation.

When a patient's symptoms of facial/arm swelling are considerably acute, postponing for several weeks might permit growth of enough venous collaterals which diminish symptoms with minimal dysfunction of access. When there is minimal elevated venous pressures, it is preferable to avoid intervention. Importantly, therapeutic intervention has been reported to be relatively worthless for asymptomatic central venous lesions (46), so one should avoid interventions for asymptomatic central venous lesions.

STEP-BY-STEP TECHNIQUE

PERIPROCEDURAL MEDICATION

The purpose of antithrombotic therapy for CVD angioplasty with or without stenting is to prevent stent thrombosis and/or in-stent restenosis. Regardless of rising rates of venous angioplasty and stent implantation, few studies have been conducted to predict the best antithrombotic therapy regimen (47). Most current practices, based on previous experience in managing CVD, rely on data reported from arterial stent implantation. For patients undergoing planned SVC, brachiocephalic or subclavian venous intervention, we usually give 81 mg to 325 mg of aspirin and clopidogrel 75 mg daily prior to the procedure for 5-7 days. In case of untreated patients, a loading dose of clopidogrel 300-600 mg (4-8 tablets) and 81 mg of aspirin are usually given before the procedure. Unfractionated heparin 50-70 units/kg with target activated clotting time (ACT) 250-300 sec is administered. The use of thrombolytic agents or glycoprotein IIb/IIa agents for central vein obstruction is unnecessary and not recommended as an initial treatment. Less data is available for the application of thrombolytic agents during central venous thrombosis (48). Therefore, it is not recommended as an initial treatment regimen. Catheter-based thrombolysis is effective for the treatment of central vein blockage secondary to acute thrombosis, particularly in combination with a staged surgical correction for the anatomical etiology for venous constriction for the case of Paget-Schroetter syndrome. (49,50).

VASCULAR ACCESS

Non-invasive pre-procedural imaging such as CTV or MRV, if available, is helpful to make a plan and choose an appropriate vascular access. A venous access can be obtained using surface or anatomic landmarks or imaging guidance, usually doppler ultrasound. In short lesions of the central veins, preferred access is usually from the arm using the AV shunt vein. Percutaneous needle entry to the AV fistulas or grafts is straightforward, because these shunt veins usually are engorged and felt of palpable thrill. In some cases, when large balloons or stent devices are used, transfemoral venous access can be used to avoid injury of the AV shunt veins. In case of subclavian or brachiocephalic vein occlusion, two-way approach via common femoral and AV shunt vein is occasionally necessary to facilitate wire crossing and to achieve continuous monitoring of the stent implants in central veins. In tortuosity vessel and occlusion, stents also can be placed over a guide wire forming a loop through ('body-flossing' technique or through-and-through technique) the AV shunt vein and femoral vein in order to provide support for the transvenous segment and possibly prevent stent embolization into the pulmonary circulation.

If patients don't have an AV shunt vein, upper extremity venography can be performed by using hand contrast injection into the venous system via an 18- to 20-gauge needle or angiocath in the superficial veins on the dorsum of the hand. With this technique, cephalic or basilic vein can be located and punctured using a 4F-micropuncture set or a radial set (as used for percutaneous coronary intervention). Using a doppler ultrasound guidance is also helpful for getting vascular access of a non-palpable superficial vein.

DIAGNOSTIC CENTRAL VENOGRAPHY

A diagnostic venogram can be performed through a venous access from the common femoral vein or the arm or using a 6-Fr Judkins right (JR) or multipurpose (MP) catheter which is cannulated into the brachiocephalic or subclavian vein. Digital subtraction central venography is well visualized in the anteroposterior (AP) projection.

For a short stenotic lesion, imaging evaluation is mainly performed by venography via the dialysis access and advancing a catheter up to the lesion to obtain imaging. Diagnostic DSA from the outflow vein of the AV access to the SVC is achieved (Fig. 14-5). This diagnostic imaging is used to define anatomy, pathology of CVD and collateral pathways. After venography, simple endovascular intervention can be performed through this arm access.

In case of central venous occlusion, following primary identification of the distal end of the lesion from the arm access, a second common femoral vein access usually is required and inserted with a 6-Fr JR4 or MP catheter advanced up to the brachiocephalic occlusion lesion. Further venography by synchronous contrast injection from both ends of the lesion is performed once more to delineate the position and length of the occluded segment and to characterize collaterals and the point at which the central vein segment becomes patent again (Fig. 14-6).

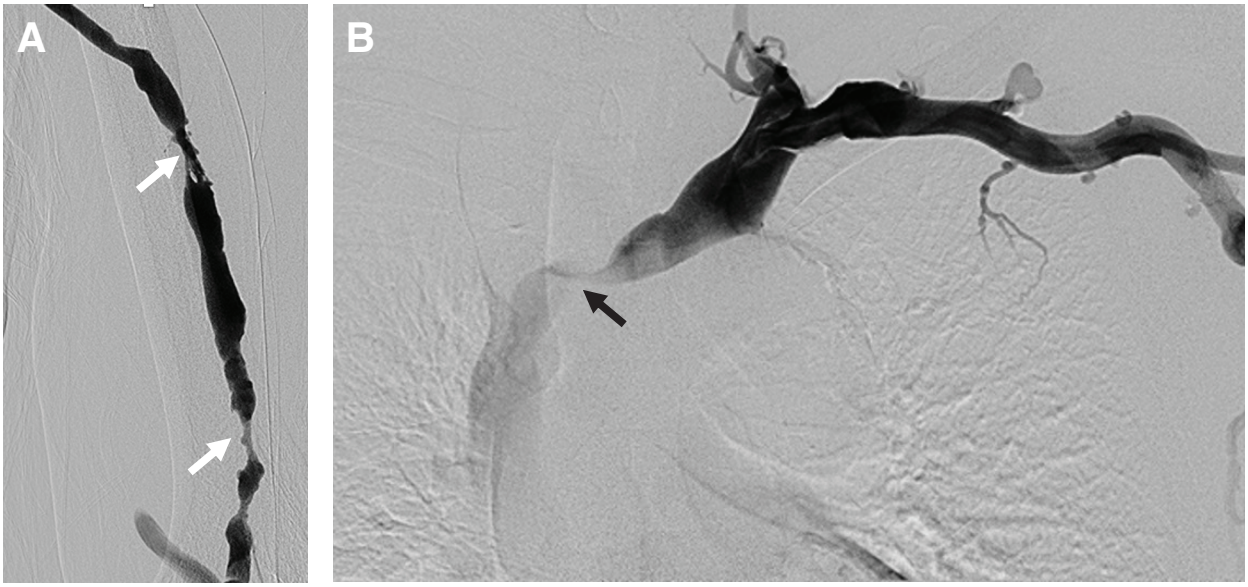


Figure 14-5. Diagnostic venogram from AVF access. A: Digital subtraction venography injected from a 19-gauge Jelco® catheter in the left brachiocephalic vein graft revealed two sequentially severe stenosis of the vein graft (white arrows). B: Central venography revealed patent left axillary and subclavian veins with severe stenosis of the proximal left brachiocephalic vein (black arrow).

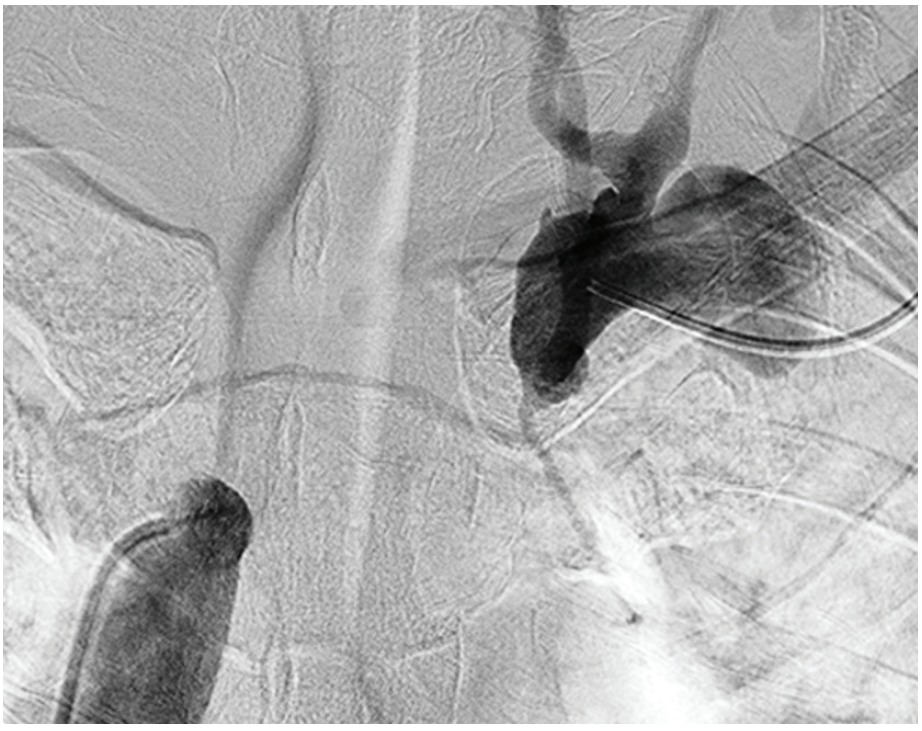


Figure 14-6. Diagnostic digital subtraction venography of left brachiocephalic vein occlusion by injecting contrast at the proximal and distal end of lesion. Round tip of occlusion with side branch is unfavorable anatomy for traversing the lesion from forearm access.

Quantitative analysis of the venogram should be performed with particular attention to the length of the diseased segment, to measure diameter of the normal reference vein before and after the abnormal segment and to measure severity of stenosis. A measured calibration of diagnostic catheter or a radiopaque ruler placed in the field of view at the time of imaging can be used to perform quantitative analysis of the images where intervention is planned.

Currently, diagnostic venography of the central veins has regained its importance because of the increasing number of dialysis-dependent patients. The indications of arm and central venography are shown in Table 14-2.

Table 14-2. Indications for arm/central venography

To evaluate:

- Anatomy for preoperative planning of dialysis access surgery (arteriovenous fistula or graft creation)
- Anatomy prior to central venous catheter (e.g., peripherally inserted central catheter [PICC], long-term central venous catheters, ports) or pacemaker placement
- Upper extremity superficial and/or deep vein thrombosis
- CVD secondary to long-term indwelling catheters, mediastinal fibrosis, thoracic malignancy, or radiotherapy
- Hemodialysis shunt dysfunction
- Central venous catheter related stenosis/occlusion

SHEATH PLACEMENT

A 20-gauge Jelco® catheter over needle is usually used to enter the venous outflow of the AV fistula or graft or superficial vein (e.g., cephalic or basilic) from the arm. Importantly, sterile technique is used and local anesthesia infiltrated in area of access prior to access needle introduction. Once the needle tip is confirmed to be within the lumen with pulsatile backflow of blood, a 0.018" guide wire is introduced and passed through the lumen of vein graft. Sometimes, passing a guide wire via needle into vein is difficult which is caused by guide wire in side branch, guide wire against stenosis/occlusion or valve. It is recommended to pull back the wire and redirect under fluoroscopy or advance the Jelco® catheter over wire and perform AV fistulogram and central venogram to define anatomy or pathology. DSA using hand injection with 50:50 contrast and saline is then performed. If a patient has central venous obstruction and requires angioplasty or stenting, the 20-gauge Jelco® catheter is switched for a 6-Fr radial sheath placed into the graft access. The sheath (together with the dilator inserted) is then introduced over the guide wire to the vein graft. A small superficial skin incision can be made where the guide wire enters through the skin to enable smooth crossing of the sheath. Once the sheath is completely advanced, the guide wire and the dilator assembly can be removed. The radial sheath can accept 0.035" guide wires over which it can be exchanged out for a larger diameter sheath (7-Fr) and longer sheath (25 cm) depending on the procedure.

In case of failure from arm approach, a common femoral vein is normally used. A 6-Fr 70 cm Flexor Raabe®sheath (Cook), DuraSheath (BMV Medical), or Destination® guiding sheath or a 7-Fr 70-80 cm Brite-tip sheath (Cordis) is inserted in the common femoral venous access site and positioned near the ostium of the brachiocephalic vein. For stent diameter up to 14 mm, 7-Fr diameter sheaths can be employed to delivered the stents.

CROSSING THE LESION

A 5-Fr or 6-Fr JR4 or MP diagnostic catheter is placed inside a sheath and advanced up to the stenotic lesion or occlusion site of the brachiocephalic or subclavian vein. In general, it is preferable to treat the lesion from the hemodialysis access, since it is simpler and a shorter distance when compared with working from the femoral approach, which may necessitate a long introducer sheath to provide stable access. A 0.035" J-tip or angle-tip hydrophilic Glidewire® (Terumo) is usually the wire of choice to cross via the stenotic or occluded vein segment (Fig. 14-7). The "J" shape tip protects from "digging" into the wall of the vein and potentially perforating out of the side of the vein. Typical techniques include using coaxial support catheters and sheaths, back and front ends of guide wires, and sharp needle techniques.

If the 5-Fr or 6-Fr catheter is not advanced, it is switched for a 4-Fr hydrophilic catheter (Glidecath®, Terumo) to cross or needed pre-dilatation with a 0.014" or 0.018" small profile coronary or peripheral balloon. When the stenotic lesion is traversed with the hydrophilic guide wire, the catheter is then forwarded over the lesion and switched for a 0.035" Amplatz super stiff™ wire (Boston Scientific) that offers increased support to passage of a balloon catheter across the lesion.

Wiring techniques for central venous occlusion

For occlusions that are unable to be easily crossed, two-way approach should be considered from AV shunt access and common femoral vein. With an occlusion, one should closely examine the venogram on both ends for any "nipple" or focal beak-like area (venous stump) that can be a starting point to begin probing with the Glidewire® (Fig. 14-8). Antegrade approach from the arm access is sometimes useful to cross to another end