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Techniques in Vascular and Interventional Radiology

Central Venous Interventions

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Central venous stenosis or occlusion is a common and vexing problem in patients undergoing hemodialysis. Typical presenting symptoms include arm swelling and prolonged bleeding after hemodialysis. Despite multiple treatment approaches, these stenoses tend to recur and progress over time. A thorough preprocedure evaluation, methodical procedural approach and awareness of potential complications are all essential to try to preserve vascular access and improve patients' quality of life.
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Introduction

There are approximately 660,000 patients in the United States with ESRD, 63.7% of whom patients undergo renal replacement therapy through hemodialysis.[1] Central venous stenosis (CVS) and occlusion (CVO) are common and frustrating problems in the management of these patients. Central veins are defined as the subclavian veins, brachiocephalic veins and the superior vena cava (SVC). For lower extremities, the iliac veins and inferior vena cava (IVC) are considered central veins. CVS and CVO typically manifest with arm swelling and prolonged bleeding after dialysis (Fig. 1). Asymptomatic CVS may be discovered when elevated static venous pressures or elevated pump pressures are detected with surveillance and monitoring. When severe and uncorrectable, access loss can occur either because of thrombosis or because intentional ligation was necessary for persistent severe symptoms.

Etiology and Mechanism

The incidence of CVS/CVO in dialysis patients is unknown; only symptomatic patients or those with dysfunctional accesses undergo venography. The underlying pathophysiology of central stenosis in patients with ESRD is likely multifactorial. The 2 most common risk factors are prior or existing central venous catheters (CVC) or existing cardiac implantable electronic device (Figs. 2-4). The highest incidence of CVS related to prior catheter placement is from subclavian insertion, which should be avoided whenever possible.[2] Compared with right internal jugular CVCs, there is a higher incidence of CVS/CVO after left internal jugular CVCs. This is attributable to the multiple turns made by a left sided catheter causing multiple points of contact between the catheter and vessel wall.[3-5] Peripherally inserted central catheter placement is imprudent in any patient with chronic kidney disease stage 3 or greater (GFR <45).[6-9] Minimizing the use of central venous catheters, by instituting early placement of arteriovenous grafts and fistulas, is a key component in combatting central stenosis.

CVS and CVO can occur without previous CVC or cardiac implantable electronic device, likely caused by intimal injury from high pressure, high-volume flow induced by upper arm grafts and fistulas.[10] Rare causes of CVS and CVO include thoracic outlet syndrome, fibrosing mediastinitis, and postradiation therapy.[11]

Clinical Manifestations of CVS or CVO

Stenosis or occlusion of the subclavian and brachiocephalic veins may cause progressive ipsilateral arm swelling. Bilateral brachiocephalic vein or SVC stenosis or occlusion may present with SVC syndrome. Other common manifestations include prolonged bleeding, low flows and elevated venous pressure. On physical examination, one may find prominent collateral vessels around the shoulder and upper chest (Fig. 5). Note that

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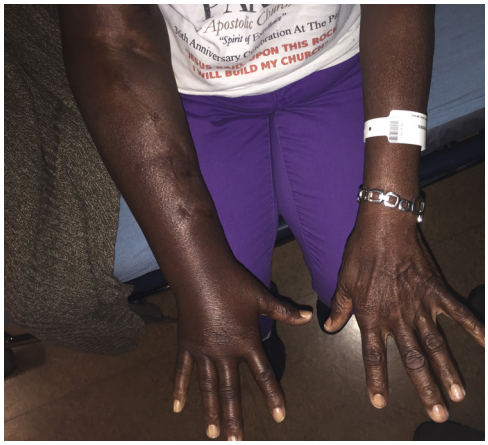

Figure 1 A 63-year-old female with ESRD, who had bilateral dialysis catheters placed while waiting for right upper extremity fistula maturation. Seven months after it matured she developed symptomatic right arm swelling. (Color version of figure is available online.)

severe stenoses or occlusions may be asymptomatic because of these often robust collaterals.[12,13] Intriguingly, a recent study revealed higher incidence of symptomatic central stenosis in patients with arteriovenous grafts (AVGs) when compared with arteriovenous fistulas (AVFs),[13] possibly related to greater capacitance of AVFs than AVGs.

Indications for Intervention

Whether or not to intervene on a CVS/CVO should be determined by both clinical symptoms and venographic findings; intervening on angiographically significant (>50%) but asymptomatic stenoses should be avoided as this practice has been shown to induce more rapid progression of the lesion.[12,14] Nonprogressive mild symptoms such as mild arm edema or isolated elevated venous pressures may be managed conservatively so that collaterals could develop over time obviating the need to treat. Moderate to severe arm or face swelling, low access flows, cerebral venous hypertension, prolonged bleeding time, progressive significant fistula dilation and access thrombosis are all indications for intervention (Table 1).[15,16] Retrograde flow up the ipsilateral internal jugular vein with drainage through the contralateral internal jugular vein places the patient at risk for subsequent symptomatic cerebral venous hypertension and should also be considered an indication for treatment (Fig. 4). Translesional pressure gradient exceeding 5-10 mm Hg within central venous system indicates a presence of hemodynamically significant lesion[17-19]. This value, just like the angiographic appearance, must not be the sole trigger for intervention.

Endovascular Intervention

Central Vein Stenosis

With CVS, the general method of treatment is similar to balloon angioplasty at other locations. Using a 23-45 cm long sheath allows for greater wire, catheter, and balloon stabilization. A long sheath also allows one to perform high-quality venograms as the tip can be placed just peripheral to the site of stenosis. If CVS is suspected, one should start initially with a 7 Fr sheath as high pressure balloons >10 mm in diameter do not go through a 6 Fr sheath. The guidewire should be placed into the IVC for all central venous procedures. Steps for performing a standard balloon dilation of a CVS are as follows:

(1) Access downstream from the arterial anastomosis in an antegrade direction. Perform initial venogram confirming central venous stenosis.
(2) Place 7 Fr 23-45-cm long sheath.
(3) Cross the stenosis with the combination of directional catheter and angled glidewire and advance them into IVC. Exchange glidewire for stiff, nonhydrophilic guidewire such as Rosen wire (Cook Medical, Bloomington, IN), or Amplatz Wire (Cook Medical or Boston Scientific Corp., Marlborough, MA). Advance the sheath to a few centimeters peripheral to the site of stenosis. Repeat a venogram through the sheath, ensuring that one has not unwittingly crossed centrally through a collateral vein.
(4) Administer heparin (40-50 U/kg or 3000 U bolus).
(5) Choose an appropriately sized balloon based on adjacent normal vessel diameter. As a rough guide: subclavian vein: 10-14 mm, brachiocephalic vein: 12-16 mm, and SVC: 14-20 mm.
(6) Perform angioplasty. Ensure waist effacement occurs. Keep balloon inflated for 1-2 minutes.
(7) Remove balloon over the wire. Repeat venogram through sheath, keeping wire in place. Treat findings as follows. *Residual stenosis*: repeat angioplasty with

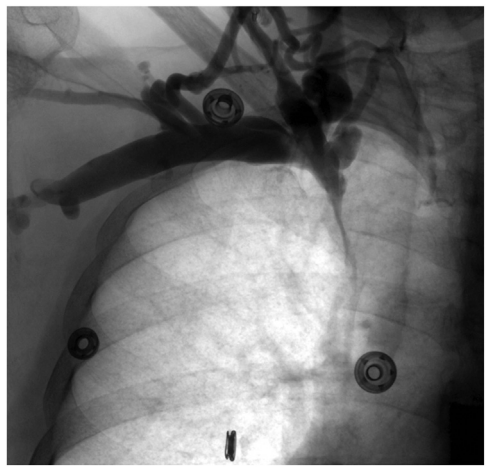

Figure 2 (Same patient as Fig. 1) Venogram shows severe stenosis of the right brachiocephalic vein and upper SVC.