Endovascular Management of Chronic Upper Extremity Deep Vein Thrombosis and Superior Vena Cava Syndrome

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ABSTRACT

Chronic upper extremity deep vein thrombosis (UEDVT) and superior vena cava syndrome (SVCS) are important and underdiagnosed entities that are associated with significant morbidity, and both are becoming increasingly common due to the use of indwelling catheters and implantable central venous access devices. Currently, as many as 25% of patients with an indwelling catheter are diagnosed with chronic UEDVT or venous stenosis. SVCS is most commonly seen in the setting of malignancy, especially lung cancer and lymphoma. Endovascular management of chronic UEDVT and SVCS is accepted as an important first-line treatment given its high overall success rate and low morbidity as compared with medical and surgical treatments. In this article, the indications for treatment, complications, and success rates of the endovascular management of UEDVT and SVCS are reviewed. Relevant findings on presentation and physical exam as well as various imaging modalities and imaging findings are evaluated. Details of preprocedure evaluation, relevant anatomy, and avoidance of potential complications are discussed.

KEYWORDS: Upper extremity deep vein thrombosis, superior vena cava syndrome, venous stenosis, catheter-related deep vein thrombosis

Objectives: Upon completion of this article, the reader should be able to explain the presentation, diagnostic evaluation, and endovascular treatment of chronic venous obstruction of the upper extremities and superior vena cava.

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Upper extremity deep vein thrombosis (UEDVT) and superior vena cava syndrome (SVCS) are important and frequently underdiagnosed entities. Both are associated with significant morbidity and may have devastating consequences such as loss of vascular access and debilitating upper extremity pain and swelling.¹

In the past decade, UEDVT, defined as thrombosis of the brachiocephalic, axillary, subclavian, brachial, ulnar, radial, or interosseous veins, has been found to be considerably more prevalent than previously believed; this is largely due to the increasing use of indwelling catheters and implantable central venous access devices for dialysis, chemotherapy, bone marrow

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transplantation, and parenteral nutrition.^{2–4} Catheter-associated UEDVT and central stenosis has been reported in as many as one-quarter of patients with indwelling central venous catheters.⁵ The remainder of cases of chronic upper extremity venous obstruction are most commonly related to dialysis access, hypercoagulable states, and mechanical compression due to underlying anatomic abnormality.⁶ SVC syndrome, defined as stenosis or occlusion of the SVC leading to obstruction of the venous outflow of the head and upper extremities, is seen most commonly in the setting of malignancy, especially lung cancer and lymphoma. Obstruction of the SVC may be secondary to external compression or direct invasion. Two to 4% of patients with lung cancer develop SVCS at some time during their disease course.⁷ Overall, ~60–85% of SVCS is found in the setting of malignancy, while benign causes are found in the remaining 15–40%.⁸ According to a 2008 study from the Mayo Clinic, mediastinal fibrosis accounts for 50% of cases of benign SVCS and the remainder are associated with indwelling central venous catheters and pacemaker wires.⁹

PATHOGENESIS

UEDVT may be categorized based on etiology. Primary thromboses are rare and found in the absence of recognized risk factors. These account for 1–9% of cases UEDVT and are found in 2 persons per 100,000 in the general population per year.^{10,11} Primary thrombosis includes those that develop from extrinsic compression of the axillary or subclavian veins by tendons, muscles, or bony thorax in the subcoracoid or costoclavicular spaces (Paget-Schroetter syndrome). Primary thrombosis predominantly affects younger patients involved in athletics or activities requiring repetitive motion, and develops as a result of damage of the wall of the vein with subsequent thrombosis. Secondary thromboses are those found in the presence of known risk factors, and are considerably more common than primary thromboses. Indwelling catheters and implantable central venous access devices account for up to 63% of secondary thromboses.¹⁰ Catheter-related thrombosis is caused by several factors. The vessel wall may be damaged during placement or infusion of medication. The catheter may also impede blood flow resulting in areas of stasis within the vein. Patients with incorrectly placed catheters are considerably more likely to develop catheter related thrombosis.¹² In addition to indwelling catheters and implantable central venous access devices, other UEDVT risk factors include hypercoagulable states, trauma, and malignancy (Table 1). As opposed to patients with primary thrombosis, those affected with secondary thrombosis tend to be elderly and more often systemically ill.¹³

As previously described, obstruction of the SVC may be secondary to invasion or compression by adjacent

Table 1 Risk Factors for Upper Extremity and Central Venous Thrombosis

Central venous catheter
Pacemaker
Hypercoagulable syndromes
Malignancy
Extrinsic compression
Tumor invasion (Pancoast)
Injection of sclerosing medications
Trauma
Radiation

pathologic processes involving the mediastinum and right lung or by thrombosis of blood within the SVC. Rapidly progressive SVC syndrome is considerably more likely to be due to malignant etiologies. The rate at which symptoms become evident is dependent on the rapidity of SVC occlusion. SVCS related to benign etiologies, especially mediastinal fibrosis, typically requires many months or years to become symptomatic as the slow disease progression allows formation of robust collateral pathways for return of venous blood to the right atrium from the azygous (Fig. 1), internal mammary, paraspinal and lateral thoracic systems (Fig. 2).¹⁴

CLINICAL PRESENTATION

Although axillary or subclavian vein thrombosis may be asymptomatic, the most common presentation is vague shoulder or neck discomfort and upper extremity edema.²

Figure 1 Digitally subtracted venogram showing chronic and superior vena cava syndrome related to an indwelling central venous catheter. Decompression of the head, neck, and upper extremities is achieved through a markedly dilated azygous vein.

Figure 2 Nonsubtracted venography performed with simultaneous contrast injections into the bilateral antecubital veins showing chronic obstruction of the superior vena cava related to a left internal jugular hemodialysis catheter. Note the presence of well-formed collateral pathways via the lateral thoracic veins as well as retrograde flow within a dilated azygous vein.

Chronic occlusion of the peripheral upper extremity veins may present with a hard, palpable, cord-like vein. If thrombosis leads to obstruction of the superior vena cava, patients may present with arm and facial swelling, stridor, blurred vision, dyspnea, or vertigo.¹⁵ Patients with SVC obstruction may also report positional headache, retro-orbital pain, confusion, syncope, dysphagia, or chest pain, and in severe cases, cervical venous pressure may increase by 20–40 mm Hg. In some cases, orthopnea may be noted because the amount of blood flow to the upper torso is elevated when the patient is placed in the supine position.¹⁶ The presence of stridor is especially ominous as it may be associated with laryngeal edema and impending airway compromise. Other concerning findings include confusion and obtundation, which are indicative of cerebral edema.⁷

EVALUATION

The diagnosis of chronic UEDVT and SVCS may often be made based on history and physical examination. Focused physical examination can document the extent of arm, facial, or neck swelling, chest wall collateralization, elevation of neck veins, and respiratory compromise. Facial swelling and distention of chest wall collaterals are typically exacerbated when the patient is in the supine position.⁷ The presence of well-developed collateral circulation, both on physical exam and diagnostic imaging, is key to the diagnosis of chronic as opposed to acute disease.

Table 2 Imaging Features of Chronic Venous Thrombosis

Occluded, string-like vessel
Linear intraluminal webs
Mural irregularity and thickening
Stenoses
Hyperechoic intraluminal filling defects (ultrasound)
Dilated, well-developed collateral drainage

Imaging is the mainstay for definitive diagnosis and characterization of chronic UEDVT and SVCS (Table 2). Duplex ultrasound is the initial imaging test of choice as the technique is cost effective, noninvasive, does not involve ionizing radiation, and has high sensitivity for peripheral (distal subclavian, axillary) UEDVT.¹ Ultrasound does have significant limitations as acoustic shadowing from the clavicle will limit visualization of the central subclavian and brachiocephalic veins. Contrast venography provides excellent characterization of venous anatomy and is not subject to the limitations of duplex ultrasound. Venography may be necessary to confirm a diagnosis of UEDVT or SVCS if clinical suspicion remains high despite negative duplex ultrasound evaluation.¹² Venography is mandatory prior to interventions such as venoplasty or stent placement and is also necessary to assess the response to these treatments. There are several drawbacks to venography. The use of iodinated contrast may be associated with nephrotoxicity or allergic reaction. Even though commercially available contrast agents are rated pregnancy class B, many centers refrain from use in pregnant patients.¹⁷ Venography is often technically challenging as cannulation of peripheral veins of the upper extremities may be difficult in the setting of venous obstruction with soft tissue edema. If chronic UEDVT or SVCS is secondary to an indwelling catheter, venous cannulation for venography may be especially challenging as venous patency is often the reason for central venous catheter placement. Magnetic resonance angiography (MRA) and computed tomography angiography (CTA) provide accurate, noninvasive methods for detecting central venous stenosis or obstruction. Both correlate extremely well with venography and can provide more detailed data regarding the level and extent of obstruction, as well as the formation of collateral pathways. The presence of collateral vessels on CT or MRI is a strong indicator of SVC syndrome with a sensitivity and specificity of 96% and 92%, respectively.¹⁸ If the etiology of venous obstruction is unknown, CTA is the best choice of imaging as it may provide detailed evaluation of adjacent soft tissue structures (Fig. 3). As with venography, there are several drawbacks to CTA and MRA. The use of intravenous (IV) contrast may be contraindicated in some patients, and though noncontrast MRA examinations may be performed using two-dimensional (2D) time of flight techniques, the anatomic detail is inferior to

Figure 3 Contrast-enhanced axial computed tomography image of a patient presenting with acute superior vena cava (SVC) syndrome. The image shows a large mediastinal mass encasing and narrowing the SVC. Tissue obtained from the mass yielded a diagnosis of lymphoma.

gadolinium-enhanced techniques. CTA exposes the patient to considerable ionizing radiation, and should be used sparingly, especially in young patients.

TREATMENT

The treatment of chronic UEDVT and SVCS depends on the underlying cause, chronicity, and clinical presentation. The mainstay of treatment of acute thrombosis is anticoagulation with the addition of endovascular adjuncts such as mechanical thrombectomy or catheter-directed thrombolysis in some cases.¹⁹ Unlike acute UEDVT and SVCS, anticoagulation and catheter-directed or mechanical thrombolysis have little role to play as the organization of the thrombus severely limits their effectiveness.

The traditional method of treatment of malignant central venous obstruction is radiotherapy and chemotherapy, which is effective in 90% of cases. Surgery is rarely indicated. Clinical response to chemotherapy and radiotherapy is seen after ~1 week; there is a 20% recurrence rate, even after the use of the total permissible dose of radiation.²⁰ The high recurrence rate is due to progression of disease, postradiation fibrosis, or complicating thrombosis.²¹ Because of the relatively long latent period between medical treatment and clinical response and the high rate of recurrence, endovascular management with venous stenting should be initiated early and may be considered a first-line intervention. Balloon venoplasty with stent placement provides nearly instantaneous symptomatic relief with a low risk of side effects and high long-term patency. Stenting of SVCS can be especially useful when urgent intervention is indicated for patients without a tissue diagnosis or who have already undergone radiation therapy or chemotherapy with poor response.^{22–24} A recent study of stenting malignant SVCS showed primary and secondary patency rates of 88% and 95%, respectively, over a mean survival period of 5.4 months.²⁵

Stenting of benign obstruction secondary to chronic UEDVT and benign stenosis of the central veins is undertaken less frequently than stenting in the setting of malignancy, although the number of cases is increasing. Obstruction in the setting of catheter-related stenosis or chronic thrombosis may be improved or relieved by removal of the catheter, but this may not be an option in patients with tenuous venous access or catheter dependence. Balloon angioplasty is the mainstay of treatment, and stent placement is reserved for patients in whom angioplasty has failed due to elastic recoil or restenosis of the vessel. Benign obstructions are frequently difficult to treat successfully with venoplasty alone and may require multiple reinterventions and eventual stent placement to maintain venous patency.²⁶ Bornak et al published a series of nine patients undergoing endovascular treatment of benign lesion and reported a 67% one-year patency rate with 2 patients requiring repeat interventions.²⁷ A larger series of 19 patients undergoing endovascular treatment of benign lesions was published in 2005, which reported 93% patency at a median follow-up time of 29 months with 3 patients requiring reintervention.²⁸

TECHNIQUE

Due to patient apprehension and the often prolonged nature of complex endovascular procedures, conscious sedation in addition to local anesthesia at puncture sites is recommended in all but the most straightforward cases or in cases in which there are contraindications to sedative medications. Venoplasty can elicit significant pain when the adventitia of the veins is stretched to full dilation. Expansion of metallic stents may also elicit pain, especially when placed in the SVC or brachiocephalic veins. In most cases of chronic UEDVT and SVCS undergoing endovascular treatment, procedural anticoagulation is not necessary. One exception is seen in cases of complicated or prolonged SVC venoplasty and stent placement, in which patients should receive 3000–5000 units of heparin IV to prevent acute SVC thrombosis. If the SVC obstruction is relieved following the procedure, no additional heparinization is necessary.

The site of venous occlusion or stenosis will dictate the appropriate site for vascular access. Stenoses located in the right brachiocephalic vein or SVC are best approached from right internal jugular or right common femoral vein access. Stenoses of the left brachiocephalic vein are best approached from left axillary or femoral vein access. Subclavian lesions may be approached from the ipsilateral brachial or axillary vein. Care should be taken when performing axillary vein puncture as trauma to the brachial plexus may be incurred, especially when large diameter catheters or introducer sheaths are employed. Brachial plexus injury can be avoided by placing the puncture site more centrally near the junction of the brachial and axillary veins.²⁶ A hemostatic valve sheath

should be utilized in all cases. If SVC lesions are being treated, sheaths up to 10F may be required to facilitate the passage of large diameter balloons and stents. In most cases, 0.035-inch guidewires with 5F Cobra or multi-purpose catheters are sufficient to cross venous obstructions. When stent placement is performed, maximum

stability of the delivery device may be insured by the use of semirigid Amplatz[®]-type guidewires (ev3 Endovascular Inc., Plymouth, MN). Tight occlusions of the left brachiocephalic vein secondary to chronic thrombosis (Fig. 4), fibrosis, or mediastinal tumor may require combined bilateral axillary or combined axillary and

Figure 4 Subtracted and nonsubtracted images showing chronic obstruction of the left brachiocephalic vein in a patient presenting with left upper extremity pain and edema. Access was gained via a left internal jugular approach (A) and the lesion was predilated prior to stent placement (B). A bare metal self-expanding stent was placed across the lesion (C), and a final digital subtraction venogram showed resolution of the stenosis (D).

Figure 5 Subtracted (A) and nonsubtracted (B) images showing chronic stenosis were of the superior vena cava (SVC) at the confluence of the right and left brachiocephalic veins. The stenosis was crossed and guidewires were placed from both left and right internal jugular access. Bare metal “kissing” stents were then placed across the stenotic segment of SVC.

femoral access points. When the guidewire has traversed the occluded segment, it is grasped with a snare or basket on the opposite side and retrieved through the hemostatic valve sheath. This allows insertion of a balloon or stent over the guidewire, which is then controlled at both ends.²⁶ Prior to stent or balloon deployment, venography of the involved area is performed for characterization and acquisition of vessel diameters. If feasible, the stent length should extend ~2 cm proximal and distal to the stenosis.

Peripheral stenoses involving the axillary, subclavian, and brachial veins should rarely, if ever, undergo stent placement due to high rates of stent failure and occlusion. If stenting is absolutely necessary, short, flexible stents with minimal metal surface should be utilized. Self-expandable metallic stents should be used as balloon expandable stents have smaller diameters, less flexibility, and higher risk of migration with respiratory motion.²⁹ In rare cases of venous tumor invasion, covered stents may be indicated to prevent tumor ingrowth. When sequential stents are employed, especially in the central veins, stents should be placed first in the distal position and then more proximally in relation to the venous access site. In cases of tight SVC or brachiocephalic vein stenoses, predilation of the lesion may be required. Overdilation of the SVC should be avoided as it may be associated with dysrhythmias or even cardiac arrest.²⁶ If the proximal SVC or confluence of the brachiocephalic veins is treated, simultaneous “kissing” stents can be placed in both brachiocephalic veins with parallel courses in the SVC (Fig. 5). In general, covering of large tributaries to the central veins should be avoided to allow for future catheterization. The inflow of the azygous vein, however, is routinely covered when stent-

ing the SVC and is generally without clinically evident side effects.

COMPLICATIONS

Balloon venoplasty and venous stenting are safe procedures with relatively few complications. The most ominous complication is stent migration as repositioning of a fully deployed venous stent is nearly impossible. Stent migration may be avoided by ensuring the stent diameter is large enough and the entire stenosis is covered by the stent.³⁰ If stents embolize to the heart or pulmonary artery, removal should be performed as soon as is feasible to prevent damage to cardiac valves and pulmonary vascular endothelium. Venous perforation is rare and can be prevented by avoiding overdilation, especially in the SVC. Pulmonary edema secondary to rapid increase in venous return to the heart following stent placement has been reported.³¹ Acute thrombosis may occur following venoplasty or stent placement and can be treated with thrombolytics, systemic anticoagulation, or restenting in some cases. Tissue proliferation within a stent placed for benign disease should be treated with balloon venoplasty without additional stent placement. Both peripheral and central stent placement may lead to compression of adjacent nerves as in the cases of SVC stents causing recurrent laryngeal nerve paralysis with postprocedure hoarseness and brachial vein stents causing median nerve paralysis. Infectious complications are rare and are easily prevented by the use of meticulous sterile technique. Venous puncture site complications are similar to those seen with other venous access procedures and include venous thrombosis, hematoma, arteriovenous fistula formation, and venous pseudoaneurysm.

CONCLUSIONS

Endovascular treatment of chronic upper extremity and superior vena cava obstruction with venoplasty and stent placement is both safe and effective and should be considered first-line management in most cases. With proper technique, the technical success rate is high for the treatment of both malignant and benign disease. In the setting of malignancy, stent placement can provide near instant relief of symptomatic SVCS and is an important adjunct to chemotherapy and radiation therapy. In benign cases of obstruction, complication rates are low and only a small percentage of patients will require reintervention.

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