Endovascular Reconstruction of Malignant IVC and SVC Obstruction

A safe and effective treatment option to relieve the symptoms of chronic occlusion.

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Caval occlusion of malignant etiology is an insidious pathologic entity, resulting in substantial morbidity and limiting quality of life in severely ill and/or terminal patients. Relief from this condition relies upon astute recognition of the pathology and skilled intervention. Patients with long-standing chronic occlusions of the inferior vena cava (IVC) secondary to malignancy may present a diagnostic challenge. Onset can be slow, and the cause may not be obvious (ie, acquired symptomatology vs congenital defect). Symptoms and presentation vary between affected individuals based on various factors, including clot distribution, level of occlusion, activity level, and collateralization. Occlusion may commonly present as a dull aching pain in the extremities, as well as symptoms of venous claudication, in which lower limb swelling and discomfort are precipitated by exercise and relieved by rest and elevation. Venous ulceration can be seen in long-standing cases.

IVC SYNDROME

Intrahepatic occlusion of the IVC results in a particularly devastating symptom complex. The so-called IVC syndrome, initially described at our institution, results from intrahepatic constriction of the vena cava by primary or metastatic neoplasm. This constriction, in turn, causes a combination of signs and symptoms, the most noticeable and disabling of which is the rapid onset of ascites and anasarca of the lower extremities. This form of anasarca is differentiated from other causes of edema by its occurrence being only below the diaphragm. In cases of malignant compression of the IVC, attention should be paid to mass effect on adjacent venous structures, such as the hepatic and renal veins. Hepatomegaly may be present. Ascites and edema respond poorly to diuretics, and proteinuria may be present as a result of back pressure on the renal veins. Imaging typically demonstrates intrahepatic compression of the vena cava (Figure 1), often with increased development of collateral vessels, depending on the duration and severity of the obstruction.

TREATMENT OF IVC OCCLUSIONS

Although the specifics of treatment vary from patient to patient, there are some general principles governing the treatment approach. Anticoagulation is largely ineffective in relieving lower extremity symptoms, and thrombolytic therapy is of value only in acutely thrombosed IVCs. Initial therapy may involve external beam radiation to the region of the tumor exerting mass effect, as well as chemotherapy and medical optimization (diuretics, analgesics, etc.). In the past, venous bypass surgery was the only option in the most severe, refractory cases; however, the surgical option has had limited efficacy and applicability, with substantial associated morbidity. Surgical resectioning remains an option in patients for whom complete resectioning and cure may be feasible. In the majority of cases, endovascular recanalization is...
now largely considered the preferred method for palliation after failure of conservative therapy.\(^6\)

Preprocedural imaging can be of great value by allowing assessment of the clot burden, the stability and diameter of the native IVC, and the presence of collateral and branch vessels.\(^7\) Selection of the imaging modality is operator-dependent, but may include MR venography, CT venography (Figure 2), and/or ultrasound. These imaging modalities may also identify a superimposed acute thrombus, enabling the operator to plan the procedure with the expectation of thrombolytic infusion.

**TECHNIQUE**

For endovascular therapy, access is typically achieved via a transfemoral venous approach. A larger-caliber sheath (10–16 F) is typically used to allow stent placement. A 5-F catheter is advanced into the common iliac vein or the inferior aspect of the IVC, and venography is performed, usually demonstrating the level of obstruction and the presence or absence of collateral veins. Pressure transduction may be performed to assess the level of venous hypertension below the level of the obstruction. The occlusion is then crossed, which usually involves the use of a hydrophilic wire and catheter, although other devices and catheters may be employed. Once the catheter is across the lesion, a repeat pressure is measured to assess the venous gradient. If the gradient is considered significant (> 5 mm Hg), the vessel is subsequently recanalized.

Recanalization usually begins with venoplasty. Large-caliber, noncompliant balloons (eg, the 24-mm Atlas balloon, Bard Peripheral Vascular, Inc.) are used and are sized according to the nearest visible portion of normal appearing IVC. Given the extrinsic compression of the vessel by the tumor, it is unusual to achieve normal vessel caliber or durable patency with venoplasty alone. For this reason, metallic stents are usually placed at the level of stenosis/occlusion. However, if a substantial reduction in gradient and a good angiographic result are achieved, it may be preferred to perform cavoplasty alone, especially in patients with longer expected survival and in whom long-term stent patency may be a concern (Figure 3). When stents are deployed, they are typically self-expanding bare-metal stents (eg, Gianturco-Rosch Z-stents [Cook Medical] or Wallstents [Boston Scientific Corporation]).\(^8\) If suboptimal dilatation is achieved with initial cavoplasty and there is concern for caval rupture, the operator may prefer to place a balloon-expandable stent (eg, Palmaz stent, Cordis Corporation) deployed to a safe diameter, with a plan for more gradual recanalization of the IVC and repeat dilatation performed in a subsequent procedure. Covered stents may sometimes be employed in cases where there is increased concern for caval rupture. After postdeployment balloon dilatation, repeat pressure transduction is performed across the lesion to verify reduction/elimination of the gradient.\(^9\) Completion venography is then performed. Although bare-metal stent placement across branch vessels may be performed with relative safety,\(^6\) malignant stenosis or occlusion in adjacent branch vessels may need to be treated with venoplasty and/or stenting (Figure 4). After venoplasty and stenting, the intravascular devices are removed, and hemostasis is achieved with manual compression.

**SUPERIOR VENA CAVA SYNDROME**

Long-standing superior vena cava (SVC) occlusion presents differently than IVC occlusion. The symptoms of SVC syndrome include shortness of breath, cough, arm swelling, chest pain, dysphagia, orthopnea, distorted vision, hoarseness, stridor, headache, nasal congestion, nausea, pleural effusions, light-headedness, as well as severe swelling of the face, neck, and upper extremities.\(^10\) SVC occlusions are associated with malignant etiologies in the majority of cases, most commonly, primary lung neoplasms.\(^11\) However, an increasing number of SVC occlusions result from the placement of intravascular devices, including pacing wires, central venous catheters, and dialysis access catheters.
Improvement in flow was seen on poststenting images (E). seen in the patient’s hepatic vein (C), which was stented (D). Improvement in flow was seen on poststenting images (E).

TREATMENT OF SVC OCCLUSIONS

Patients presenting with SVC syndrome should promptly be imaged to assess the nature and level of occlusion. Frequently, collateral venous circulation may be demonstrated on these imaging studies (CT venography, MR venography), most importantly via ayzygous to hemiazygous collateral venous return. These collateral pathways are inadequate in a symptomatic patient, and the occlusion must be opened to re-establish flow and lower the venous pressure.

In patients with less-severe symptoms who are treatment naive, initial therapy may include chemotherapy and radiation directed toward debulking the tumor that is causing SVC compression. In cases where symptoms persist, or in which rapid resolution is required to stabilize the patient, endovascular intervention is indicated. The approach to intervention should be guided by preprocedural imaging and most commonly can be achieved via transjugular or transfemoral puncture. As with IVC occlusions, the intervention requires crossing the lesion with a wire and catheter (usually hydrophilic), and then flow can be re-established through the use of balloons and stents (most often bare metal, balloon expandable) to optimize radial strength and precise positioning (Figure 5). Some case series suggest that patency is improved when a covered stent is placed, although the risks of migration or exclusion of branch vessels is higher.

In practice, most SVC stents are still bare metal. Patients should experience a rapid improvement in symptomatology once the flow is re-established.

COMPLICATIONS

As with any procedure, complications are expected in the management of caval stenosis/occlusions. The most common complications to consider when performing these procedures include bleeding/caval rupture, stent migration, and rethrombosis. These complications are rare, and careful intervention utilizing good basic procedural skills will help to substantially mitigate such risks.

CONCLUSION

Although IVC and SVC occlusion are presentations that can be difficult to diagnose and manage, there are viable treatment options. Endovascular recanalization of IVC and SVC occlusions is safe and effective in relieving severe symptoms of chronic occlusion. There is always the possibility of complications, but these are rare and may be avoided with careful attention and technique.

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