Malignant Venous Obstruction: Superior Vena Cava Syndrome and Beyond

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Abstract

Venous obstruction in the cancer population can result in substantial morbidity and, in extreme cases, mortality. While venous obstruction can be caused by both benign and malignant etiologies in this population, the management of malignant venous obstruction as a palliative measure can be somewhat nuanced with respect to nonprocedural and procedural management, both with respect to treatment of the underlying malignancy as well as treatment of venous hypertension, which may be associated with venous thrombosis. Symptom severity, primary malignancy, functional status, and prognosis are all fundamental to the patient workup and dictate both the timing and extent of endovascular intervention. The morbidity and mortality associated with malignant obstructions of central venous structures, specifically the superior vena cava and inferior vena cava, can be significantly improved with endovascular management in appropriately selected patients. Thus, the pertinent literature regarding the clinical presentation, workup, and endovascular management of malignant central venous obstruction syndromes, with directed attention to superior vena cava syndrome and inferior vena cava syndrome, will be reviewed in this article.

Keywords
- interventional radiology
- venous obstruction
- malignancy
- lung cancer
- stent
- angioplasty

Objectives: Upon completion of this article, the reader will be able to describe the common etiologies of malignant superior vena cava obstruction; the clinical and imaging findings of superior vena cava obstruction; and the interventional radiologic techniques used to treat the disease process.

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Malignant venous obstruction of the superior and inferior vena cava (IVC) can result in significant morbidity and mortality. Venous obstruction may occur as a result of myriad of etiologies, including malignancy and infection from intravascular devices. While some of these etiologies are well characterized, particularly in superior vena cava (SVC) thrombosis, intrinsic vessel stenosis, and benign extrinsic compression, existing literature is sparse regarding

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Table 1 Common clinical findings in patients with superior vena cava obstruction

Neurological	Laryngopharyngeal	Facial	Chest wall and upper extremities
Headaches	Cough	Nasal stuffiness	Neck and chest wall venous distention
Blurry vision	Tongue swelling	Conjunctival edema, periorbital edema	
Papilledema	Dyspnea	Facial edema/plethora	Upper extremity swelling and plethora
Decreased level of consciousness	Stridor/laryngeal edema	Proptosis[58]	

malignant IVC obstruction. Discussion of treatment of symptomatic venous obstruction should be performed in a multidisciplinary approach, with input from medical oncology, radiation oncology, surgical oncology, hematology, and interventional radiology. Here, we will review stent revascularization of the superior and IVC as well as iliac veins, discussing indications, outcomes, and complications.

Superior Vena Cava Obstruction

Superior vena cava syndrome affects over 15,000 patients annually in the United States alone. While the severity of symptoms varies widely, severe SVC syndrome may be fatal. Typical symptoms of SVC syndrome can be categorized into four domains: neurological, laryngopharyngeal, facial and chest wall, and upper extremities (►**Table 1**).[1] Prior to the advent of antibiotics, the two most common causes of SVC syndrome were syphilitic thoracic aortic aneurysms resulting in SVC compression and bulky mediastinal adenopathy caused by tuberculosis.[1–4] In fact, the first reported case of SVC syndrome was described by William Hunter, a Scottish physician, in 1757 in a patient with syphilitic aortitis.[5] However, due to medical advancements, the etiology of SVC syndrome has evolved. Malignancies now account for ~70% of cases, but benign causes, such as thrombosis or stenosis from central venous catheters and pacemaker leads, are now increasing in prevalence.[6,7] Approximately 78 to 85% of malignant SVC obstructions occur secondary to lung cancer. Given its overall predominance (80% of all lung cancers), non-small cell lung is the most common cause of malignant SVC syndrome. However, small cell lung cancer, which accounts for only 15% of all lung cancer, is another common cause, accounting for 22% of all cases of malignant SVC syndrome.[1] Given its central location and its rapid growth, on a per patient basis, small cell lung cancer is approximately five times more likely to cause SVC syndrome than non-small cell lung cancer.[7,8] Lymphoma, usually non-Hodgkin's lymphoma, accounts for 10 to 15% of cases and the remainder are from other causes, most often breast cancer metastases.[1,9] In fact, SVC syndrome is the harbinger of yet undiagnosed malignancy in up to 60% of cases. Primary mediastinal malignancies such as germ cell neoplasms (►**Fig. 1**) or thymic tumors are responsible for the rest of the causes of malignant SVC syndrome.

The SVC is a thin-walled compliant vessel which drains the venous outflow from the head, upper extremities, and upper thorax into the right atrium. It accounts for approximately one-third of venous return to the heart.[1] Therefore, acute obstruction of the SVC results in significant elevation in intracranial venous pressures, which may increase to 40 mm Hg, from a normal range of 2 to 8 mm Hg.[1]

When the SVC is occluded, the body must rely on various venous collaterals to return blood to the heart.[10] Severity of SVC symptoms is inversely proportional to the development of the venous collaterals. There are four main collateral pathways from the SVC to the IVC: the largest direct collateral pathway from the SVC to the IVC is through retrograde blood flow via the azygos-hemiazygos vein consisting of the azygos vein, hemiazygos, intercostal, and lumbar veins.[10] The other collateral pathways include the internal and external mammary pathway (internal mammary, superior epigastric, inferior epigastric, and superficial thoracic veins), lateral thoracic pathway (lateral thoracic to thoracoepigastric, superficial circumflex, long saphenous, femoral veins), and the vertebral pathways (innominate vein, vertebral veins; intercostal, lumbar and sacral veins; azygos and internal mammary pathways).[10,11] Another collateral results in the "hot quadrate sign" seen in SVC obstruction. This is due to diversion of blood into the internal thoracic vein then into the superior epigastric vein, subsequently to the superior vein of Sappey, which drains into the left portal vein. This collateral pathway results in increased uptake on nuclear scans and enhancement on CT scan in the superior aspect of segment 4 of the liver which may be interpreted as hepatic pseudolesions[12–14] (►**Fig. 2**).

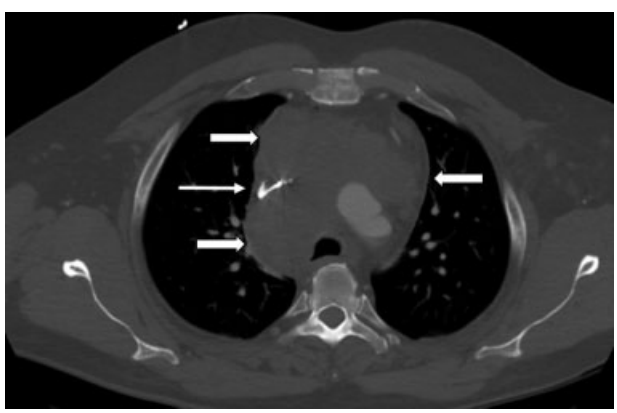

Fig. 1 A 50-year-old man with malignant mediastinal germ cell tumor. Axial contrast-enhanced CT demonstrating the infiltrating mediastinal tumor (thick arrows) with significant mass effect upon the superior vena cava (SVC) (thin arrow) resulting in near-complete SVC obstruction causing SVC syndrome.

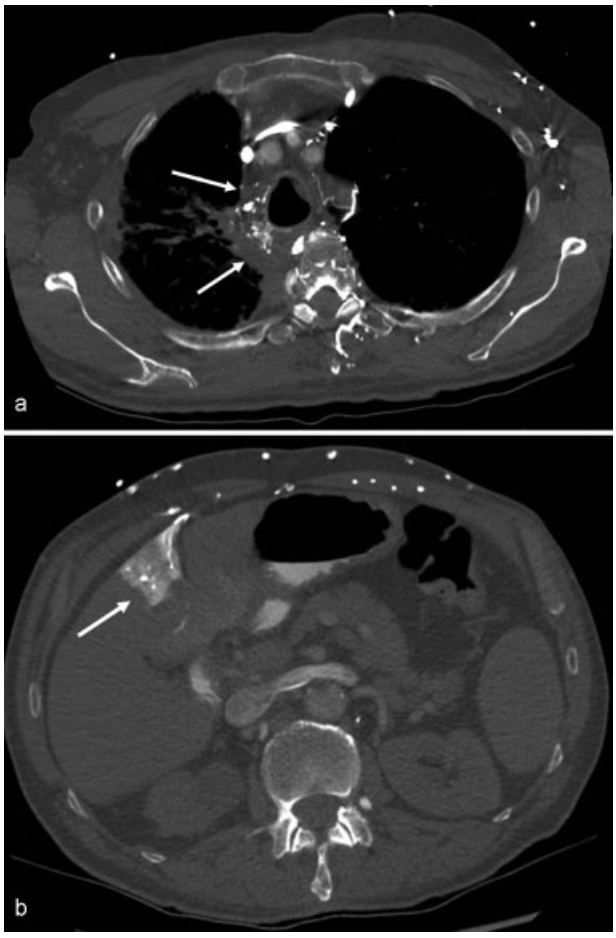

Fig. 2 A 74-year-old man with chronic obstructive pulmonary disease and metastatic appendiceal carcinoma to the mediastinum resulting in superior vena cava (SVC) occlusion. (**a**) Axial contrast-enhanced CT demonstrating tumor invasion into the mediastinum causing SVC obstruction (arrows). (**b**) Axial contrast-enhanced CT showing a perfusion abnormality in the superior aspect of hepatic segment 4 by drainage from the superior vein of Sappey, which connects branches of the superior epigastric and internal thoracic veins to peripheral portal branches of the left hepatic lobe ("Hot quadrant sign").

Collateral diversion of blood flow is highly dependent on the level of obstruction of the SVC, with the most important distinction being if the occlusion occurs above or below the origin of the azygous vein. If occlusion is below the level of the azygous vein, blood may be easily diverted to the IVC via retrograde flow of blood through the azygous vein (►**Fig. 3**). However, if the occlusion occurs above or across the origin of the azygous vein, blood can be diverted only through the smaller venous collaterals described above resulting in more severe symptoms.

Because it takes the body weeks to develop sufficient collateral pathways, the severity of symptoms is also dependent on the rapidity of SVC occlusion. Thus, patients may be asymptomatic if the azygous vein is patent and the speed of SVC occlusion is slow. On the other hand, patients may present with respiratory compromise due to edema of the larynx and pharynx and obtundation due to cerebral edema if the occlusion occurs rapidly. Death due to SVC compression is uncommon, only occurring in 3 in 1,000 cases.[6] Although the median life expectancy of patients with malignant SVC obstructions is 6 months, the aggressiveness of the underlying malignancy is the greatest predictor of survival.[1] Therefore, the etiology and histologic grade of malignant SVC syndrome must be elucidated not only to help guide therapy but to aid in prognosis.

Non–life-threatening symptoms of SVC syndrome include headache, cough, interstitial edema of the face resulting in periorbital and facial swelling, neck vein distention, chest wall vein prominence due to superficial collaterals, as well as upper extremity swelling and plethora.[1] Patients' symptoms may be exacerbated by maneuvers which increase supracardiac venous pressures such as laying down, leaning forward, or Valsalva maneuver.

Although the symptoms and physiologic signs of patients with SVC syndrome may be characteristic, confirmation with imaging is mandatory. Initial chest radiographs obtained in the emergency room are abnormal in 84% of cases, demonstrating mediastinal widening in 64% of cases.[15] Contrast-enhanced CT venogram is the mainstay of imaging and will help delineate the degree of SVC obstruction, etiology of obstruction, and collateral venous supply. Magnetic resonance imaging may also be used and offers time-resolved imaging, which helps further elucidate the level of obstruction and collateral venous supply. CT is particularly useful for demonstrating any evidence of superimposed thrombus, native diameter, length of SVC occlusion, and involvement of any additional venous structures. Knowledge of the above characteristics aids in planning for endovascular treatments, such as stent sizing and stent deployment landing zones as well as possible requirement for thrombolysis.

Treatment of Superior Vena Cava Syndrome

When severe symptoms such as hypotension or laryngeal/cerebral edema are present, SVC syndrome is a true emergency. Fortunately, most patients present prior to this stage, allowing for further workup with cross-sectional imaging before proceeding with treatment.[15] Additionally, if histology of the offending tumor is unknown, biopsy may be required to guide palliative or curative treatment such as chemotherapy or radiation to debulk or decrease size of the mass.[11] A PET-CT scan may also be helpful to further stage the malignancy, including nodal involvement in setting of lung cancer. Thus, the ultimate treatment of malignant SVC syndrome is predicated on tumor staging, histology, and prognosis.[16] Although this article will focus on endovascular interventions, chemotherapy and radiation will be briefly reviewed.

Chemotherapy may be the first line of treatment in patients who do not present with life-threatening symptoms, as symptoms may take up to 2 weeks to improve in even the most chemotherapy-sensitive tumors.[16] Radiation alone has been advocated for SVC syndrome due to small-cell lung cancer, improving symptoms in up to 80% of patients; however, Nicholson et al demonstrated that endovascular stents provide faster and greater symptomatic improvement when compared head to head with radiation alone.[17–20] Moreover, the local edema caused by radiation may exacerbate patients' symptoms in the short term.[21] Additionally, maximum cumulative radiation limits are often reached prior to durable symptom

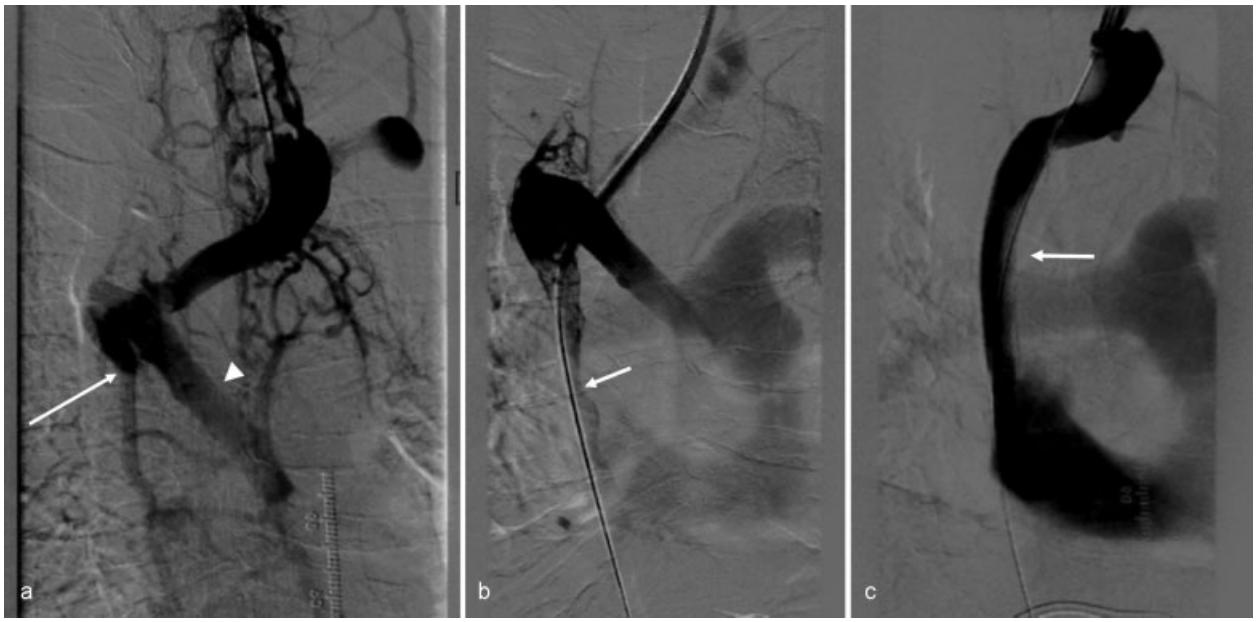

Fig. 3 Superior vena cava (SVC) obstruction in the setting of breast cancer resulting in SVC syndrome. (**a**) SVC occlusion (long arrow) with multiple collaterals including lumbar veins and azygos vein (arrow head). (**b**) Wire access across the native SVC (arrow). (**c**) Endovascular stent recanalization of the SVC resulting in resolution of collateral venous flow; only the SVC (arrow) was visualized on digital subtraction venogram after the SVC was reopened.

relief.[22] Also, radiation itself may cause SVC syndrome in the long term due to fibrosis.[23]

Finally, chemoradiation has been found to have the best outcome in epithelial tumors, as it allows for maximal tumor response.[16] However, chemoradiation is plagued by numerous side effects and low response rates in certain tumors.[24] Corticosteroids may temporize symptoms in certain types of steroid-responsive malignancies such as lymphoma or thymoma but otherwise have not been shown to improve symptoms.[25] It is important to consider the expected rapidity of resolution of external SVC compression, especially in younger patients who have highly radiosensitive or chemosensitive malignancies prior to stent placement.

Endovascular treatment as first-line therapeutic treatment for malignant obstruction of the SVC remains controversial, owing to lack of rigorous evidence, but has been advocated by multiple authors.[24,26–28] Advocates of first-line venous stenting cite immediate resolution of symptoms, usually within 24 to 72 hours, high technical success rate, low complication rate, and lack of interference with subsequent treatments such as chemotherapy and radiation treatments, and ability to treat without the need for confirmation of histologic diagnosis.[24] Stenting is typically reserved for the following three scenarios: (1) patients with severe acute symptoms such as respiratory distress from laryngeal edema or airway obstruction and altered mental status from elevated intracranial pressures, (2) patients who have continued persistent moderate symptoms despite chemotherapy, and (3) patients in whom chemotherapy and radiation are contraindicated. Technical success for endovascular therapy of the SVC is high, between 84.5 and 100%.[27,29,30]

Different techniques have been proposed to allow for wire access through the occluded vena cava. When possible, a single access via a femoral approach is often used which allows for venograms of the bilateral brachiocephalic veins, evaluation of the brachiocephalic vein confluence, and length of SVC narrowing. When simple recanalization techniques fail, advanced techniques such as sharp recanalization with back-side of hydrophilic wires, needles, or radiofrequency wires have been successful, but should be used judiciously due to the potential risk of hemopericardium or hemothorax.[31] When using sharp recanalization, it is advised to have a target so that the three-dimensional anatomy on multiple fluoroscopic obliquities or fluoroscopic cone-beam CT is clear.

Fagedet et al showed that the clinical failure rate significantly increased in the setting of superimposed SVC thrombosis, which was more complex to treat.[27] When SVC thrombus is present, it is best to perform thrombolysis within 5 days after symptom onset. Gray et al found that technical success decreased from 88% to less than 25% after 5 days.[32] Intracranial metastases and cerebral venous infarct from elevated venous pressures must be ruled out before giving lytic medications.

The SVC is usually compressed by the tumor rather than invaded by it; venous stenting in concert with angioplasty is uniformly required to maintain SVC patency (►**Fig. 4**). Unfortunately, the recurrence rate of SVC syndrome due to stent occlusion is quite variable. Lanciego et al showed a primary stent patency rate of 86.6% and primary assisted patency rate of 93.3% over their 15 years of experience, in 149 patients.[24] Stent patency was approximately 85% at 6 months and 75% at 24 month.[24] However, other authors cited stent occlusion leading to SVC syndrome recurrence in up to 41% of cases due to stent invasion, compression, or thrombosis.[30] Despite the possibility of high relapse rates, repeat stenting appears to be effective in up to 75% of cases.[27]

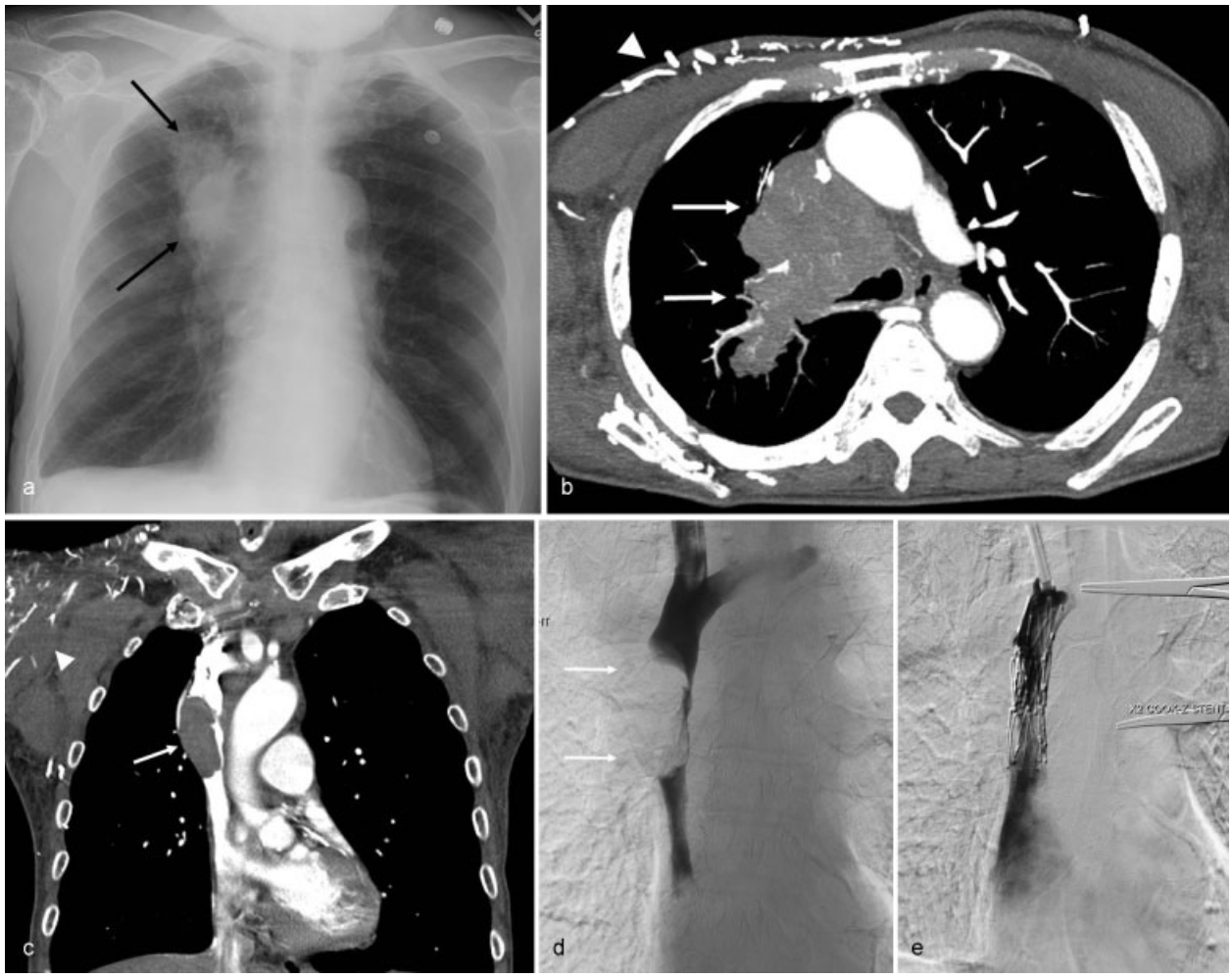

Fig. 4 Superior vena cava (SVC) syndrome due to infiltrating mediastinal mass. (**a**) Frontal chest radiograph demonstrating right perihilar opacity (arrows). (**b**) Axial contrast-enhanced CT demonstrating aggressive-appearing invasive mediastinal mass (arrows) resulting in near-complete SVC occlusion and numerous chest wall venous collaterals (arrow head). (**c**) Coronal contrast-enhanced CT showing significant compression upon the SVC from the mediastinal mass (arrow) with numerous soft-tissue venous collaterals (arrowhead). (**d**) Digital subtraction venogram with severe long-segment narrowing of the SVC (arrows) with poor distension of the lower SVC due to low flow due to obstruction. (**e**) Placement of two 15 mm × 50 mm (diameter × length) Gianturco-Roche Z-stents, resulting in wide SVC patency and resolution of SVC syndrome.

Owing to the propensity of tumor invasion and relapse, there is controversy as to whether covered stents such as expanded polytetrafluoroethylene stents may result in improved patency (►**Fig. 5**). Gwon et al demonstrated improved patency of covered stents versus noncovered stents; however, survival and clinical success rate did not differ.[30] Nonetheless, covered stents may cover collateral veins or even the contralateral brachiocephalic vein. Additionally, large diameter covered stents are often not readily available and require larger access venotomies compared with bare metal stents.[27] When comparing Wallstents (Boston Scientific, Marlborough, MA), Gianturco-Roche Z-stent (Cook Medical, Bloomington, IN), and Palmaz Genesis stents (Cordis Corporation, Hialeah, FL), no significant difference in outcomes were identified.[33] Caution, however, is suggested whenever using balloon expandable stents as they, unlike self-expanding stents, can migrate once the underlying tumor has shrunk.

Anticoagulation has been validated during angioplasty and stent placement and has become the standard of care. Thus, 3,000 to 5,000 units of heparin is recommended during SVC angioplasty and stenting. While short-term anticoagulation has been recommended (3–4 days postprocedure), there is insufficient evidence to support long-term anticoagulation in this patient population.[29,33] Long-term anticoagulation can be considered on a case-by-case basis depending on the risks of rethrombosis and patient history.

When malignant SVC obstruction occurs in the setting of bilaterally occluded brachiocephalic veins, only a single in-line venous outflow needs to be reopened to allow for SVC symptom resolution. Dinkel et al demonstrated that when both brachiocephalic veins are occluded, recanalization of one of the two brachiocephalic veins is sufficient to alleviate SVC syndrome with lower stent thrombosis and complication when compared with kissing stents from bilateral brachiocephalic vein stenting.[34] Thus, it is inadvisable to attempt bilateral brachiocephalic and SVC recanalization to relieve SVC syndrome. Recanalization of both brachiocephalic veins and placing "kissing stents" into the SVC is advocated only if there is symptomatic upper extremity edema with concomitant SVC syndrome (►**Fig. 6**).

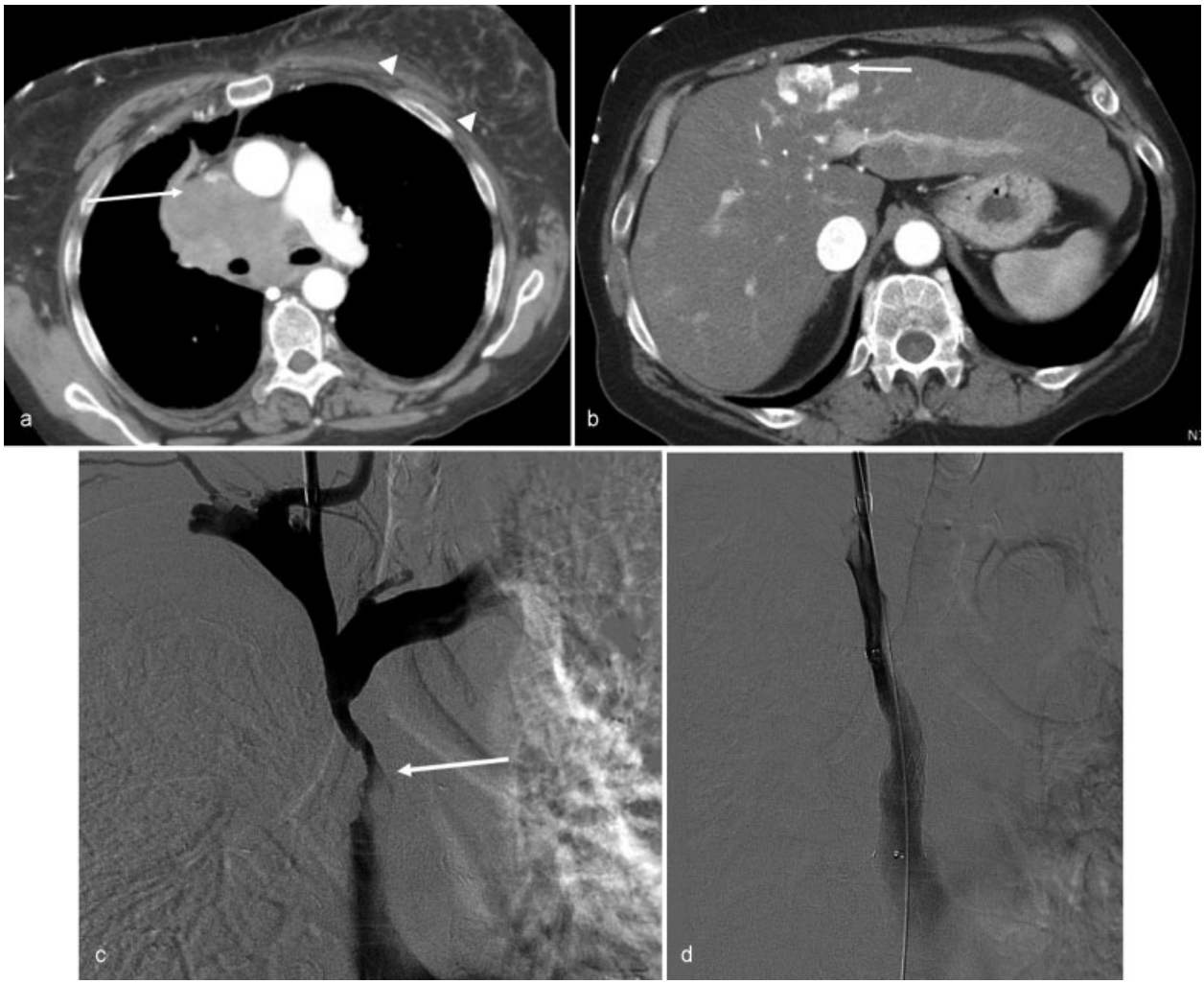

Fig. 5 A 58-year-old woman with metastatic squamous cell lung cancer with extensive mediastinal invasion and compression of the superior vena cava (SVC) (arrow) resulting in SVC syndrome (**a**)—note left breast edema (arrowheads). (**b**) Collateral flow from SVC occlusion demonstrating "hot quadrate sign" (arrow). (**c**) Digital subtraction venogram demonstrating near occlusive narrowing of the upper SVC (arrow) and retrograde flow up the left brachiocephalic vein. (**d**) Endovascular stent using a 13.5-mm Bard Fluency Plus (Bard, Tempe, AZ) covered stent with immediate resolution of SVC syndrome.

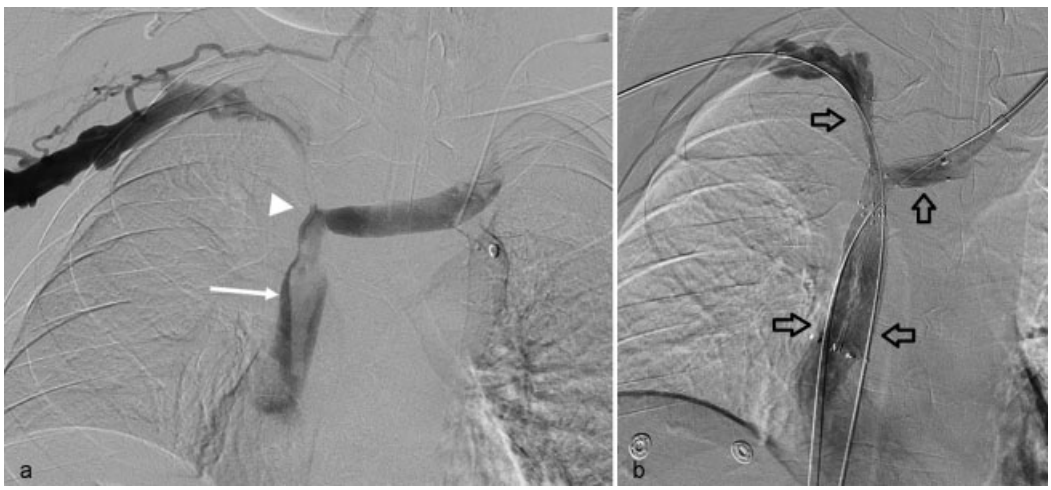

Fig. 6 A 52-year-old man with small cell lung cancer and severe respiratory distress from SVC syndrome requiring intubation. (**a**) Digital subtraction venogram showing severe narrowing of the right brachiocephalic vein and confluence of the left brachiocephalic vein (arrowhead) and SVC with large mobile tumor thrombus (arrow). (**b**) Bilateral brachiocephalic "kissing" stents into the superior vena cava (arrows). Patient's symptoms improved significantly over the next 24 hours.

Recanalization of the SVC carries inherent risks; the most dreaded complication is caval rupture resulting in hemopericardium and cardiac tamponade. Fortunately, SVC rupture is rare, occurring in 0.1 to 1.8% of cases. However, hemopericardium resulting in cardiac tamponade frequently culminates in death despite aggressive intervention.[6] The superior sinus of the pericardium extends to cover one-fourth to one-third of the length of the SVC.[35]

The role of predilatation of the SVC narrowing has also garnered debate; however, we feel that predilatation of the narrowed or occluded SVC is warranted to allow for accurate stent placement and minimize postdeployment dilatation and thrombosis. Fagedet et al demonstrated that a stent diameter greater than 16 mm was associated with increased risk of SVC rupture and pulmonary edema.[27] Complications associated with angioplasty and stent placement occur in 3 to 7% of patients; early complications include acute pulmonary edema from reopening the SVC, stent migration, pulmonary embolism, bleeding, and hematoma at the insertion site.[1,36–38] Late complications include bleeding, stent reocclusion, and death.[24] Increased caution should be exercised in patients who have undergone recent radiation; although increased risk of injury after radiation has been demonstrated only in the major arteries, it stands to reason that after radiation therapy the SVC may be more predisposed to rupture with aggressive anPatients with malignant SVC syndrome frequently require central venous access devices for medication administration, hydration, or even nutrition. The dilemma of placing a central venous access after SVC stenting is all-too-common as SVC thrombosis and occlusion is known to occur as direct result of central venous catheters.[41,42] However, Clark et al demonstrated that in 33 patients, there was no significant difference in symptomatic central venous stent occlusion or asymptomatic in-stent stenosis as a result of a central venous catheter placed at the same time as SVC stenting in the setting of malignancy.[43]

Inferior Vena Cava Obstruction

Narrowing of the IVC may arise as a result of congenital anomalies, which are usually incidental and asymptomatic, membranous obstruction of the IVC (►**Fig. 7**), bland thrombus, or impingement or ingrowth of tumors into or upon the IVC. The most common primary tumor of the IVC is leiomyosarcoma which mainly affects women in their fifth to sixths decade of life and portends a poor prognosis.[44] Much more commonly, a myriad of tumors may encroach and narrow the IVC such as hepatocellular carcinoma (HCC), adrenocortical carcinoma, renal cell carcinoma, and Wilms' tumor via direct local extension, as well as metastases from lung and kidney.[44,45] Additionally, mass effect from tumor metastases or lymphoma into retroperitoneal lymph nodes may also occur. Thrombus as a result of hypercoagulability or direct tumor thrombus may also result in flow limiting venous narrowing.[46]

Symptoms of IVC obstruction vary greatly from asymptomatic to debilitating and correlate with rapidity of IVC narrowing or occlusion. Symptoms of IVC occlusion or severe narrowing are diverse but correlate with increased intravenous pressure below the level of obstruction. In fact, the pressure gradient across the IVC occlusion may be as high as 20 mm Hg; 3 mm Hg gradient across venous stenoses have been shown to be significant.[47,48] Increased venous pressure results in lower extremity edema, anasarca to the level of obstruction—always occurring below the diaphragm, pelvic heaviness with possible scrotal edema and pain, and new ascites. The increased intravenous pressure and edema decrease patient's ability to ambulate and exercise, possibly resulting in venous ulcerations. Renal vein thrombosis may result if the occlusion is cephalad to

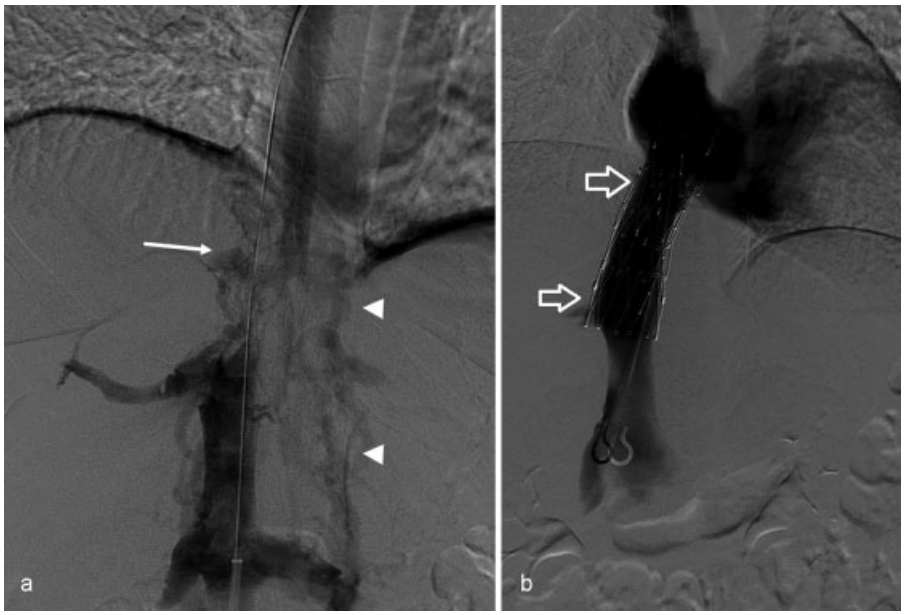

Fig. 7 (**a**) Membranous occlusion of the suprarenal inferior vena cava (IVC, arrow) resulting in a hepatic vena cava-Budd-Chiari syndrome and abnormal liver function tests and lower extremity swelling. Note the enlarged lumbar collaterals (arrowheads). (**b**) Recanalization of the suprarenal IVC with Gianturco-Roche Z-stents (arrows), resulting in significant improvement in liver function.

the renal veins (►**Fig. 8**). Renal vein outflow obstruction may result in renal insufficiency, proteinuria, and hematuria.[47,49–51] Similarly, if the obstruction involves the hepatic veins, symptoms of Budd-Chiari may occur including abdominal pain, hepatomegaly, and ascites, as well as splenomegaly, abdominal wall varices, and lower extremity swelling. While obstruction of hepatic venous drainage from any cause, including malignancy, is termed *Budd-Chiari syndrome*, the subtype secondary to hepatic IVC obstruction is termed *hepatic vena cava Budd-Chiari syndrome*.[52] Unfortunately, the crippling edema and ascites in patients with IVC occlusion is often refractory to diuretic therapy due to the underlying IVC occlusion and severely increased lower extremity venous pressures; endovascular intervention with stent placement is often necessary.

There are limited therapeutic options for these end-stage patients; one must remember that these procedures are performed for palliation and improvement in quality of life. Chemotherapy options are available to treat the underlying malignancy; however, these may prove too toxic in these sick patients. Additionally, surgical bypasses of the IVC with concurrent tumor resection or IVC and tumor resection without reconstruction have all been described,[53] but is fraught with complications including myocardial infarction, chyle leaks, pulmonary emboli, and renal failure requiring

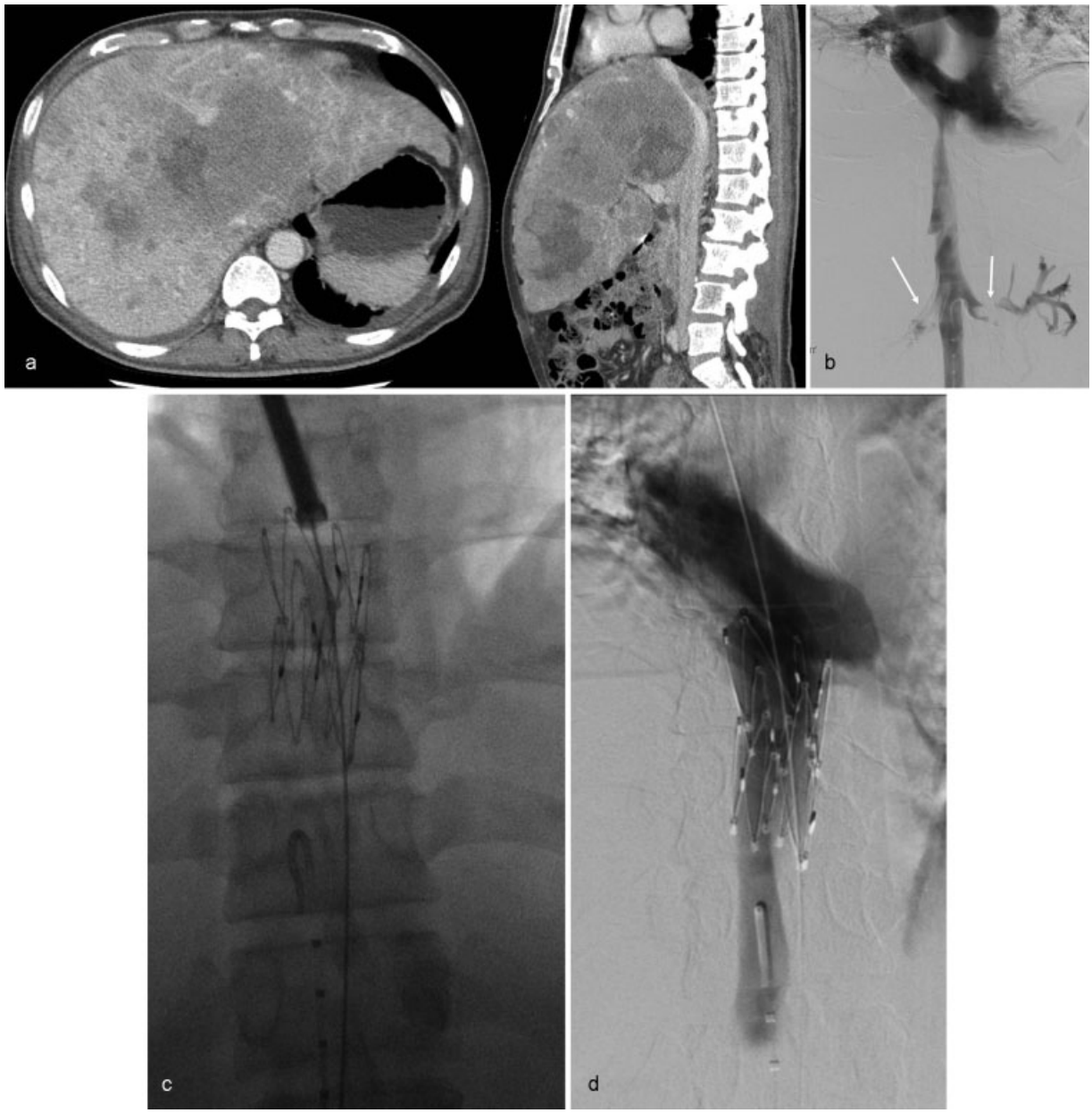

Fig. 8 A 54-year-old man with massive metastatic rectal carcinoid tumor to the liver. Patient was noted to have external compression of the intrahepatic inferior vena cava (IVC) resulting in disabling lower extremity edema. (**a**) Axial and sagittal contrast-enhanced CT demonstrating extensive liver infiltration from metastases. (**b**) Digital subtraction venogram demonstrating severe IVC narrowing with gradient across the lesion measuring 18 mm Hg. Filling defects compatible with bilateral renal vein thrombi are demonstrated (arrows). (**c**) Placement of a 30 mm × 5 cm (diameter × length) Gianturco-Roche Z-stent. (**d**) Digital subtraction venogram demonstrating patency of the intrahepatic IVC.

hemodialysis especially in the fragile oncology population.[53,54] Radiation therapy has also been attempted for malignant obstruction of the cava with suboptimal results.[19] Moreover, the local edema caused by radiation may exacerbate patients' symptoms in the short term.[21]

Although the literature regarding stenting for malignant IVC obstruction is scarce, the available literature demonstrates promising outcomes and high technical success of 78 to 100%.[21,47,49,55] Razavi et al demonstrated a primary patency rate at 19 months of 80% and primary assisted patency rate of 87%.[56] In our institution, we usually obtain either triple phase CT (noncontrast, arterial phase and delayed phase) or CT venograms prior to IVC recanalization procedures to evaluate the length of IVC occlusion, possible involvement of the renal or hepatic veins, the presence of thrombus, and patency of veins both caudal and cephalad to the mass. Cross-sectional imaging greatly aids with preprocedure planning; venous access sites depend on the extent of the mass; however, a solitary femoral or femoral and internal jugular accesses are preferred. Once the lesion is crossed and landing zones for the stent are delineated, we routinely perform venoplasty prior to stent deployment. Review of the literature has demonstrated that Gianturco-Roche Z-stents and Wallstents are favored due to their large available diameter. Devcic et al contend that Gianturco-Roche Z-stents suffer from low radial strength and large interstices which are both suboptimal in the setting of malignant obstruction and resulted in a higher incidence of reintervention.[49] We favor Wallstents due to their large available diameter size and lengths; however, they too are burdensome as they are prone to unpredictable shortening and underdistention during deployment which increases risk of stent migration. Devcic el al described similar problems with documented complications including stent extension and migration into the right atrium most commonly if the majority of the stent was deployed cephalad to the IVC narrowing or if the stent was placed from the suprarenal IVC to the right atrium.[49] Therefore, extra care and planning must be taken in these types of situations as stent migration into the heart has been associated with arrhythmias and hemopericardium.[21,57] Other documented complications include fever, pain, bleeding from anticoagulation, pulmonary emboli, and septic shock.[21]

Additional Venous Obstructions

While SVC and IVC obstructions often receive the greatest attention with respect to endovascular symptom palliation in oncology patients, obstruction, stenosis, and thrombosis in more peripheral deep venous structures are not uncommon in the oncology population. For example, iliofemoral and iliocaval stenosis as a result of extrinsic compression from lymphadenopathy, peritoneal implants, or from iatrogenic causes such as surgical intervention or radiation should be considered and evaluated in patients with extremity edema of unknown etiology, especially if asymmetric. Additionally, in oncology patients with recurrent or central DVT, further inquiry into venous compression should be undertaken to exclude lesions where more aggressive treatment can result in improved symptom palliation.

Conclusion

Palliation of malignant venous obstruction is multifaceted, requiring multidisciplinary expertise for optimal management. Obstruction of the SVC, IVC, and iliac veins can be a source of morbidity and mortality in the cancer population. In all cases, the acuity, extent of hemodynamic compromise, anatomic location of the obstruction, and presence/extent of thrombus often dictate the initial management and whether urgent endovascular intervention is needed. In all cases, consideration for the symptom severity, underlying etiology, prognosis, tumor biology, and ability to tolerate chemoradiation should be considered. Endovascular stenting of both the SVC and IVC has shown rapid symptom alleviation and low complication rates and should be considered as an option in patients with malignant obstruction.

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