

Superior Vena Cava Rupture and Cardiac Tamponade Complicating the Endovascular Treatment of Malignant Superior Vena Cava Syndrome: A Case Report and Literature Review

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Case Report

A 47-year-old man with known metastatic small cell lung cancer presented with 1 week of right arm swelling, facial flushing, shortness of breath, right hand weakness, and intermittent blurry vision. Physical examination revealed right upper extremity edema and facial plethora. Computed tomography (CT) of the chest confirmed enlargement of a right upper lobe mass causing near-complete occlusion of the right brachiocephalic vein and superior vena cava (SVC) (►Fig. 1), consistent with SVC syndrome (SVCS). After extensive discussion regarding the risks and benefits of the procedure, the patient elected to proceed with endovascular reconstruction of the central veins.

Bilateral basilic vein access was obtained, and digital upper extremity venography confirmed near-complete occlusion of the SVC (►Fig. 2). The occlusion was traversed using a 0.035-inch wire and a 5F catheter, and intraluminal crossing was confirmed with contrast injection. Through-and-through access was obtained via the right common femoral vein, and the SVC and brachiocephalic veins were dilated using 8- and 10-mm balloons (►Fig. 3). Venogram immediately following the 10-mm angioplasty demonstrated contrast extravasation into the mediastinum and likely the pericardial space, indicative of SVC rupture (►Fig. 4). Ultrasound revealed a thin pericardial effusion, which, given the patient's history of radiation, was deemed sufficient to cause tamponade. Overlapping 13 mm × 5 cm Viabahn-covered stents (Gore Medical, Flagstaff, AZ) were placed from the central aspect of the left brachiocephalic vein to the atriocaval junction (►Fig. 5). However, the

patient rapidly decompensated, and despite aggressive resuscitation following advanced cardiac life support (ACLS) protocol, including pericardiocentesis, the patient expired on the table.

Discussion

SVC Syndrome

SVCS results from elevated venous pressure in the upper extremities and head caused by obstruction of the SVC. The dominant cause of SVCS has varied over time, and the majority of cases today are caused by malignancy, typically primary bronchogenic carcinoma. Benign processes, such as stenosis and thrombosis induced by central venous catheters, are responsible for an increasing minority of cases.¹

The majority of patients with SVCS experience face, neck, and upper extremity swelling; dyspnea at rest; and cough. Chest pain, dizziness, syncope, and headache occur in 10 to 20% of patients.¹ While these symptoms can be alarming to patients, death due to SVCS may be as infrequent as 3 in 1,000 cases.² Stridor, altered mental status, and syncope may indicate possible neurologic compromise or impending respiratory collapse and should prompt rapid treatment.

Primary treatment of SVCS is directed at the underlying abnormality, which for malignant conditions usually involves chemotherapy and/or radiation therapy. Endovascular therapy can offer rapid and enduring symptomatic relief and is an important adjunctive therapy. It is indicated when the patient desires rapid symptomatic relief, displays signs of respiratory or neurologic compromise, and in cases of treatment-resistant malignancy.³

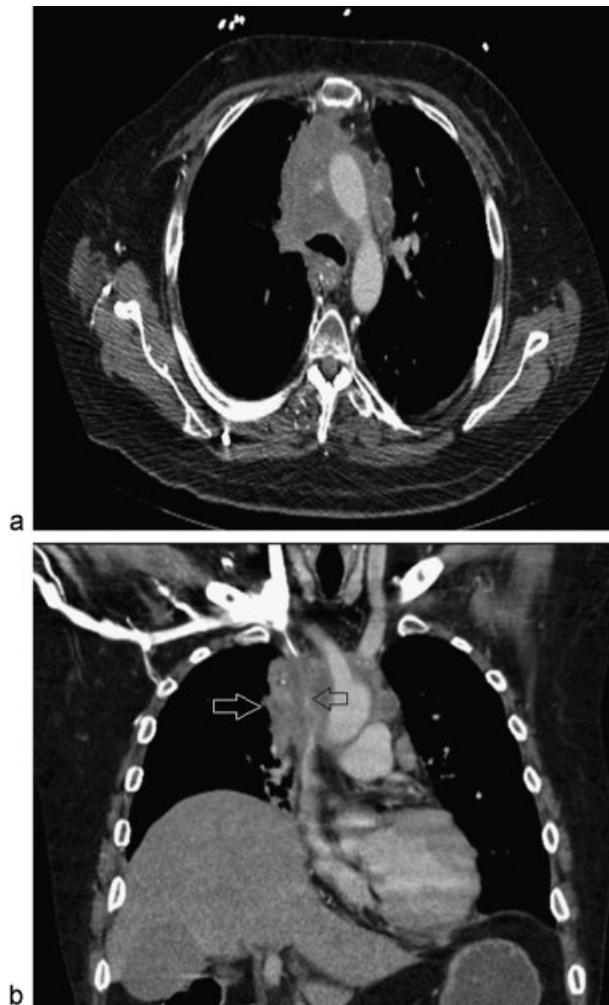


Fig. 1 Axial (a) and coronal (b) contrast-enhanced CT images of the chest performed 4 days prior to the attempted venous reconstruction. A spiculated right upper lobe mass (white arrow) causes near-complete occlusion of the SVC (black arrow).

Therapeutic Stenting of the SVC

Charnsangavej and colleagues first described endovascular treatment of SVCS in 1986. It has since become a widely accepted therapy because of its favorable safety and efficacy profile.⁴ In a review of 19 studies involving 884 cases of SVC stenting, Nguyen and colleagues found a primary efficacy rate of nearly 95%, with major complications occurring in only 4% of cases and death in 2%. Of the 17 fatalities, 7 were caused by severe hemorrhage, 3 by respiratory events, and 4 by cardiac complications—including 1 cardiac tamponade.⁵

In this initial description of SVC stenting, Charnsangavej and colleagues used Gianturco Z stents.⁴ Since then, an array of stents have been used to treat SVCS, but there is a paucity of data assessing individual stent performance. However, several observations may help guide stent selection. In a series of 164 patients, Fagedet and colleagues found an increased rate of complications when stents greater than 16 mm were used.⁶ Other authors have proposed that self-expanding stents may be better suited for use in tortuous vessels and when occlusion extends into the brachiocephalic veins.⁵



Fig. 2 Digital subtraction venogram obtained prior to intervention. The SVC and right brachiocephalic vein are occluded and collateral vessels (arrows) are present.

Finally, when thrombus extends into both brachiocephalic veins, unilateral brachiocephalic vein stenting effectively relieves symptoms while avoiding the higher complication and occlusion rates associated with bilateral brachiocephalic vein stenting.^{6–8}

Symptomatic relief after stent placement is usually rapid. Close to 90% of patients experience some degree of relief within 24 hours of the procedure and another 5% within 5 days.^{3,9} Although stents do occlude, most patients do not require reintervention. Gwon and colleagues found a stepwise decrease in stent patency at 3, 6, and 12 months—79, 67,

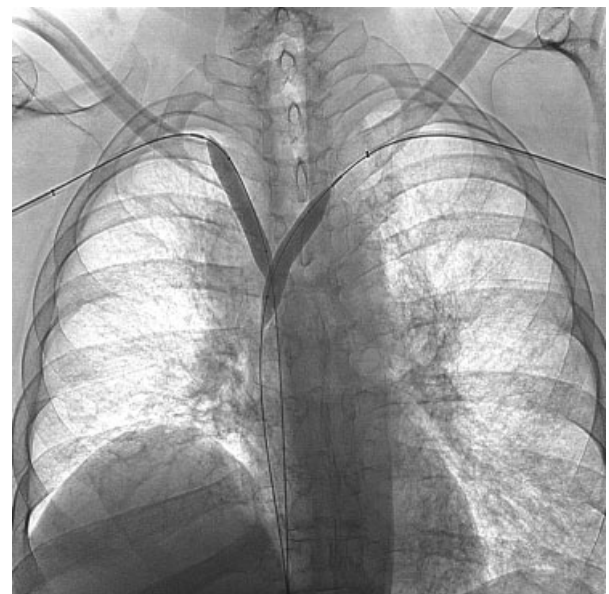


Fig. 3 Fluoroscopic image obtained during angioplasty of the bilateral brachiocephalic veins.



Fig. 4 Digital subtraction venogram. Contrast material in the pericardium (arrow) is indicative of SVC rupture and evolving cardiac tamponade.

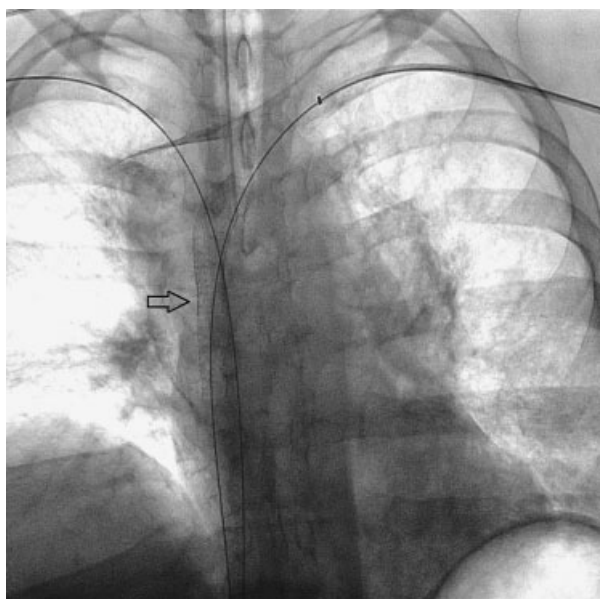


Fig. 5 Fluoroscopic image of the thorax after placement of overlapping 13 mm × 5 cm Viabahn-covered stents (arrow). An endotracheal tube is now visible.

and 48%, respectively. However, patency rates with covered stents remained greater than 90% at 1 year follow-up, which may suggest a benefit to using these devices when SVCS is caused by infiltrative or aggressive tumors.⁹

Guidelines for intraprocedural and postprocedural anticoagulation are scant. Although some authors prescribe postprocedural anticoagulation, there is a trend toward using antiplatelet therapy with aspirin and clopidogrel alone. Fagedet and colleagues found a nonsignificant trend toward

increased bleeding with heparin, warfarin, and aspirin, and no decrease in reocclusion rates, which brings into question the utility of these therapies.⁶ Three to 6 months of post-procedure, antiplatelet therapy with aspirin and clopidogrel is an unproven but common therapeutic regimen.

SVC Rupture and Cardiac Tamponade as a Complication of Stenting

SVC rupture and cardiac tamponade are among the most catastrophic complications of SVC stenting. They are fortunately rare. Nguyen and colleagues found only one case of SVC rupture among the 884 cases reviewed (0.1%).⁵ Other authors have reported rates of tamponade as high as 1.8%.⁶ A total of 16 cases of SVC perforation and hemopericardium have been described in the literature.^{10–24} The majority of ruptures became clinically apparent near the time of stenting—seven intraprocedurally, six within 1 hour of the procedure, and one more within 24 hours. However, fatal ruptures caused by strut erosion into the pericardium have been reported at 3 and 6 months postprocedure.^{20,22} Seven of these 16 perforations resulted in death. Of the nine surviving patients, three were successfully treated with pericardial drainage, three with covered stent placement, and three with open surgery.

Several techniques may help mitigate the risk of SVC rupture. Extravascular wire crossing of the stenosis can result in caval perforation.¹¹ To avoid this, a loop snare advanced from a femoral access site to the inferior aspect of the occlusion can be used as a target for the advancing wire.²⁵ Once the stenosis is crossed, the snare can be used to create through-and-through access to facilitate stent placement.

Vascular perforation can also occur during angioplasty.^{14,24} There is no clear consensus on whether angioplasty should be performed prior to stent placement, and the use of this technique will largely depend on operator experience and patient anatomy. When angioplasty is used, conservative serial dilations are probably safest. If rupture is identified, the balloon should be immediately reinflated to slow the rate of extravasation.

Perforation of the SVC by stent struts can lead to intrapericardial hemorrhage and tamponade.^{16,18,20} Some authors postulate that the risk of hemopericardium can be decreased by placing the inferior border of the stent above the pericardial reflection, which can be reliably predicted by the level of the carina on fluoroscopy.^{14,26,27} While the lowest 3 to 8 cm of the SVC are contained within the pericardium, intrapericardial hemorrhage from SVC perforation above this level can still occur because the medial aspect of the SVC adheres to the pericardium up to the level of the aortic arch.^{12,26} Thus, suprapericardial stent placement likely decreases, but does not eliminate, the probability of intrapericardial perforation.

Diagnosis of SVC Rupture and Cardiac Tamponade

Despite meticulous preprocedure planning, stenting across irradiated and tumor-infiltrated vessels can lead to perforation and pericardial tamponade. Minimizing mortality from rupture and tamponade requires prompt recognition and treatment. In all the reported cases of intraprocedural venous rupture, contrast extravasation and pericardial pooling were

fluoroscopically evident.^{11,14,17,21,23,24} However, familiarity with the physical, electrocardiographic, and echocardiographic findings of cardiac tamponade can confirm angiographic findings and is necessary to diagnose postprocedure SVC rupture and cardiac tamponade.

Beck's triad of hypotension, increased jugular venous distension, and muffled heart sounds is taught as the classic clinical presentation of cardiac tamponade.²⁸ However, the triad is neither sensitive nor specific enough to be relied upon for diagnosis.²⁹ Patients with tamponade, including tamponade due to SVC rupture, almost universally demonstrate tachycardia and hypotension.³⁰ Additionally, patients with SVC rupture most commonly demonstrate dyspnea, hemoptysis, diaphoresis, and chest pain. These findings should prompt ultrasound interrogation of the pericardium.

The presence of pericardial fluid in the inelastic pericardium causes the heart to swing back and forth and leads to atrial and/or ventricular infolding. The "swinging heart" sign can occasionally be observed during echocardiography and fluo-

roscopy. This motion also results in alterations in the magnitude of the QRS complexes on electrocardiogram, that is, electrical alternans (→Fig. 6).³¹ Atrial infolding can be quantified by the index of biatrial collapse—biatrial diameter at the mid-atria divided by the diameter at the level of the mitral and tricuspid valve during end diastole. Values of 0.85 and below are sensitive (100%) and somewhat specific (82%) for tamponade.³² Specificity increases to close to 100% when infolding persists throughout systole.³³

Treatment of Rupture and Tamponade

Successful management of SVC rupture and cardiac tamponade requires reestablishing the integrity of the SVC and restoring cardiac output. The extent and order of therapy will be highly dependent on the clinical situation, particularly the patient's cardiovascular status. If SVC extravasation is noted prior to cardiovascular collapse, reestablishing the integrity of the SVC must be prioritized. When rupture is identified after collapse, resuscitation should follow ACLS protocol,

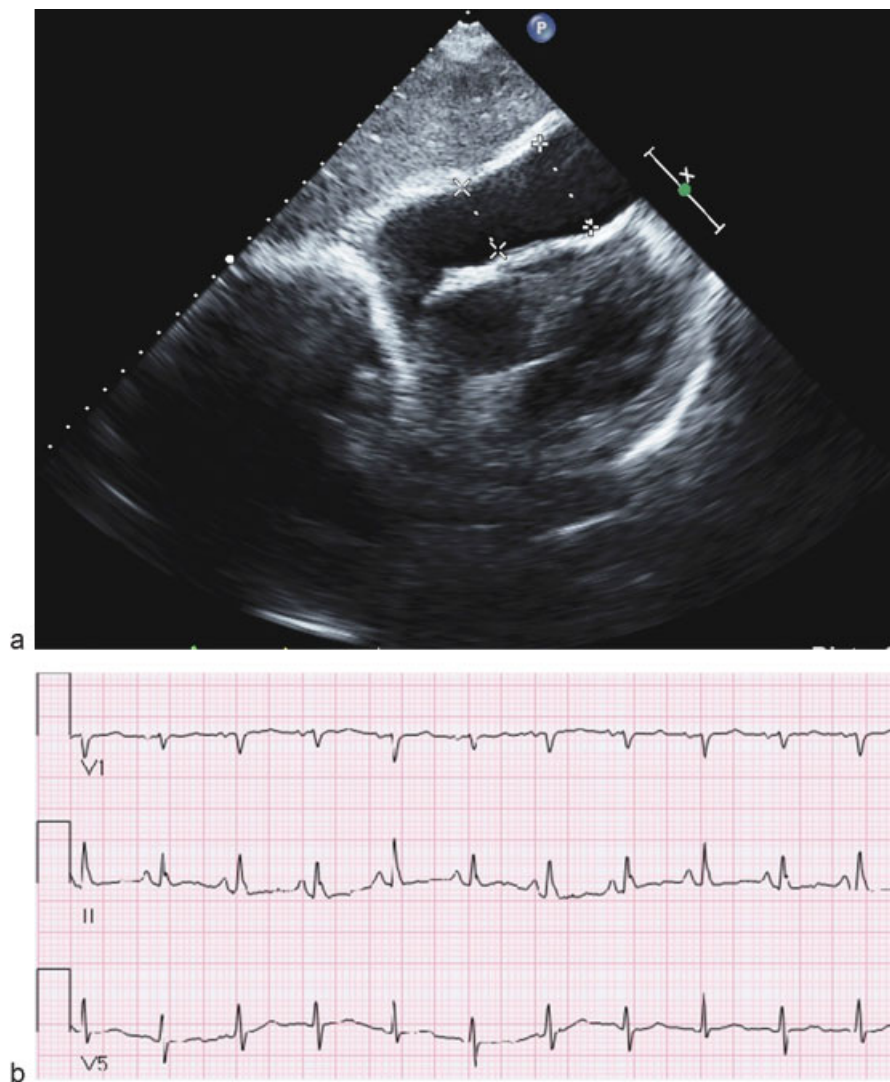


Fig. 6 Images obtained from another patient with cardiac tamponade. (Images courtesy of Kyle Harry, MD). (a) Transthoracic echocardiogram demonstrating a large pericardial effusion (calipers). (b) Electrocardiogram demonstrating electrical alternans, noted by alterations in the magnitude of the QRS complexes.

which will include the treatment of tamponade and extravasation.

When extravasation is identified, anticoagulation should be immediately stopped and reversed if possible. If an occlusion balloon is available or rupture occurs during angioplasty, the balloon should be inflated to slow the rate of extravasation. If the patient is sufficiently stable, covered stent placement may offer definitive endovascular therapy.^{14,34,35} A conservative interventionalist may consider having an appropriately sized covered stent available when performing central venous reconstructions. In all cases of rupture, cardiothoracic surgical colleagues should be notified early in case open repair is needed.

Restoring cardiac output through rapid pericardiocentesis can be lifesaving. When performing central venous reconstruction, it is prudent to have the chest prepped and an ultrasound and pericardiocentesis tray available. The optimal entrance site when using ultrasound guidance is the "point at which the largest fluid collection is closest to the body surface and from which a straight needle trajectory avoids vital structures."³⁶ This site is often located in the fifth left intercostal space.³⁶ At this site, care should be taken to avoid the internal mammary artery, which is found between 0.5 and 1.5 cm (mean: 0.98 cm) from the lateral sternal border.³⁷ In well-trained hands, emergent ultrasound-guided pericardiocentesis is extremely safe. In a review of 92 emergent pericardiocenteses, Tsang and colleagues reported no deaths and only three major complications.³⁸ This procedure can also offer definitive therapy; 3 of the 16 reported cases of SVC rupture with cardiac tamponade required no further therapy after pericardiocentesis.^{10,11,13}

If ultrasound is unavailable and the patient demonstrates strong evidence of cardiac tamponade, a blind pericardiocentesis utilizing a subxiphoid approach can be considered, although it is associated with higher complication rates than ultrasound-guided pericardiocentesis.³⁹ With this approach, the sheath needle is placed in the angle between the xiphisternum and the left costal margin and advanced toward the left shoulder. The needle is directed at 30 to 45 degrees cranially until it is past the costal margin, where the angle is decreased to 15 to 30 degrees and advanced into the pericardium.⁴⁰

In unstable patients with rapid intrapericardial hemorrhage, central venous reinfusion of drained pericardial blood may be used as a temporizing measure until definitive therapy is available.

Conclusion

Endovascular stenting of malignant SVCS is a safe and effective therapy that can help maximize a patient's quality of life. However, the procedure carries a small risk of major complication, including SVC perforation and cardiac tamponade. Preprocedure planning can minimize the risk of severe complications. In the event of SVC perforation and cardiac tamponade, rapid diagnosis and treatment can be lifesaving.

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