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Percutaneous stenting as treatment for chylothorax from superior vena cava syndrome: A case report

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Abstract

Superior vena cava syndrome is a condition that results from obstruction of the superior vena cava, the etiology of which can be benign or malignant. The impaired venous return can cause facial and upper extremity swelling, dyspnea, and neurologic symptoms. Chylothorax is a rare complication of superior vena cava syndrome. We report a case of a 69-year-old male with end-stage renal disease, who developed both a chylothorax and a contralateral simple pleural effusion secondary to superior vena cava syndrome. He was successfully treated with percutaneous endovascular stenting.

Keywords

superior vena cava syndrome (SVC), chylothorax, percutaneous endovascular stenting

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Introduction

Superior vena cava (SVC) syndrome (SVCS) is caused by obstruction of the SVC and can present clinically with facial and neck edema, distended neck and chest veins, dyspnea, epiphora, altered mental status, and dizziness. Intravascular devices are the leading cause of benign SVSC.[1,2] Central venous catheters (CVC) increase the risk for thrombotic vascular occlusion, causing elevated venous hydrostatic pressure, and fluid accumulation in the pleural space.[3,4] We present a 69-year-old male with end-stage renal disease (ESRD) who developed a chylothorax and contralateral simple pleural effusion due to SVCS from long-term CVC use. In addition, we discuss the pathophysiology, diagnosis, and treatment of pleural effusions resulting from SVCS.

Case report

A 69-year-old male with ESRD on hemodialysis via left upper extremity arteriovenous fistula presented with chest wall pain after being transferred from an outside hospital. He had a long-standing history of multiple right-sided CVC placements for hemodialysis access. Otherwise, he had no known history of central venous thrombosis, infection, or malignancy. He had been treated for right chylothorax with chest tube placement 3 weeks prior to presentation. Initial computed tomography (CT) of the chest revealed new right-sided loculated effusions. Thoracentesis of the right side drained a milky yellow fluid. Pleural fluid analysis showed elevated triglycerides (791 mg/dL) and a pH of 8.00, with no evidence of malignancy on cytology, and no evidence of infection on fluid stain and culture. Fluid chylomicrons were not available from outside hospital records.

On the 15th day of admission, the patient was noted to have significant bilateral upper extremity pitting edema, concerning SVCS. He had no other symptoms, such as headaches or facial edema. The right chylothorax had been continuously drained throughout admission. CT angiography (CTA) showed occlusion of the right internal jugular, right brachiocephalic veins, and SVC (Figures 1 and 2). The patient also later developed a new left pleural effusion.

Interventional radiology was consulted for percutaneous stenting. Initial digital subtraction angiography (DSA)

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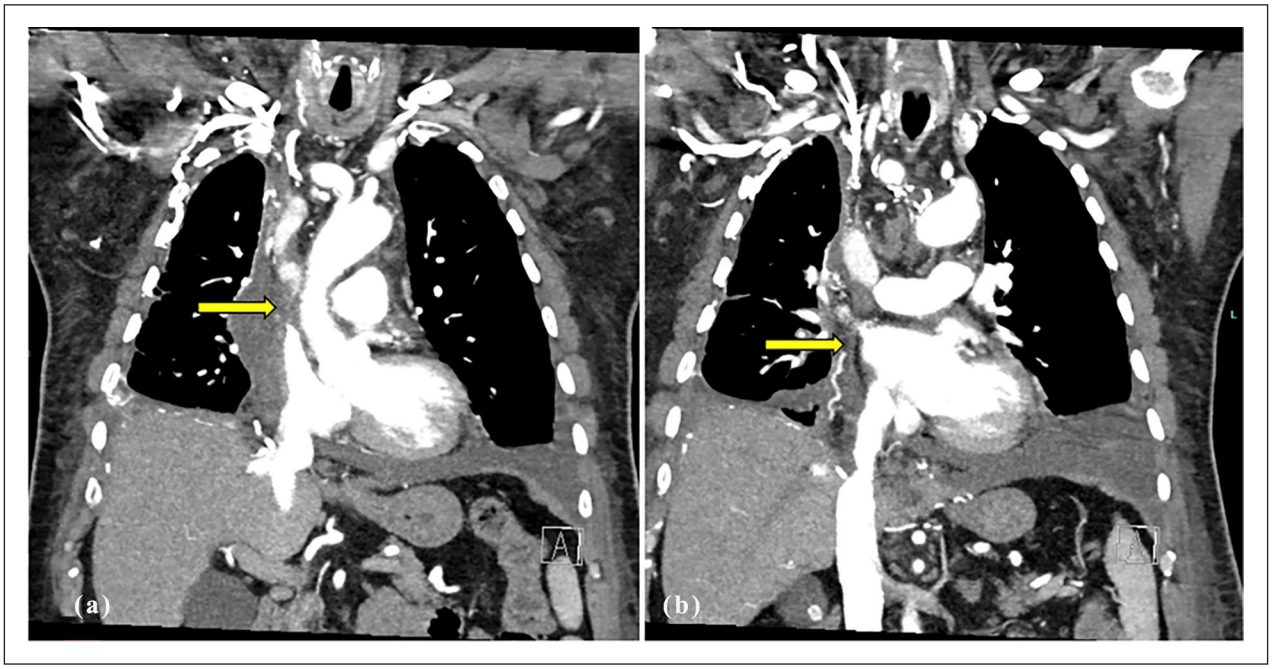

Figure 1. Coronal CT angiogram demonstrating occlusion of the superior vena cava (demonstrated by the arrows in (a, b)).
CT: computed tomography.

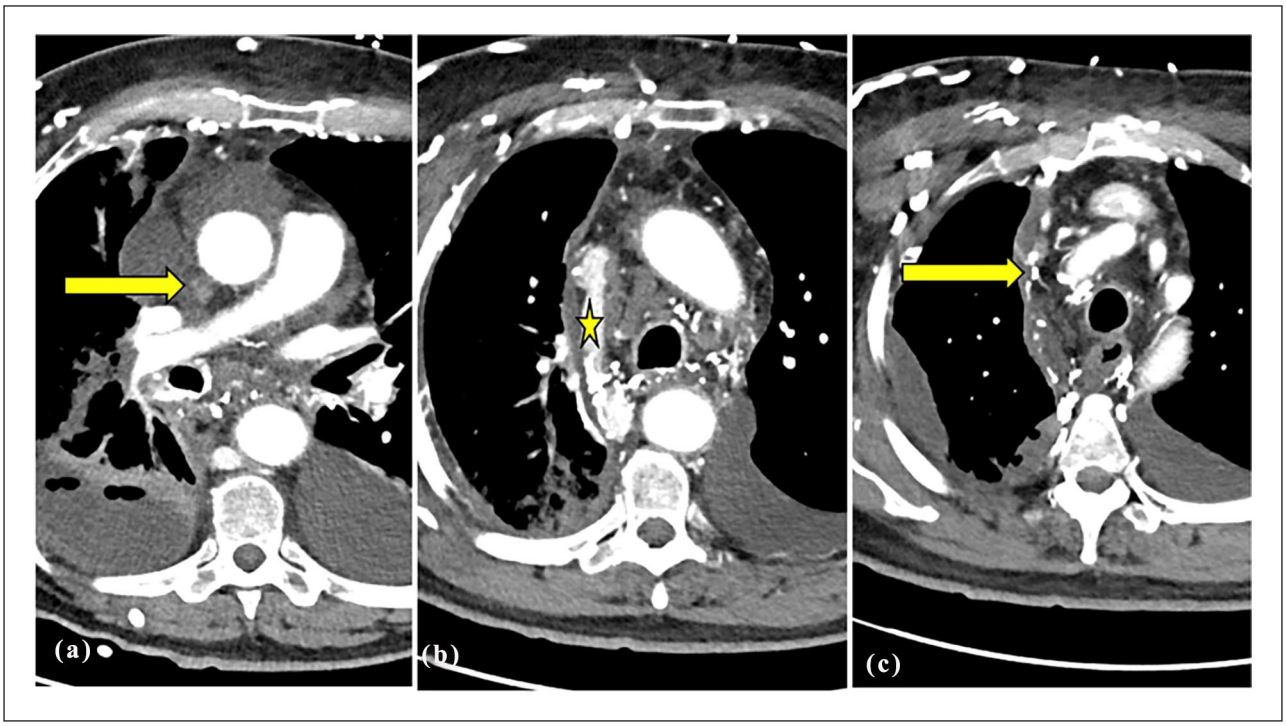

Figure 2. Axial CT angiogram images showing the occluded segments of the superior vena cava (demonstrated by the arrows in (a and c)). Most of the venous drainage occurs through the azygous vein (demonstrated by a star in (b)).
CT: computed tomography.

confirmed SVC occlusion both cranial and caudal to the junction of the azygos vein and left brachiocephalic vein (Figure 3(a)). In addition, the internal jugular vein was occluded, and the external jugular and subclavian veins were atrophic. There was collateral flow through the azygos and hemiazygos veins and multiple chest wall collaterals (Figure 3(b)).

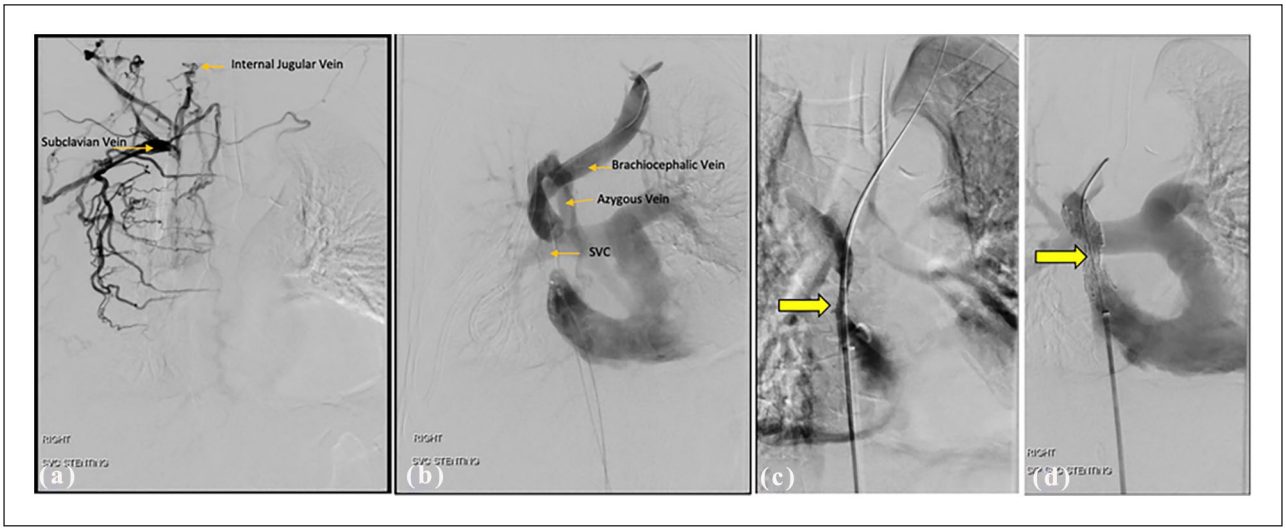

Figure 3. Digital subtraction angiogram shows an occluded internal jugular and superior vena cava, multiple chest wall collaterals, and an atretic right subclavian vein (a). Most of the venous drainage is through the left brachiocephalic vein and azygous vein (b). Post-intervention digital subtraction angiogram showed patency and preferential flow through the superior vena cava (demonstrated by the arrows in (c, d)) and no venous drainage through the azygous vein (d).

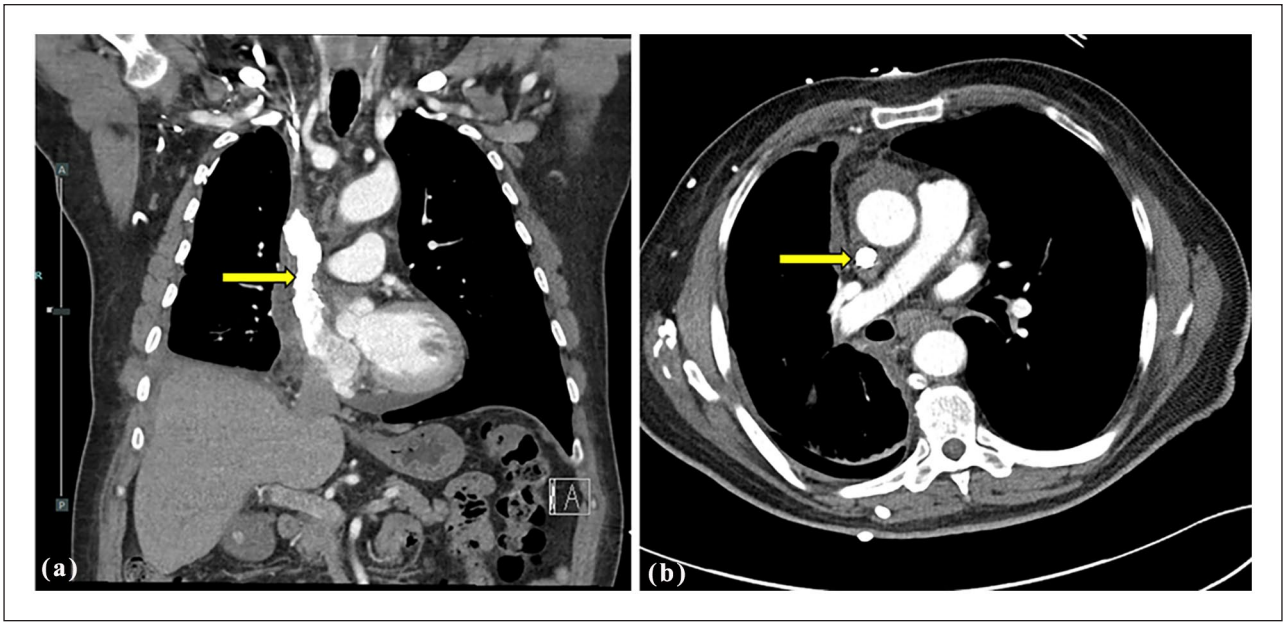

Figure 4. Post-intervention coronal and axial CT angiogram demonstrates a patent stent with significant interval improvement in bilateral pleural effusions (demonstrated by the arrows in (a, b)).
CT: computed tomography.

Following angioplasty of the caudal SVC occlusion with a 6×80 mm EverCross balloon catheter (Medtronic, Minneapolis, MN, USA), the SVC was stented with an 18×60 mm self-expanding Abre stent (Medtronic). Additional angioplasty of the stent was performed using an 8×80 mm EverCross balloon catheter (Medtronic). Post-intervention DSA showed patency of the SVC and no further opacification of the azygos vein (Figure 3(c) and (d)).

The patient then underwent ultrasound-guided left thoracentesis. Unlike the recurrent right-sided chylothorax, the left pleural effusion was "simple," with pleural fluid analysis revealing triglycerides of 28 mg/dL and absence of chylomicrons.

Post-interventional CTA showed patency of the SVC stent and significant improvement in bilateral effusions (Figure 4). The patient was discharged on octreotide, aspirin,

and bumetanide. Chart review up to 8 months later revealed that the patient remained asymptomatic and without pleural effusions on chest radiograph. He was subsequently lost to follow-up.

Discussion

Chylous effusions are characterized by the presence of chylomicrons, triglyceride levels >110 mg/dL, and a milky appearance of the pleural fluid.[5] The causes of chylothorax can be broadly categorized as traumatic or nontraumatic. Traumatic chylothorax accounts for about half of all cases and commonly has iatrogenic etiologies.[6] Nontraumatic causes include malignancies, particularly lymphomas, and predisposing factors involving inflammatory processes that affect lymphatic circulation.[4,6]

Regardless of the etiology, lipoprotein electrophoresis, which identifies chylomicrons in the pleural fluid, is the gold standard for diagnosis, as relying solely on triglyceride levels can be misleading; levels may fall below 110 mg/dL in malnourished patients or those with surgical trauma, risking a missed chylothorax.[7]

Pleural effusions are a well-documented complication of SVCS, though the reported frequency varies widely, ranging from 6% to 70%.[8] The variability in these estimates is mainly due to a lack of large-scale studies, relying instead on case reports and small case series. Even less is known about the frequency of chylothorax associated with SVCS, as pleural fluid analysis has been omitted in most cases, which is essential to distinguish chylous effusions from other types.

The pathophysiology of effusions due to SVCS depends on the etiology, that is, iatrogenic, malignant, and primary lymphatic dysfunction. The widely proposed mechanism in cases like our patients is increased central venous hydrostatic pressure. Obstruction of the SVC raises the intraluminal pressure in the venous system and pleural lymphatics, resulting in extravasation of plasma and chyle into the pleural space.[9–11] This mechanism has been attributed to isolated chylothorax and simple pleural effusion.[8] However, to our knowledge, the occurrence of simultaneous contralateral simple pleural effusion and chylothorax has yet to be documented in the literature.

While there are no definitive guidelines for treating chylothorax, initial management generally involves conservative measures, such as transitioning to a low-fat, medium-chain triglyceride-based diet or, in severe cases, implementing total parenteral nutrition.[5] Pharmacological therapies, like octreotide, can help reduce intestinal chyle production. Dietary restrictions were recommended following the patient's initial treatment for right chylothorax and again after stenting, but there is no documentation of the patient's adherence to these recommendations. Therefore, it is not possible to evaluate the impact of dietary changes on this patient's presentation. There are few reports of successful treatment with these measures alone because their efficacy is limited without addressing the underlying cause of lymphatic leakage.[12]

Endovascular stenting is the first-line treatment for SVCS and should remain the primary treatment for patients with associated pleural effusions.[13] In this case, venous obstruction was the primary driver of chyle leakage, as evidenced by the resolution of fluid collections following stent placement. Previous attempts to treat the effusions were unsuccessful because they did not address the underlying cause. Several case reports have described patients with CVC-induced SVCS who were successfully treated with stents alone or with stenting followed by dietary and pharmacological therapies.[10,14,15] Recurrent effusions following stenting are rare, with two longitudinal studies reporting no recurrence at 14- and 42-month follow-ups.[16,17]

Pleural effusions, especially chylous effusions, are presented in the literature as a rare complication of SVCS. This may be true depending on the etiology, but the prevalence of effusions from SVCS related to intravascular devices is likely higher than previously thought. In 2006, Rice et al. conducted the most extensive case series on SVCS, analyzing 78 patients.[1] In cases of benign SVCS, 70% were attributed to intravascular catheterization, pleural effusions were found in 58%, and two of the five analyzed samples were chylous. Given this data, physicians should maintain a high index of suspicion for SVCS in patients with indwelling intravascular devices and pleural effusions. Early identification of SVC stenosis can facilitate direct treatment of the effusions.

With over five million annual CVC placements in the United States, the prevalence of device-associated effusions is likely underestimated.[18] Awareness of this complication is critical, as timely recognition and appropriate treatment can significantly improve outcomes. Large-scale studies are needed to assess the frequency of pleural effusions associated with SVCS secondary to intravascular devices. Future studies should evaluate treatment outcomes and complications associated with stenting versus conservative management. These data will provide evidence for physicians to optimize treatment strategies and prevent delays in definitive management.

Conclusion

This case highlights the importance of recognizing SVCS as a potential cause of pleural effusions, particularly in patients with indwelling intravascular devices. Endovascular stenting effectively resolved the chylothorax and contralateral simple effusion by addressing the underlying venous obstruction. Physicians should be aware of this complication related to CVCs and proceed with diagnostic imaging to prevent treatment delays. Further research is needed to elucidate effusion frequency, optimize treatment strategies, and support the role of stenting as a first-line therapy.

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Ethical considerations

Our institution does not require ethical approval for reporting individual cases or case series.

Consent for publication

Written informed consent has been obtained from the patient for publication of the case report and accompanying images.

Author contributions

Arta Kasaeian: manuscript preparation, literature search, manuscript editing. Taylor Hoffman: manuscript preparation, manuscript editing, preparation of images. Mohammad Ghasemi Rad: manuscript editing. David Wynne: manuscript editing. David Léon: manuscript editing, preparation of images.

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