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MEDICINE

Acute Hemodynamic Compromise Following Superior Vena Cava Stent Placement: a Case Report

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Abstract

Symptomatic relief of superior vena cava (SVC) syndrome caused by tumor obstruction is achieved by placement of a percutaneous superior vena cava stent. Complications are rare. Even more uncommon is acute hemodynamic compromise from acute hemopericardium during placement of an SVC stent. Point of care ultrasound (POCUS) in the interventional radiology (IR) suite allows for rapid diagnosis and guidance of pericardial drainage and hemodynamic management.

Keywords Superior vena cava syndrome · Pericardial effusion · Hemodynamic compromise · Interventional radiology · Point of care ultrasound (POCUS) · Transesophageal echocardiography (TEE)

Introduction

Superior vena cava (SVC) syndrome occurs in 15,000 people in the USA every year [1] and was first described by William Hunter in 1757 [2]. SVC syndrome is caused by the gradual extrinsic compression or abrupt intrinsic blockage of the SVC. The prevalence of SVC syndrome has grown with the advent of increased implantable cardiac medical devices or long-term tunneled central intravascular access, which can result in acute thrombosis, culminating in SVC syndrome. [3, 4] Adenocarcinoma of the lung is probably the most common cause of SVC syndrome seen in malignant diseases [5]. Other conditions which may cause SVC syndrome are secondary to infection such as tuberculosis or syphilitic aortic aneurysm [6]. Radiation therapy, as a potential etiology in the development of SVC syndrome, has also been described [7]. The most common presentation is swelling of the face, neck, and upper extremities. Patients with malignancy may also present with dyspnea, cough, dilated chest veins, and weight loss. Life-threatening complications, such as cerebral and laryngeal edema, may occur [8].

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Case Presentation

A 78-year-old female with a past medical history of chronic obstructive pulmonary disease (COPD), coronary artery disease (CAD) (medically managed), atrial fibrillation, congestive heart failure (CHF), with baseline left ventricular ejection fraction (LVEF) 35–40% on recent transthoracic echocardiogram showing apical hypokinesis, normal left ventricular wall thickness, moderately dilated left ventricle and grade I diastolic dysfunction, dyskinetic intraventricular septum from a left bundle branch block (LBBB). The patient had stage IIIb squamous cell lung cancer treated with multiple rounds of FOLFOX (5-fluorouracil, leucovorin, oxaliplatin) chemotherapy developed right arm and bilateral breast swelling. The patient, however, never received

radiation or steroid therapy. Anasarca of the thorax resulted in the patient experiencing symptoms of chest tightness along with right arm swelling and pain. The patient was extremely uncomfortable and complained of increased chest tightness with inspiration. A CT scan of the chest with contrast demonstrated a hilar mass 5 × 4 cm compressing the superior vena cava with unchanged occlusion of the right upper lobe bronchus and complete collapse of the right upper lobe. The bronchus intermedius remained encased by the mass with increased luminal narrowing. Increased marked stenosis of the superior vena cava from compression of the adjacent lung mass was appreciated. The presence of multiple enlarging bilateral pulmonary nodules was consistent with progression of disease. No pleural or pericardial effusion was seen. The patient presented for placement of a SVC stent under monitored anesthesia care (MAC) with removal and placement of a new MediPort catheter through the SVC stent. On physical exam, the patient was uncomfortable in the supine position, complaining of increased chest tightness with inspiration from generalized chest swelling and persistent right arm pain. Vital signs (VS) were stable with BP 138/66, respiratory rate 24 bpm (breath per minute) with an oxygen saturation 95% on room air, and EKG findings of atrial fibrillation with a ventricular response in the 70's. MAC proceeded with intravenous (IV) midazolam 2 mg, fentanyl 25 mcg, and a propofol infusion of 50 mcg/kg/min. The Interventional Radiologist first obtained a venography from the right femoral and right internal jugular access site which demonstrated high-grade stenosis of a short segment of the SVC from extrinsic compression as well as a small burden of clots within the SVC. A through and through access was established by a 0.035-in. guidewire from the right common femoral vein to the right internal jugular vein. The right chest wall MediPort was removed. Initial attempt at advancing a 20 mm × 55 mm Wallstent through the SVC obstruction was not successful due to stiffness of obstruction. Therefore, angioplasty of the SVC obstruction was performed with a 14 mm × 40 mm XXL vascular balloon using manual insufflation with a syringe based on fluoroscopic monitoring of the shape of the waist of the balloon to 14 mm. This was followed by deployment of a 20 mm × 55 mm Wallstent, standard at our institution. The first stent immediately migrated distally toward the right atrium, and a second coaxial 20 mm × 55 mm Wallstent was deployed coaxially extending the entire stent assembly from the level of the innominate vein to the right atrium. Post dilation and post stent venography demonstrated good flow within the superior vena cava all the way to the right atrium. A new MediPort was placed using the indwelling right chest wall subcutaneous pocket. The patient then acutely developed hypotension. Initial treatment with 120 mcg intravenous phenylephrine produced no improvement in hemodynamics. Immediately available CT of the chest with contrast performed in the IR suite demonstrated a new small pericardial effusion probably hemopericardium. Endotracheal intubation was followed by an arterial line insertion into left radial artery and a norepinephrine infusion for blood pressure support. Transesophageal echocardiography (TEE) (video 1) was performed for initial diagnosis which helped with decision-making about pericardial drain insertion. TEE demonstrated a new pericardial effusion, mildly dilated and diffusely hypokinetic left ventricle, with septal dyskinesis secondary to LBBB, and a LVEF 30% by visual estimate. The SVC stent was seen extending into the right atrium. A MediPort within the SVC stent was appreciated. The right ventricular systolic function appeared decreased as well. Based on CT and echocardiographic findings, it was concluded that hypotension is caused by hemopericardium from SVC rupture at the time of balloon plasty. Transthoracic POCUS was performed for hemodynamic management. Acute accumulation of the fluid into the pericardial space compromised diastolic relaxation of the right ventricle which ultimately led to the increase in right atrial pressure and dilatation of the right atrium and IVC, nicely demonstrated in the subcostal view on transthoracic echocardiogram (videos 2 and 3). In addition, CT images of the chest with contrast prior to procedure (Figs. 1 and 2) and after the procedure (Fig. 3) nicely demonstrate new accumulation of sanguineous fluid in the pericardial space. A pericardial drain was placed with initial 150 ml sanguineous drainage resulting in improvement in hemodynamics. The patient was subsequently transferred to the ICU for further management and care, extubated the next day, and discharged to a regular floor after day 3. The pericardial drain was removed on day 7 and the patient was discharged home. The patient ultimately died from progression of malignant disease 3 months later.

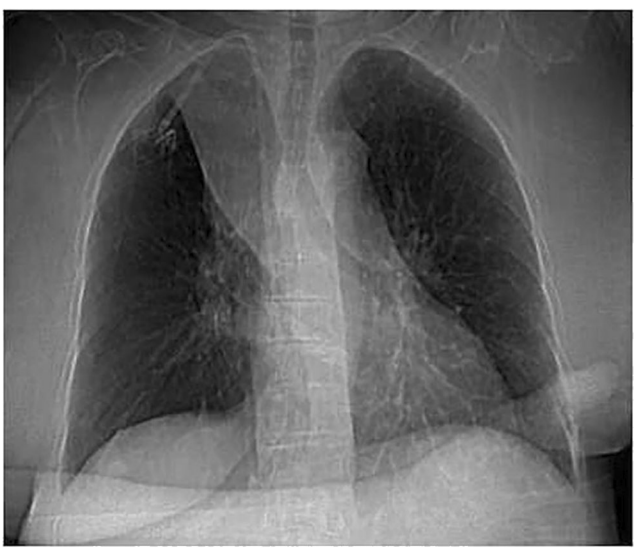

Fig. 1 Pre-procedure chest CT scan

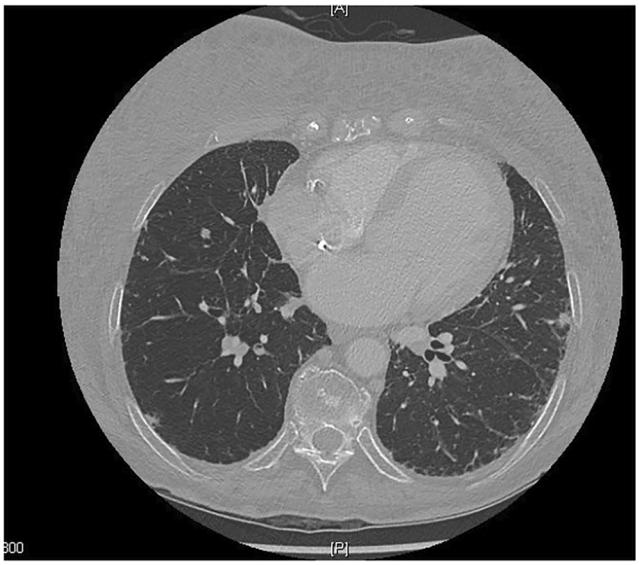

Fig. 2 Pre-procedure chest CT scan

Case Discussion

SVC syndrome is caused by the extrinsic compression of the SVC. In our patient, a large lung tumor was pressing on the SVC resulting in SVC syndrome. Treatments for SVC syndrome include surgery, radiation therapy, chemotherapy, endovascular surgery, and steroids. No randomized trials have compared the efficacy of these therapies. Systematic review of the literature has shown endovascular treatment with percutaneous stenting provides an effective treatment strategy in patients with SVC syndrome either caused by malignant or benign disease [9]. This procedure provides immediate symptomatic relief and improvement in quality of life in the advanced stages of the disease. Due to the ease of the procedure and low morbidity and mortality rate between 2.5 and 3.5%, endovascular superior vena cava stent placement is the mainstay of treatment in SVC syndrome. [10] Complications associated with stent placement are stent migration and venous thrombosis. Other life-threatening complications which an anesthesiologist should be aware of are acute hemopericardium from extravasation or rupture resulting in pericardial tamponade [11], acute right heart failure from fluid overload, pulmonary embolism, and intracerebral hemorrhage [12]. Recurrence of SVC syndrome has been reported at 11–15% and is associated with progression of disease and the use of stainless-steel stents. Among this group, a high degree of successful re-stenting occurs with reintervention [13]. Acute hemopericardium can present as sudden hemodynamic compromise. This presents a daunting challenge to the anesthesiologist [14]. Availability of transthoracic POCUS or transesophageal echocardiography (TEE) at this time can result in the immediate diagnosis of the sudden hemodynamic compromise and timely interpretation and intervention can be lifesaving.

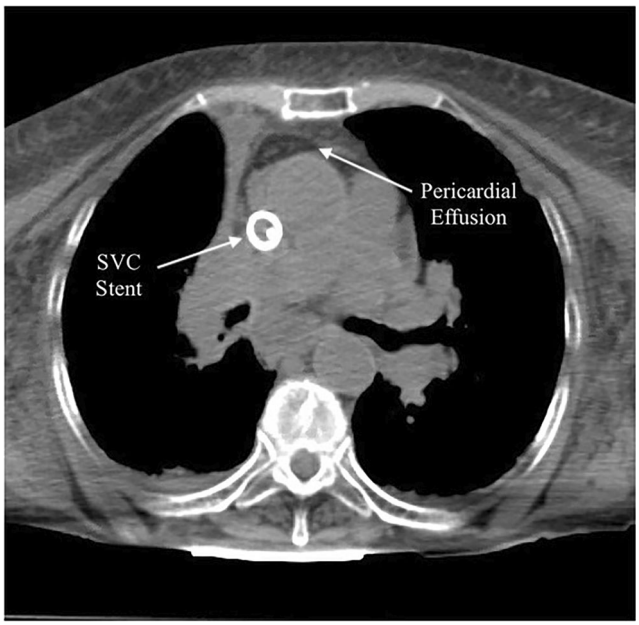

Fig. 3 Chest CT scan. Post SVC stent placement. New pericardial effusion

In our patient, acute hemodynamic compromise culminated from a combination of intrinsic patient factors and iatrogenic insults. Acute hemopericardium most likely occurred due to a rupture during balloon plasty of the SVC obstruction. It was likely a small rupture considering the late onset of hemodynamic instability and no significant extravasation was seen on the venogram. Migration of the first SVC stent protruding into the right atrium led to rapid ventricular response in the setting of pre-existing atrial fibrillation. Pre-existing LV systolic dysfunction and a history of heart failure classified the patient as elevated cardiac risk for the procedure.

Conclusion

Placement of a superior vena cava stent can improve a patient's quality of life and this procedure is widely accepted as a first-line therapy for SVC syndrome. Complications from endovascular stenting are rare; however, serious, potentially life-threatening complications such as acute hemopericardium do occur. Pericardiocentesis and prompt resuscitation by fluids are considered first-line management in hemodynamic compromise from acute hemopericardium as a complication of SVC stent placement. This case highlights the important role of the anesthesiologist trained in basic perioperative transesophageal echocardiography (TEE) and POCUS to diagnose this potentially life-threatening complication in a timely fashion, and to implement appropriate management strategy. POCUS was also used to further guide medical therapy and assess treatment. Basic TEE and POCUS training during anesthesia residency programs continues to gain momentum.

This case highlights the continuing need for development and standardization to ensure competency in POCUS and basic TEE for all anesthesiologists.

Authors' Participation Vaibhav Anand contributed to 50% writing.
Majid Maybody contributed to 30% writing, and to editing.
Gregory W Fischer contributed to editing.
Anahita Dabo-Trubelja contributed to 50% writing.

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Data Availability Chest CT scans and POCUS images are secured on the picture archiving and communication system (PACS).

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethics Approval Not applicable.

Consent to Participate Yes.

Consent for Publication The participant has consented to the submission of the case report to the journal.

Code Availability Not applicable.

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