# Acute Superior Vena Cava Syndrome in the Setting of an Intravascular Device

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# Acute Superior Vena Cava Syndrome in the Setting of an Intravascular Device

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## Introduction

Superior Vena Cava (SVC) syndrome is often associated with a malignancy that causes obstruction of the SVC resulting in decreased venous drainage from the head and neck. It most commonly presents with neck, facial, and upper extremity swelling, dyspnea or cough. The clinical effects of SVC syndrome range from mild aesthetic disturbances to life-threatening situations and hemodynamic instability. Increased venous pressure and edema may compromise the larynx and pharynx, leading to symptoms of stridor and dysphagia. Furthermore, confusion and coma may result from increased cerebral edema. Therefore prompt recognition and treatment is necessary.

## Case Report

A 34-year-old Caucasian female presented with dyspnea and a two-month history of facial swelling and discoloration, exacerbated when bending over. Past medical history was significant for methylenetetrahydrofolate reductase deficiency, history of provoked pulmonary embolism, and T2N0 left breast infiltrating ductal carcinoma. The patient was receiving chemotherapy through a right jugular venous catheter, inserted 4 months ago. On physical exam, the patient had facial cyanosis and edema extending to the anterior chest, tachycardia and decreased breath sounds. A chest wall medi-port was in place, terminating in the SVC. The remainder of her exam was within normal limits. A CT scan of the neck, chest abdomen and pelvis revealed acute thrombosis within the SVC and extensive collaterals throughout the pericardium, esophagus, and back musculature. Subsegmental bilateral pulmonary emboli were noted within the lower lobes. A superior venocavogram revealed totally occlusive thrombus in the mid to lower portion of the SVC and a small non-occlusive thrombus in the upper portion of the SVC. The right jugular venous port was present with the tip in the midportion of the SVC with the SVC occluded below the level of the catheter tip. The patient was subsequently started on a heparin drip and underwent catheter directed tPA. After 16.5 hours of tPA repeat superior venacavogram showed that the previous thrombus in SVC had only a small decrease in size with the development of several tiny channels of flow around the thrombus to the right atrium. The residual thrombus was macerated first with a 4 mm diameter angioplasty balloon followed by a 6 mm diameter angioplasty balloon to create multiple small flow channels. tPA thrombolysis was continued for an additional 45.5 hours for a total of 62 hours. Repeat superior venacavogram showed excellent flow in the SVC with minimal fibrin sheath arising from the catheter tip. The patient had no bleeding complications from tPA therapy and the patient's facial cyanosis and edema significantly improved. The venous catheter was removed 3 days later and patient was discharged home on enoxaparin.

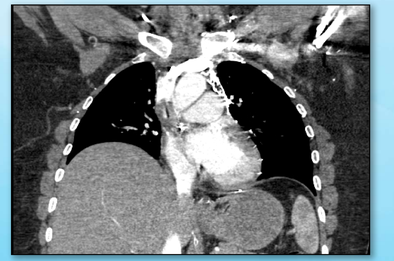

**Image 1.** Ct scan showing occluded SVC with collateral vessels.

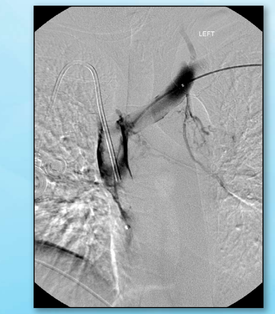


**Image 2.** Venogram showing occlusion of SVC below level of catheter tip.

## Discussion

- SVC syndrome is most commonly caused by obstruction of the SVC by invasion or extrinsic compression by malignancies such as non-small cell lung cancer, lymphoma, metastases or thymomas.
- A smaller portion of patients with SVC syndrome have non-malignant causes such as thrombosis, post-radiation fibrosis or an infectious etiology.
- Due to the increasing use of intravascular devices, such as central venous catheters and pacemaker/defibrillator leads, a larger portion of SVC syndromes now occur from benign causes which may account for up to 40% of SVC cases.[2]
- Treatment of SVC syndrome is accomplished by removing or attempting to decrease the size of the mass or thrombus causing the obstruction and is guided by the etiology and severity of symptoms on presentation.
- Options for treatment for SVC syndrome include: thrombolysis, endovascular stenting, balloon angioplasty, venous grafting or mechanical thrombectomy.
- Stent placement is a commonly used treatment and has been shown to be successful in both benign and malignant causes of SVC syndrome.[3]
- In thrombus induced SVC syndrome, thrombolysis prior to stent placement has been shown to be successful,[4,5] however, it has also shown to increase morbidity therefore making this combined strategy less desirable.[6]
- Stent placement also has its own risks such as: infection, pulmonary emboli, stent migration, hematoma formation, or SVC rupture.[7]
- In this case, due to the large extent of clot burden, the patient had a combination of catheter directed tPA and balloon angioplasty.
- After 62 hrs of tPA, infusion catheter check relieved excellent flow through the SVC therefore not requiring stent placement.
- Patient's symptoms significantly and quickly improved with the above treatment.

## Conclusion

- Due to the increasing use of intravascular devices, a larger portion of SVC syndromes now occur from benign causes.
- Percutaneous angioplasty can be used as an adjunct to tPA therapy to accelerate clot dissolution and successfully treat thrombus induced SVC syndrome, without the need for stent placement.

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