SVC Syndrome With Hepatic Pseudolesion

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INTRODUCTION

SVC syndrome is collection of clinical and radiographic symptoms commonly associated with lung malignancy. The compression of the superior vena cava leads to neovascularization and collateralization of several vascular beds. This process may lead to unusual radiographic findings which can lead the unfamiliar clinician astray. Recognition of these findings is therefore paramount to ensure timely diagnosis of the underlying malignancy and avoid unnecessary testing.

CASE

A 72-year-old female with a medical history significant for cigarette smoking and provoked PE presented to the emergency department complaining of 1 week of flulike symptoms which progressed to severe dyspnea at rest. She had recently been diagnosed with a lung mass and was scheduled for outpatient workup. She endorsed unintentional weight loss over the preceding months and, notably, progressive swallowing difficulty that began several days prior to presentation. In the emergency department, chest X-ray revealed a large mass within the anterior mediastinum with subtle nodular thickening of the right upper lobe and associated volume loss. There were also several small sub-centimeter nodules within the right upper lobe. (**Figure 1**) Given her history of tobacco use, age, and clinical prodrome, these findings were concerning for an aggressive pulmonary malignancy perhaps with a developing post-obstructive pneumonia. A follow-up CT angiography (CTA) of the chest was ordered to both rule out PE and better assess the extent of this mass. The CTA revealed a 4.5 cm mass in the right upper lobe confluent with a 14 cm bulky mediastinal lymphadenopathy which completely encased and occluded the superior vena cava. Focal enhancement of the liver was also appreciated in addition to extensive chest wall collateralizations. (**Figures 2,3**)

Subsequent abdominal imaging to investigate for potential metastatic spread revealed sub-centimeter cyst-like hypodense liver lesions, as well as hyperenhancement of the left lobe of the liver in segment IVa which subsequently normalized on delayed phase imaging (**Figure 4**). While the hyper-enhancing liver lesion was initially considered to be potentially suspicious for metastasis, the lesion location, washout on delayed-phase imaging, clinical prodrome, and presence of a known SVC obstruction together formed a more convincing picture for a hepatic pseudolesion due to collateralization of the superior vein of Sappey.

Figure 1. X-Ray Chest a/p view revealing large RUL mass.

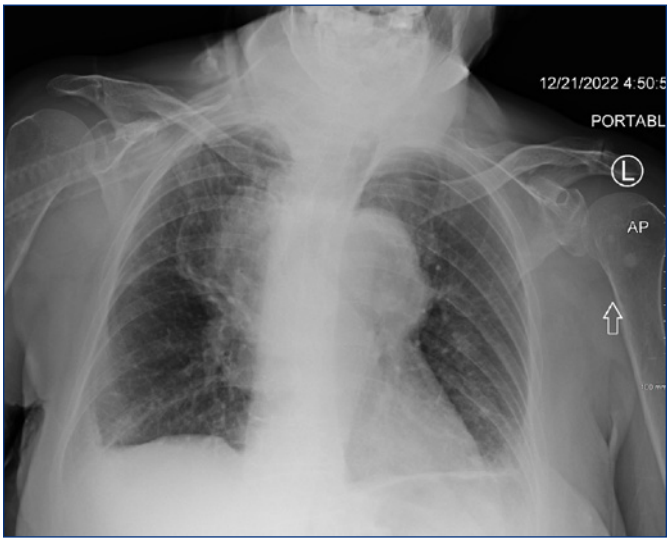


Figure 2. CTA Chest coronal view re-demonstrating large RUL mass with encasement of the SVC.

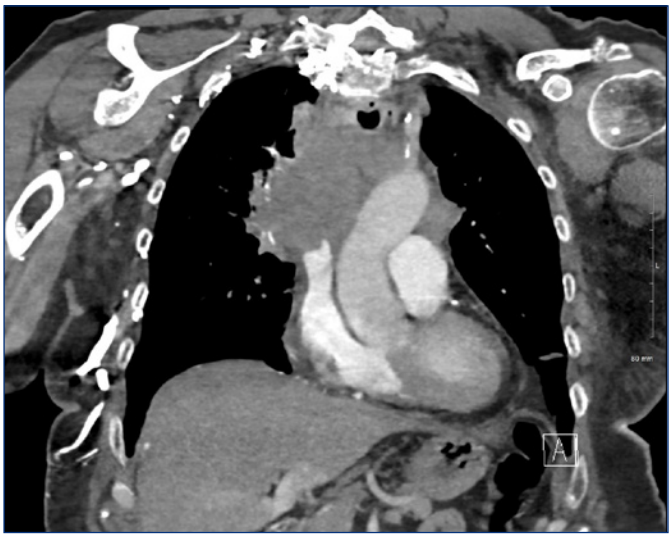


Figure 3. CTA Chest coronal view showing a large mass encasing the great vessels with mediastinal invasion and multiple contrast-enhanced thoracic wall collaterals as well as heterogenous nodular enhancement of the liver.

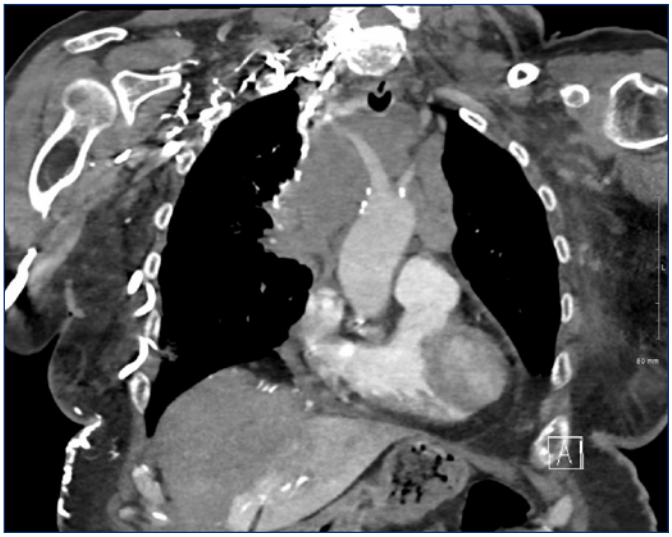
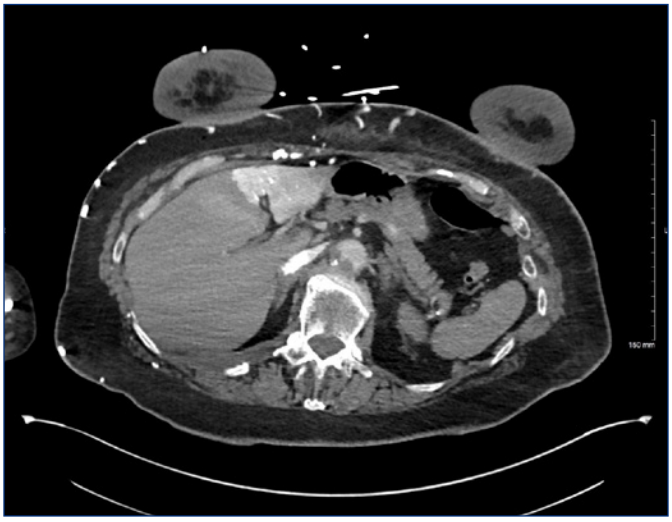


Figure 4. CT Abdomen axial view showing nodular hyperenhancement of hepatic segment IVa and engorgement of the surrounding hepatic and thoracic veins on the right.



DISCUSSION

SVC syndrome is a cluster of clinical symptoms associated with pathologic obstruction of venous return through the superior vena cava. These symptoms include upper extremity edema, JVD, and facial plethora. Many patients also present with cough and dyspnea or other complication of the underlying malignancy as this patient did. An estimated 80% of cases are caused by solid right-sided pulmonary malignancies, particularly small cell carcinomas, though rare cases associated with diffuse large B cell lymphoma have also been reported.[1] SVC syndrome tends to develop insidiously, as the SVC becomes gradually obstructed and venous collaterals form. These collaterals, particularly of the thoracic veins, are so frequently associated with SVC syndrome that their presence on chest CT carries a diagnostic sensitivity of 96%, and specificity of 92%, and they are essentially pathognomonic for SVC syndrome.[2] However, less commonly recognized is the collateralization of distant vessels such as the vein of Sappey which connects the internal thoracic vein to the hepatic portal system and eventually to the IVC. This vein normally arises within segment IVa of the liver and can produce a convincing lesion that enhances during contrast phase on CT. It can easily be mistaken for a primary liver or metastatic lesion.[3,4] Thus, a high degree of suspicion should be held for SVC syndrome in patients with known or suspected lung cancer and an arterial phase hyper-enhancing liver lesion to avoid unnecessary testing and diagnostic pitfalls.

References

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