Case Report

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Unveiling Superior Vena Cava Syndrome in a Case of Recurrent Pulmonary Tuberculosis: A Case Report

Internal Medicine Section

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ABSTRACT

Superior Vena Cava Syndrome (SVCS) is a rare but potentially life-threatening complication of lung cancer. It is most frequently caused by mechanical obstruction of the Superior Vena Cava (SVC) due to extraluminal compression by intrathoracic malignancies. Intraluminal obstruction of the SVC can occur due to thrombus formation, often associated with indwelling central venous catheters or pacemaker leads. The authors present a case report of an unusual presentation of SVCS in a 66-year-old male follow-up patient with recurrent pulmonary tuberculosis who was on anti-tuberculosis therapy and was later diagnosed with co-existent adenocarcinoma of the lung. The present case underscores the importance of considering malignancy as a potential cause of SVCS in patients with complex medical histories, including prior tuberculosis, and highlights the need to explore different aetiopathogenesis in venous thrombus formation, particularly its occurrence as a paraneoplastic phenomenon.

Keywords: Lung adenocarcinoma, Paraneoplastic syndrome, Rivaroxaban, Venous thrombosis

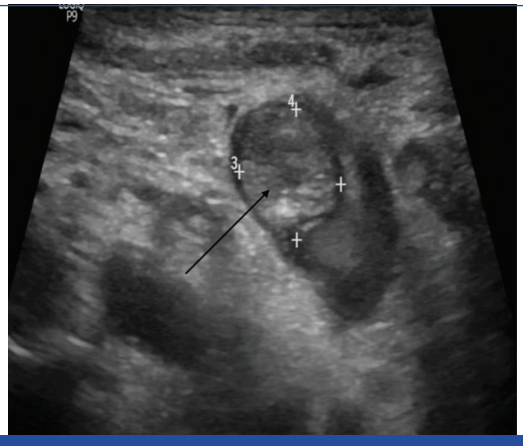
CASE REPORT

A 66-year-old male presented with diffuse swelling on the right side of the neck persisting for 20 days. The patient had a history of microbiologically confirmed recurrent pulmonary tuberculosis (rifampicin-sensitive) and had been undergoing retreatment with anti-tubercular therapy for the past month. He was previously diagnosed with pulmonary tuberculosis 10 years ago, treated with rifampicin, and declared cured following a six-month course.

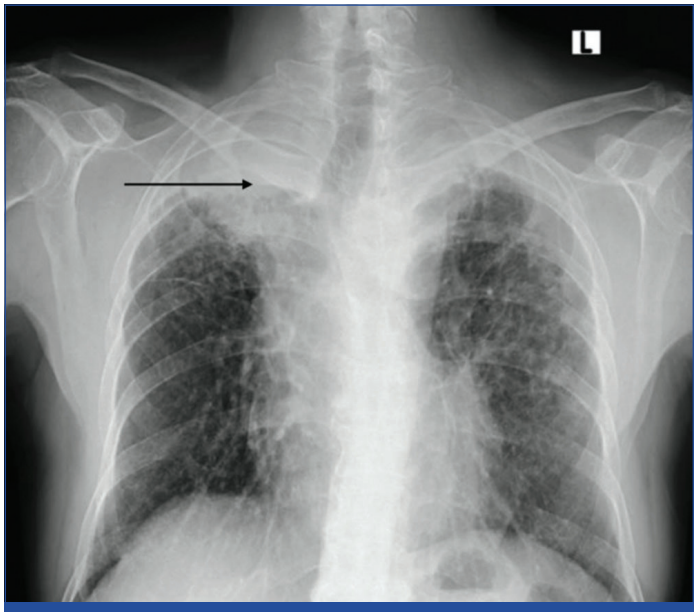
The patient was also a known case of Chronic Obstructive Pulmonary Disease (COPD) for seven years, managed with inhalers {Long-Acting Muscarinic Antagonist (LAMA) and a Long-Acting Beta2-Agonist (LABA)}. Additionally, he had a history of Type 2 Diabetes Mellitus and systemic hypertension for eight years and was a chronic smoker with a 20 pack-year history.

On examination, the swelling was non-tender and not warm, with visibly dilated veins over the neck and anterior chest wall; other systemic findings were within normal limits. The initial differential diagnoses considered included SVCS, possibly due to Tuberculosis (TB) lymphadenopathy or lung malignancy, jugular or subclavian vein thrombosis, lung cancer with nodal metastasis, and lymphoma.

Routine blood investigations revealed normal blood counts, with unremarkable renal and liver function tests. Doppler ultrasound [Table/Fig-1] of the neck demonstrated an echogenic thrombus measuring 1.5×1.3×1.2 cm within the right Internal Jugular Vein (IJV). Carotid Doppler identified bilateral intima-media thickening and a calcified atherosclerotic plaque at the right carotid bulb. Chest X-ray [Table/Fig-2] showed right upper zone opacity and emphysematous changes in both lung fields.



[Table/Fig-1]: Doppler ultrasound of the neck showing thrombus in right IJV (black arrow).

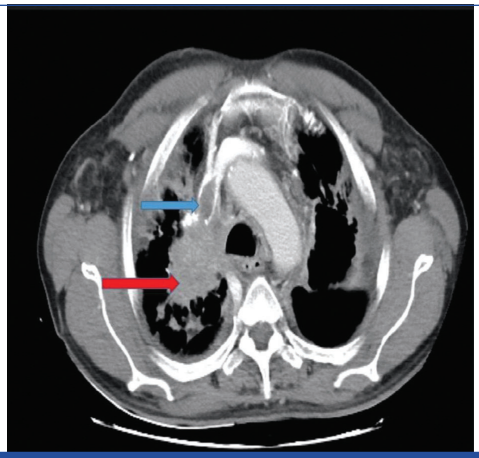


[Table/Fig-2]: Chest X-ray showing right upper zone opacity (black arrow) with old healed lesions and emphysematous changes in both lung fields.

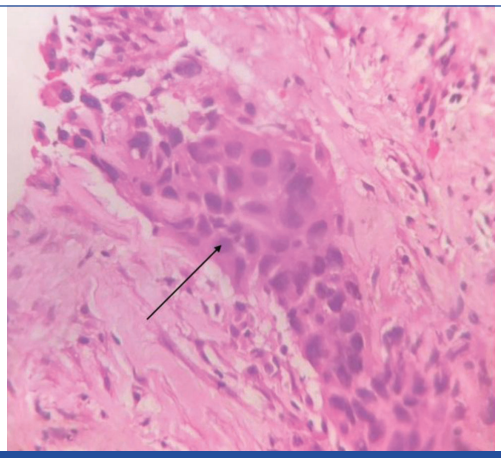
Because of the jugular vein thrombus, the patient was initiated on oral Rivaroxaban at 10 mg twice daily. A Contrast-Enhanced Computed Tomography (CECT) of the chest [Table/Fig-3] revealed a heterogeneously enhancing soft-tissue lesion measuring 5.2×4.7 cm in the apical segment of the right upper lobe, along with a thrombus at the junction of the brachiocephalic vein and the SVC measuring 2.5×1.0 cm, and another thrombus at the junction of the right IJV and the subclavian vein. No compressive mass was observed over the SVC; however, multiple collateral vessels were noted in the upper thorax.

A Computed Tomography (CT)-guided biopsy of the right upper lobe mass was performed, and histopathological examination [Table/

Fig-4] confirmed a diagnosis of poorly differentiated adenocarcinoma of the lung.



[Table/Fig-3]: CECT chest showing heterogeneously enhancing soft-tissue density lesion in apical segment of right upper lobe (red arrow) and a thrombus at the junction of brachiocephalic vein with SVC (blue arrow).



[Table/Fig-4]: Histopathological examination performed using Haematoxylin and Eosin (H&E) stain (X400) showing infiltrating tumour cells (black arrow) arranged in sheets with marked pleomorphism.

The PET-CT {Positron Emission Tomography (PET) and Computed Tomography (CT)} further revealed an irregular lesion measuring 5.7×4.2×7.0 cm in the apical segment of the right upper lobe, consistent with T4 primary malignancy, along with oedematous stranding in the mediastinum, para-aortic area, and bilateral axillary subcutaneous regions of the anterior chest wall, suggestive of sequelae from SVC obstruction. Metabolically active, discrete, sub-centimetre lymph nodes in the right paratracheal and subcarinal regions suggested Nodal Metastasis (N2). Thus, a final diagnosis of adenocarcinoma with SVC syndrome was made.

The patient was referred to a higher centre for palliative radiotherapy, where he underwent chemotherapy and radiotherapy for two months. Unfortunately, he defaulted on treatment due to poor tolerance and passed away five months after the diagnosis.

DISCUSSION

The co-existence of pulmonary tuberculosis and adenocarcinoma is rare, and the pathogenesis of this combination remains unclear. Several hypotheses have been postulated, including: coincidence without any apparent relationship; secondary infection of cancer due to immunosuppression, chemotherapy, or delayed treatment; carcinoma developing in old TB foci; and simultaneous development of both cancer and TB [1].

SVCS is a clinical condition resulting from severe obstruction or occlusion of the SVC, which can occur as a complication of both tuberculosis and lung malignancy, leading to significant morbidity and mortality [2].

In the present case, the patient was a follow-up case of recurrent pulmonary TB. Initially, SVC syndrome was suspected to be secondary to TB-related manifestations, such as mediastinal adenopathy or fibrosis. However, further evaluation revealed the presence of a coexistent malignancy.

The diagnosis of SVC syndrome is based on clinical presentation and imaging modalities, which include chest radiography, CECT scanning, duplex ultrasound, digital subtraction venography, and magnetic resonance venography. CECT imaging provides complete visualisation of the SVC, allowing localisation of the venous blockage, differentiation between thrombosis and extrinsic compression, and identification of collateral pathways [3].

In the present case, Doppler ultrasound and CECT imaging identified thrombi at two different locations, without showing any signs of external compression of the SVC, which is typically observed in malignancy-related SVC syndrome. Upon investigating the cause and pathogenesis of the thrombi, The authors found that the coagulation profile ({Prothrombin Time (PT), activated Partial Thromboplastin Time (aPTT), International Normalized Ratio (INR)} and other procoagulant markers were within normal limits. Therefore, the authors consider paraneoplastic syndrome to be the likely underlying cause of the observed venous thrombosis. This finding represents another unique and rare presentation that warrants special attention.

Paraneoplastic syndrome is a condition resulting from the presence of cancer in the body but, unlike mass effect, is not caused by the local presence of cancer cells. The syndrome may precede the diagnosis of malignancy, and its severity is unrelated to the size of the primary tumour. The most commonly described paraneoplastic syndromes are attributed to the secretion of hormones and functional peptides from the tumour or to immunological cross-reactions between normal host tissue and the tumour [4]. A similar case report of paraneoplastic thrombus causing SVC obstruction was published by Santra A et al., [5]. However, the recurrence of active pulmonary tuberculosis presenting as SVC syndrome initially raised the possibility of alternative differential diagnoses.

The incidence of venous thrombosis is approximately 40-100 cases per 1,000 person-years in patients with lung carcinoma, compared to an estimated 1-2 cases per 1,000 person-years in the general population. Patients with adenocarcinoma have a higher risk of venous thrombus than those with squamous cell lung carcinoma [4].

The mechanism of thrombus formation in malignancy involves several tumour-derived factors that disrupt normal coagulation pathways. Tumours can release Extracellular Vesicles (EVs) containing tissue factor, which activate the coagulation cascade and platelets, significantly increasing the likelihood of thrombus development. Additionally, they may shed EVs enriched with Podoplanin (PDPN), which further stimulates platelet activation and contributes to clot formation. Another pathway involves the release of Granulocyte-Colony Stimulating Factor (G-CSF), elevating circulating neutrophil counts and promoting the formation of Neutrophil Extracellular Traps (NETs), thereby enhancing the risk of thrombosis. Finally, tumours secrete Plasminogen Activator Inhibitor-1 (PAI-1), impairing plasmin generation and fibrinolysis, which inhibits the breakdown of clots and increases thrombotic risk [6].

Treatment options may include chemotherapy with or without Radiation Therapy (RT), surgical bypass, or Endovascular Therapies (ET) such as angioplasty, stenting, and catheter-based thrombus removal. Management is guided by the severity of symptoms and the identification of the underlying malignancy. A histologic diagnosis is crucial for developing a comprehensive, tumour- and stage-specific treatment plan.

CONCLUSION(S)

The present case highlights the rare but critical occurrence of SVCS in a patient with recurrent pulmonary tuberculosis, later diagnosed with adenocarcinoma of the lung. The development of SVCS in this patient was due to thrombotic obstruction rather than direct tumour compression, likely driven by a paraneoplastic syndrome. The case underscores the importance of considering malignancy as a potential cause of SVCS in patients with complex medical histories, including those with prior TB. Additionally, it serves as a reminder of the increased risk of venous thrombosis in cancer patients, particularly those with adenocarcinoma, and the need for prompt recognition and treatment of such complications.

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