

CASE REPORT

Mild Presentation of Superior Vena Cava Syndrome in a Patient with Non-Small Cell Lung Cancer: A Case Report

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ABSTRACT

Superior vena cava (SVC) syndrome can be fatal if not discovered early. Most SVC syndrome presentations are related to malignancies; lung cancer in particular. The presentations may vary, depending on severity of SVC obstruction. We report a case of mild SVC syndrome of a 70-year-old gentleman who presented with a one-month history of intermittent plethora and facial puffiness. Computed tomography scan of the chest revealed superior mediastinal mass which may represent matted lymph nodes obstructing SVC with bilateral pleural and pericardial effusion. Biopsy of the mass was done, and non-small cell lung carcinoma was diagnosed. Clinical presentation, differential diagnoses and issues relating to SVC syndrome were discussed with the emphasis on early recognition of mild symptoms and prompt management.

Keywords: Superior Vena Cava Syndrome, Lung neoplasms, Oedema

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INTRODUCTION

Superior vena cava (SVC) syndrome can cause significant morbidity and mortality. Malignancies account for 70% of its aetiology, and to date, non-small cell lung carcinoma is the most common cause of malignant SVC syndrome (1). Presentations of SVC syndrome can vary in severity, depending on the intensity of the degree of oedema, speed of collateral venous development and types of obstruction (2). Characteristically, the onset of symptoms in SVC syndrome occurs gradually over weeks to months (3).

CASE REPORT

Mr S is a 70-year-old gentleman with underlying dyslipidaemia. He has a history of ischaemic stroke in 2014 with no residual weakness. He was also diagnosed with pulmonary tuberculosis and completed treatment in 2014. He is an active smoker of twelve pack-years. He was apparently well until August 2019 when he suddenly experienced intermittent facial puffiness especially over bilateral eyelids and facial plethora for the past one month, which became prominent towards the afternoon (Fig. 1). There was no headache, cough, or dyspnoea. He had no leg swelling, fever, constitutional symptoms or any history of allergy. He went to a general practitioner's clinic twice and was misdiagnosed as acute heart failure

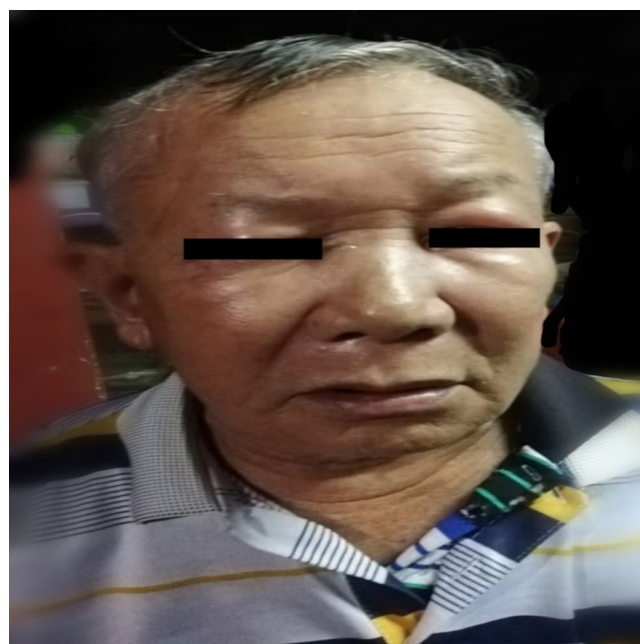


Fig. 1: Facial plethora and puffiness with mild periorbital oedema. Picture was taken by himself in the afternoon one day prior to the clinic visit in August 2019.

and treated with furosemide tablets. He was neither given a follow up nor a referral. His symptoms did not improve with the treatment, thus the family members decided to bring him to the district health clinic for a second opinion.

Upon examination, he was not in distress, and clinically he was pink. There was no obvious facial swelling or plethora as in Fig. 2. His conjunctiva was not injected.



Fig. 2: Picture of patient with no facial plethora or puffiness and resolved periorbital oedema. Picture was taken in the clinic office the next day

There was no finger clubbing. Small dilated veins were noted over the anterior chest wall. There was no cervical lymphadenopathy. He had a blood pressure of 133/87mmHg, pulse rate of 74 beats per minute and oxygen saturations of 99% under room air. Pemberton sign was negative. His respiratory rate was normal, with no stridor heard. Further respiratory examinations noted equally normal chest expansion and air entry on both sides, and there was no crepitation or rhonchi heard. There was no hepatomegaly or spinal tenderness.

Several investigations were done in the district health clinic including full blood count and erythrocyte sedimentation rate which were normal. Urinalysis revealed no proteinuria. Chest radiograph showed unchanged right upper zone fibrotic change with raised right hemi diaphragm. There was no mediastinal widening, obvious pleural effusion, or suspicious bone lesions. He was subsequently referred to a physician. A Computed tomography (CT) scan was done two weeks later which showed superior vena cava obstruction secondary to superior mediastinal mass which may represent matted lymph nodes, right lung nodules and bilateral pleural and pericardial effusion (Fig. 3 and 4). There was no liver metastasis or suspicious bone lesion. A bronchoscopy-guided biopsy was done following that, and the histopathology examination revealed minute foci of non-small cell carcinoma, favouring adenocarcinoma. He was admitted to the hospital and passed away one month after diagnosis. The cause of death was respiratory failure secondary to disseminated lung malignancy with superior vena cava obstruction (SVCO).

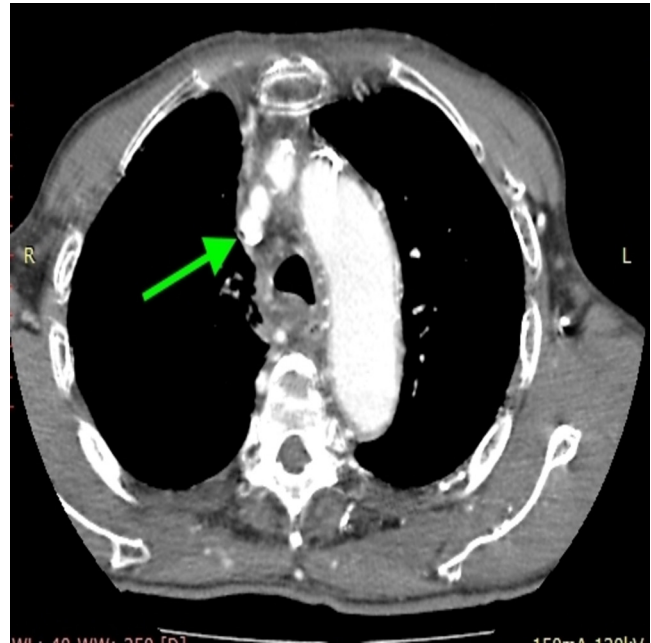


Fig. 3: Non-contrast CT axial image of patient shows patent superior vena cava just distal to the confluence and just proximal to the mass (arrow)

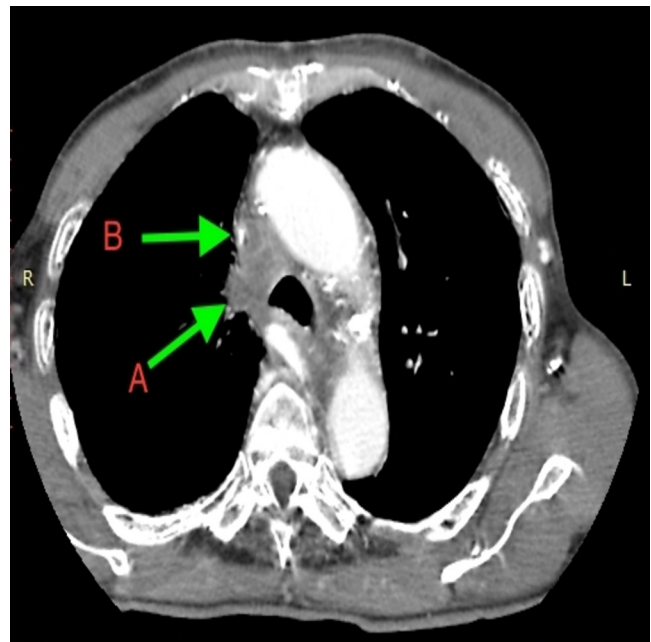


Fig. 4: Non-contrast CT axial image of patient demonstrates mediastinal mass (A) and compressed superior vena cava (B)

DISCUSSION

SVC syndrome can be categorized into four domains as suggested by Friedman, Quencer (1). The domains includes neurological (headache, blurry vision, papilledema and decrease level of consciousness), laryngopharyngeal (cough, tongue swelling, dyspnoea and stridor), facial (nasal stuffiness, conjunctival oedema, periorbital oedema, and plethora), and venous distension (dilated capillaries at upper extremities). All

the presentations can vary, ranging from mild to severe based on all four domains.

Early presentations of SVC obstruction can be misleading if other symptoms or examinations are inconclusive. However, diagnosis of SVC syndrome should not be missed at primary care settings. If not detected early, the increasing venous pressure will ultimately compromise the airway causing airway obstruction, cyanosis, hypoxia and around 10% of patients will develop cerebral oedema which resultant to death(4). Primary health care providers must recognize features of SVC syndrome, and include it as part of differential diagnoses; as it shares some clinical presentation of periorbital cellulitis, nephrotic syndrome or Cushing syndrome. Critical examinations and investigations may rule out differential diagnoses. Further investigation such as Computed tomography (CT) is the preferred imaging modality to diagnose underlying aetiology like lung carcinoma. Studies have shown that Computed tomography (CT) of lung improved early detection of lung cancer and reduced lung cancer specific mortality (5). In this case, referral to the secondary care for CT scan was a requisite. However, it was indeed a challenge for the primary care providers in this case as the referral centre was a tertiary hospital which was only reachable by a three-hour boat journey from the primary care clinic.

CONCLUSION

Presentations of superior vena cava (SVC) syndrome can be life-threatening, which require prompt diagnosis and treatment. Nevertheless, mild presentation of SVC syndrome can be missed, especially in a busy primary

care setting. Primary care providers need to be aware of early signs and symptoms of impending SVC syndrome and aggressive management and intervention should be timely to prevent further morbidity and mortality. Lung malignancy should be kept in mind; and the most common subtypes accounting for 85% of lung cancer cases is non-small cell lung cancers (NSCLC) (5).

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