

Superior Vena Cava Obstruction (SCVO)

Abstract:

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Case Report

A 68 year-old man was referred to the Emergency Department by his General Practitioner with a two week history of dyspnoea preceded by a two month history of weight loss. He also described recent onset of facial and neck swelling. He had a 40 pack-year smoking history, peripheral vascular disease and angina pectoris. Surgical history included a subtotal gastrectomy performed fifteen years previously for benign disease.

Clinical examination revealed a raised, non-pulsatile raised jugular venous pressure (JVP) with dilated veins across his anterior chest wall. On auscultation he had only a mild expiratory wheeze. Examination of his hands revealed no clubbing. Initial investigations revealed a normal full blood count, urea and electrolytes, calcium and liver function tests. Arterial blood gas on room air indicated a pH of 7.41, a pCO₂ of 6 kPa, and a pO₂ of 9.1 kPa. He had an abnormal chest radiograph and proceeded to CT thorax, shown in figures 1 and 2 respectively. CT demonstrated a mediastinal nodal mass circumferentially surrounding and markedly compressing the superior vena cava, causing superior vena cava obstruction. The patient underwent bronchoscopy and biopsy which confirmed a diagnosis of small cell lung cancer. Further staging defined this as limited stage disease. He was treated with chemotherapy to good effect which reduced the mass effect from the mediastinal nodal tissue on the superior vena cava.

Discussion

In superior vena cava obstruction (SVCO), blood must return to the right atrium via collateral circulation. The ability of the collateral circulation to accommodate this increased blood flow is dependant on the duration and degree of SVCO¹. There is typically increased venous pressure in the upper body with facial oedema and plethora, dilated veins over the neck and trunk and occasionally in the upper limbs. Patients may describe headache, dyspnoea, dysphagia or hoarseness. In severe cases, albeit rarely, coma may ensue. Causes of SVCO can be intramural, intraluminal and extrinsic. The most common cause is intrathoracic malignancy. This is most often due to non-small cell lung cancer, but SVC obstruction is also often seen with small cell lung cancer, lymphoma, metastatic disease and with primary mediastinal masses². Benign causes include venous thrombosis, TB infection, sarcoidosis and silicosis. It was historically associated with syphilitic aortic aneurysms³. Iatrogenic causes include prior radiation to the mediastinum, indwelling catheters and pacemaker leads, particularly if there have been multiple insertions.

Management of the patient with SVCO depends on the severity of symptoms and the underlying cause⁴. For small cell lung cancer and lymphoma the response to chemotherapy is typically dramatic. Radiotherapy may also be employed with good effect. In other cases, interventional radiology guided SVC stenting or venoplasty may need to be considered^{5,6} and thrombolysis in the case of SVC thrombosis. These options may also require consideration in severe cases pending a tissue diagnosis. Surgical options include venous bypass grafting. Symptom resolution following successful treatment is typically rapid. Survival depends on the course of the underlying disease and correlates with tumour histology in those cases with a malignant aetiology.

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