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Case report of a Patient with SVC syndrome in the course of pulmonary hilar and mediastinal neoplasia

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Abstract

Superior vena cava syndrome (SVCS) is a serious clinical condition caused by obstruction of the superior vena cava, usually due to malignancies. We present a case of a 67-year-old woman with SVCS secondary to a tumour in the right

pulmonary hilum and mediastinum. The patient exhibited facial and neck swelling, venous distention, and dyspnoea. Computed tomography confirmed a large polycystic mass causing complete occlusion of the superior vena cava and extensive venous thrombosis. The patient was treated with anticoagulation therapy and referred for oncological assessment. This case underscores the importance of prompt diagnosis and imaging in guiding SVCS management.

Keywords: "superior vena cava syndrome," "pulmonary neoplasm," "venous thrombosis," "mediastinal tumour," "case report"

Introduction

Superior vena cava syndrome (SVCS) is a set of symptoms caused by compression of a mass, tumour invasion and/or thrombosis of the superior vena cava (SVC), resulting in obstructed blood flow from the SVC into the right atrium. SVCS was first described by W. Hunter in 1757 in a patient with aortic aneurysm in the course of syphilis [1]. In the past, it was aortic aneurysm in a syphilitic patient and lymphadenopathy in the course of tuberculosis that were the most common causes of SVCS [2]. Currently, the most common causes of superior vena cava syndrome are malignant lung tumours [2].

We present the case of a 67-year-old woman with superior vena cava syndrome caused by a tumour in the right pulmonary hilum and mediastinum.

Case description

A 67-year-old woman was referred to the oncology outpatient clinic because of cough and dyspnoea that had persisted for several months, as well as

mediastinal shadow dilatation described on chest X-ray. Physical examination revealed swelling of the face and neck and dilatation of the superficial veins on the chest. An ultrasound examination was performed, which revealed jugular venous thrombosis and enlarged jugular nodes. It was followed by a two-phase CT scan of the neck and thorax, in which a polycystic nodule with approximate dimensions in the transverse plane of 100 mm x 60 mm and approximately 85 mm in the CC dimension was revealed in the upper, lower and partially posterior mediastinum and within the right lung cavity, undergoing heterogeneous enhancement after the administration of intravenous contrast medium (fig. 1).

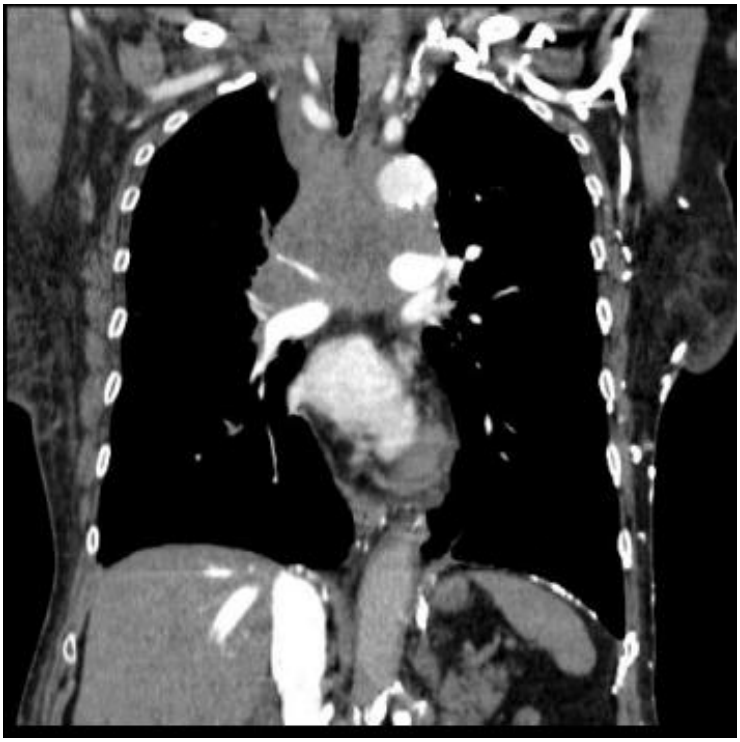
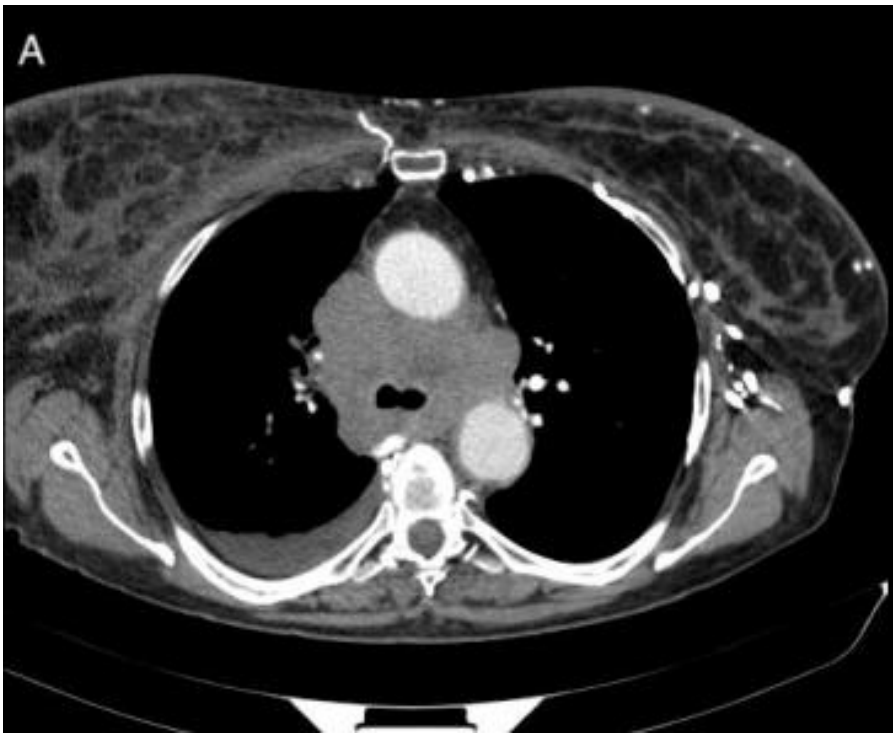


Fig. 1. Computed tomography of the chest. MPR reconstruction, coronal view.

The tumour mass constricted the trachea, the lumen of the right pulmonary artery in its middle segment, the right superior pulmonary vein and caused occlusion of the

SCV along its entire course (fig.2 A, B).



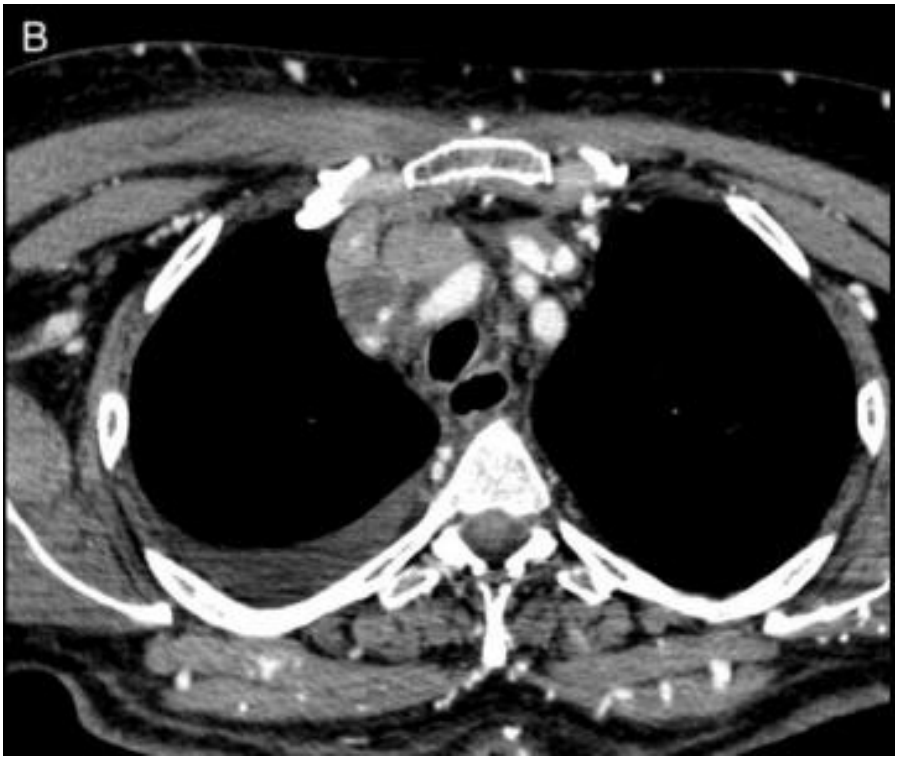


Fig. 2. A, B. Computed tomography of the chest. Axial views.

The tumour was adjacent to the thoracic aorta, raising suspicion of infiltration of its wall. A fluid sheath approximately 25-30 mm thick was visualised in the right pleural cavity, and polycystic areas of atelectasis in SP5. Additionally, the CT scan showed thrombosis (fig.3) in the:

- right sigmoid sinus,

- right internal jugular vein opacity,

- dilated right internal jugular vein,

- dilated right common jugular vein,

- dilated left common jugular vein with thrombosis from the level of the superior thyroid vein drainage of the left thyroid lobe,

- left subclavian vein at a distance of approx. 50 mm before its junction with the left brachiocephalic vein,

- left brachiocephalic vein,

- right subclavian vein at a distance of approx. 30 mm before its junction with the right brachiocephalic vein,
- superior vena cava.

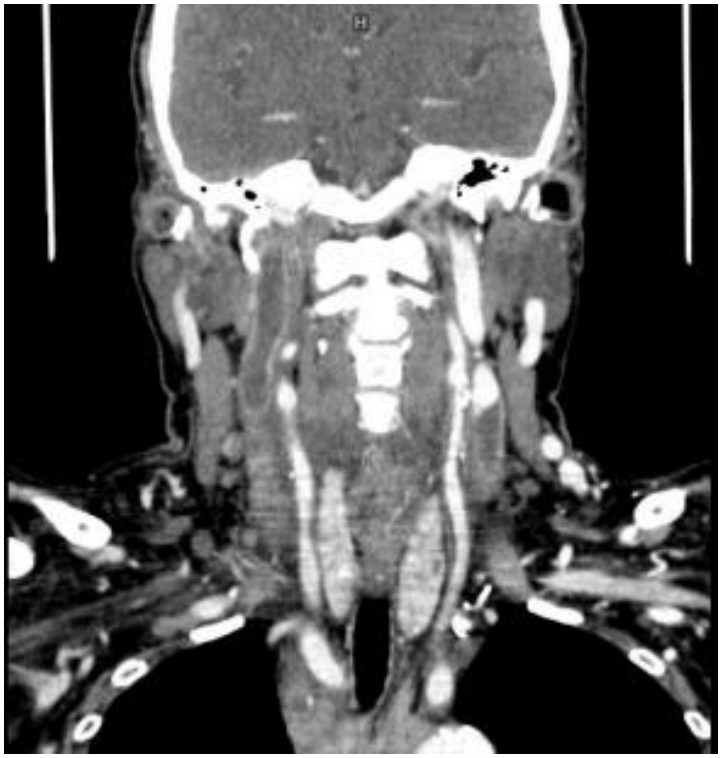
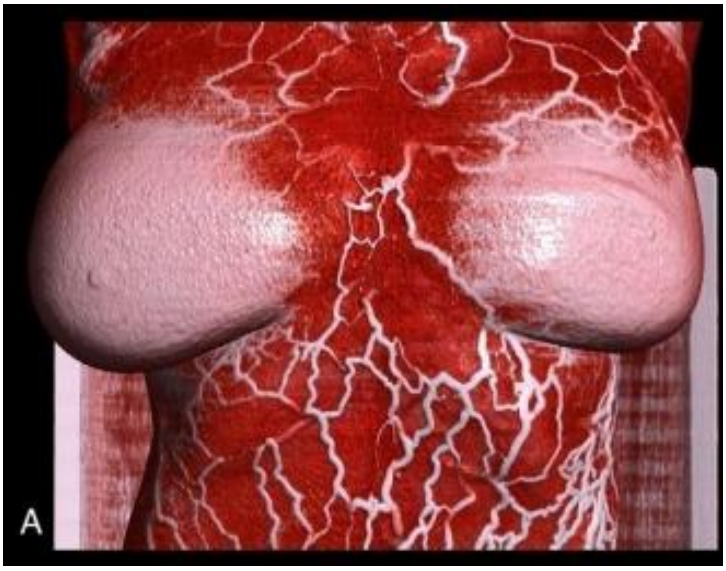


Fig. 3. Computed tomography of the neck. MPR reconstruction, coronal view.

At the craniofacial and neck level, mainly in the basin of the external jugular veins, to a greater extent the left external jugular vein, the scan revealed dilated subcutaneous venous vessels of the peripheral circulation and an extensive network of venous vessels within the subcutaneous soft tissues of the lower neck in the basin of the left and right subclavian veins.

The subcutaneous soft tissues of the chest showed an extensive network of venous collateral circulation with predominant lesions in the left anterior wall, left posterior wall and left-lateral wall (fig. 4 A, B, C).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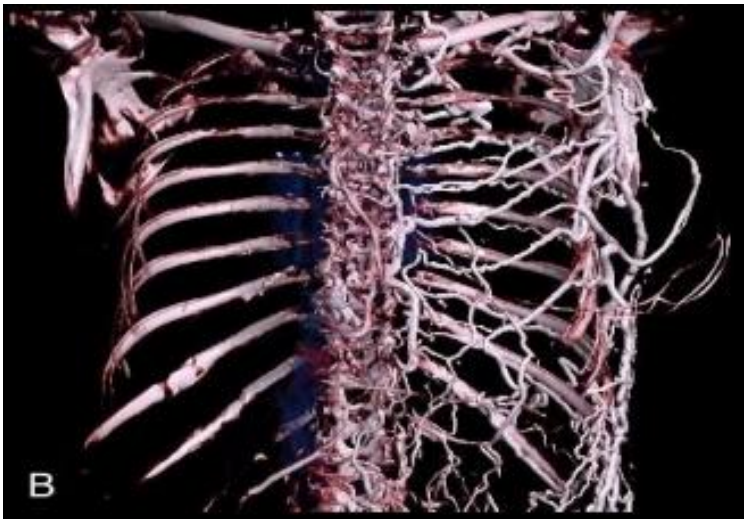
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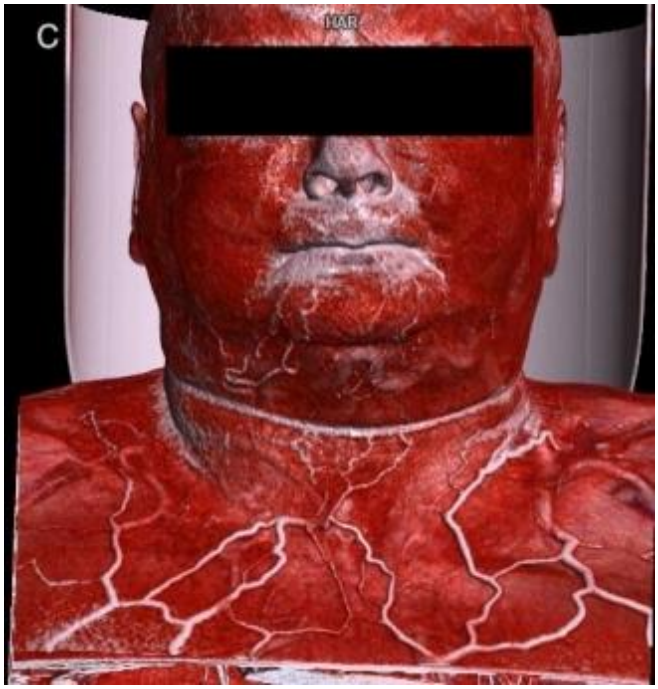

Fig. 4. A, B, C Computed tomography of the neck and chest. VRT reconstructions.

In addition, dilatation of the intercostal veins of the left chest wall, dilatation of the paravertebral veins and obstruction of the azygos vein in its upper segment were visualised. Also visible were thickened vocal folds and a shallowed glenoidal keratoconus, as well as enlarged neck lymph nodes, including the metastatic group IVb lymph node on the right side.

The radiological picture in correlation with the clinical symptoms indicated neoplasia of the right lung hilum and mediastinum with features of superior vena cava syndrome. The patient's clinical condition allowed her to function relatively normally. Anticoagulant treatment was administered due to thrombosis. The patient was referred to a specialist oncology centre for further diagnosis and treatment.

Discussion

Malignancies contribute to SVCS in approximately 65-85% of cases, the most common being lung cancer and lymphoma. Around 75% of SVCS cases are due to lung cancer (usually non-small cell). For anatomical reasons, right-

sided lung cancer has a greater tendency to cause SVCS than left-sided. 15% of SVCS cases are caused by lymphomas, usually non-Hodgkin, rarely – Hodgkin lymphomas. Other rare neoplastic causes of SVCS include thymoma, thyroid cancer, oesophageal cancer, breast cancer and germ cell tumours [2,3,4,5]. Non-cancer causes of SVCS (15-40% of cases) include thoracic aortic aneurysm, chronic mediastinitis or SVC thrombosis related to central venous catheterisation or pacemaker implantation [2,3,4,5]. SVCS is usually seen in the older age group: in the study by Armstrong et al. the mean age of the onset of superior vena cava syndrome was approximately 55 years [6].

The clinical picture of SVCS can range from asymptomatic to immediately life threatening forms. It varies depending on the anatomical level, degree of SVC obstruction and degree of development of the peripheral circulation. The clinical presentation can be acute, developing over a few days, subacute or chronic. In acute cases, the peripheral circulation is less abundantly developed, leading to more severe symptoms than in subacute and chronic cases. Due to that, in slow-growing tumours, long-term, asymptomatic SVC obstruction may occur [2,5]. The diagnosis of SVCS is mainly based on clinical symptoms [2], the most common of which are swelling of the face and neck [2,7]. Other symptoms include bruising of the face and neck, coughing, conjunctival congestion, swelling of the upper limbs, headache, dizziness, visual disturbances, difficulty swallowing, hoarseness, stridor. Usually the symptoms are exacerbated when lying down. In addition, symptoms resulting from reduced cardiac filling, such as hypotension and syncope, may occur [2, 3, 7, 8]. In severe, life-threatening cases, oedema can narrow the lumen of the nasal passages and larynx, leading to a life-threatening condition. Cerebral oedema may also occur, leading to cerebral ischaemia [4,5,7]. The type, severity and temporal development of symptoms are important in determining the need for urgent intervention. To this end, Yu et al. proposed a grading system to distinguish between severe and non-life-threatening clinical conditions [7]. Imaging diagnosis plays a key role in the diagnosis of SVCS because it can distinguish mediastinal tumours and/or thrombi in the lumen of the venous vessels as causes of SVCS, enabling the best treatment option to be selected [4].

The method of choice for SVCS imaging is computed tomography with the use of intravenous contrast agent. It is characterised by high sensitivity and specificity and can reliably identify the underlying pathology [2]. SVCS therapy has two pillars: relieving symptoms associated with SVC obstruction and treating the underlying disease [4]. The treatment options for SVC obstruction, depending on the cause, tumour grade and histopathology, include radiotherapy, chemotherapy, open surgery and intravenous recanalisation. In some cases, glucocorticosteroids may be an effective adjunctive treatment. Endovascular stents may be considered after the initial diagnosis in severe SVCS for rapid symptom relief [2,4,5,9]. The prognosis of SVCS caused by malignant tumours is usually poor – life expectancy is calculated in months and depends primarily on the type of cancer, its stage and therapeutic options. However, there is no significant difference in prognosis between cancer patients who develop SVCS and those who do not [2,10].

Summary

The cause of SVCS in the case described was a right hilar and mediastinal tumour causing occlusion of the SVC. In addition, there was an overlapping extensive thrombosis secondary to the occlusion of the SVC lumen by the compressing tumour. The clinical picture of SVCS depends on the anatomical level, degree of obstruction of the superior vena cava and degree of development of collateral circulation. In the case described, it is likely that the relatively slow growth of the tumour allowed for the development of a well-developed collateral circulation that enabled the patient to function relatively normally so that her condition did not require emergency intervention. The subjective and physical symptoms, in correlation with imaging examinations, allowed the diagnosis to be made and the patient to be referred to a specialist oncology centre for further diagnosis and treatment.

Disclosure

Authors do not report any disclosures.

Authors'contributions

Conceptualization, supervision and project administration, methodology, software, validation, formal analysis, investigation, resources, writing original draft preparation, writing review editing and visualization: Paweł Święch, Przemysław Jaźwiec, Andrzej Bazan

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Data availability statement

The data presented in this study is available upon request from the corresponding author.

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Conflict of Interest Statement

The authors declare no conflict of interest.

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