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CASE IMAGE

Unveiling the culprit: Exploring malignant thrombotic superior vena cava obstruction

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Key Clinical Message

This case image describes the complex proposed etiologies of a case of superior vena cava syndrome. Hence, different diagnostic and therapeutic modalities are needed in a multidisciplinary team approach.

KEYWORDS

catheter-induced SVC syndrome, deep vein thrombosis, lung cancer, superior vena cava obstruction, SVC syndrome

1 | CASE PRESENTATION

A 64-year-old woman recently diagnosed with Stage IVB Adenocarcinoma of the right upper lung with brain metastases planned for chemotherapy presented with a one-week history of progressive swelling in her face, neck, arms, and breasts. On examination, she had a facial plethora that worsened with arm elevation with pitting edema of the face and upper torso and prominent veins on her face, chest, and breasts (Figure 1A) with enlarged lymph nodes at post-auricular, cervical, and supraclavicular regions. Chest X-ray showed a right central lung mass with concomitant atelectasis and right pleural effusion (Figure 1B). Contrast-enhanced computed tomographic (CT) imaging of her chest revealed a right endobronchial mass with lymphadenopathy causing superior vena cava (SVC) compression and extensive thrombi in her SVC, left internal jugular, and innominate veins (Figure 1C,D). She was initially treated with intravenous heparin infusion and started the first cycle of cisplatin/remetrexel. Her symptoms continued to worsen due to the extensive clot burden suspected from the chemo-port catheter and endobronchial mass. Following multidisciplinary discussion, endovascular therapy was deemed necessary for prompt symptom resolution. Subsequently, the chemo-port catheter was removed followed by mechanical aspiration thrombectomy using 8F Penumbra Indigo System and Ultraverse balloon dilatation, resulting in satisfactory results despite residual stenosis from external compression. The patient reported slight symptom improvement, leading to discharge with therapeutic enoxaparin and a new left femoral tunnel catheter for subsequent chemotherapy cycles, alongside close oncology monitoring for additional Pembrolizumab therapy.

Superior vena cava (SVC) syndrome is caused by the severe obstruction or occlusion of the SVC, manifesting with symptoms such as cough, dyspnea, orthopnea, facial edema, upper extremity swelling, and dilation of chest wall and neck veins. The severity of symptoms varies based on the obstruction's onset, severity, and location, alongside collateral vein development. Diagnosis confirmation requires imaging modalities, including chest radiography (CXR), contrast-enhanced computed tomography (CECT), duplex ultrasound, conventional venography, and magnetic resonance venography.[1] A yearly incidence of up to 15,000 patients was reported in the United States, with a rising trend observed from 2010 to 2018, notably due to catheter or lead-associated cases. Around 70% of SVCS cases are attributed to

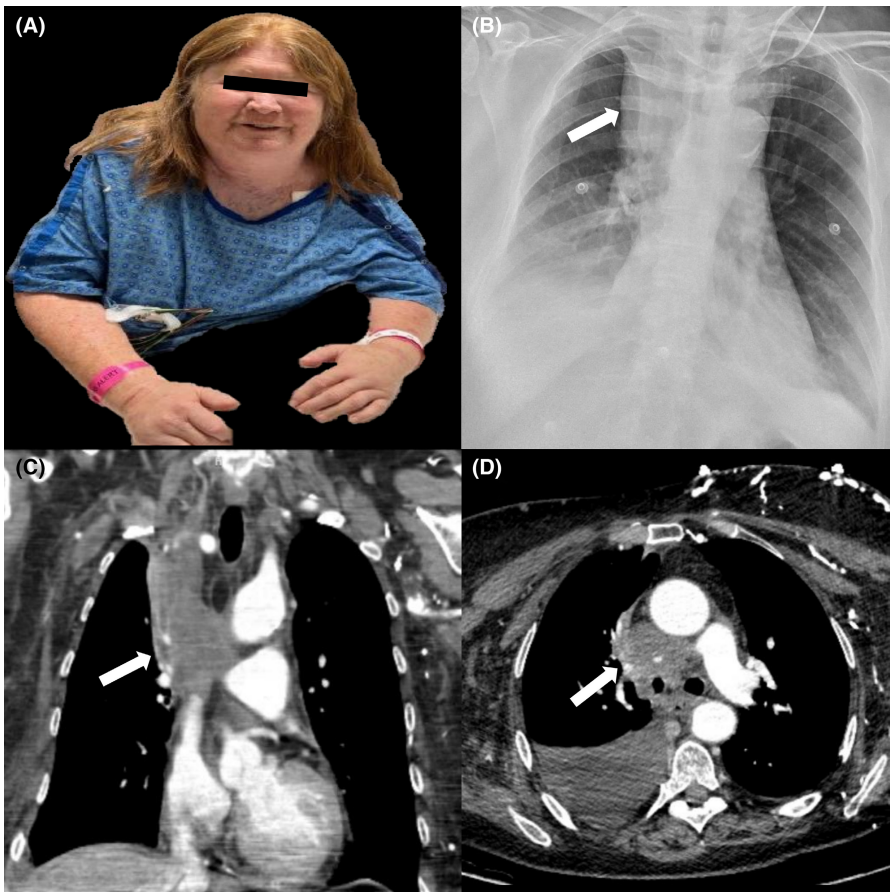

FIGURE 1 (A) Facial plethora, pitting edema of the face with prominent telangiectasias on her neck. (B) Chest X-ray showing a right-sided central lung mass with concomitant right upper atelectasis (arrow) and right pleural effusion. (C, D) Coronal and axial CT chest with IV contrast revealed a right upper lobar central lung mass with a markedly compressed superior vena cava (SVC) with intramural thrombus consistent with SVC syndrome (Arrow).

malignancy, with non-small cell lung cancer being responsible for half of them, while benign causes like device-related SVC syndrome account for 30%.[2] Here, the authors demonstrated a patient with malignancy-related SVC syndrome with an extensive clot burden from several causes, including direct compression of SVC, direct invasion, or thrombosis from lung cancer. The latter can be catheter-related or related to the hypercoagulable state caused by the malignancy. The CT scan of her chest revealed both compression from an endobronchial mass and extensive thrombosis of SVC, left internal jugular, and innominate veins. The clinical course of her symptoms a week before presentation is consistent with a subacute presentation, which in SVC compression only without thrombosis is more chronic. The presence of the chemotherapy port had likely complicated her clinical picture and resulted in part of the thrombotic occlusion on top of the mechanical compression. Management of SVC syndrome necessitates a multidisciplinary approach involving various subspecialists. Treatment options encompass chemotherapy with or without radiotherapy, surgical bypass, endovascular therapy, and catheter-based thrombus removal tailored to the underlying etiology.[3]

AUTHOR CONTRIBUTIONS

Pitchaporn Yingchoncharoen: Writing – original draft. **Firas Ashour:** Writing – original draft. **Romelia Barba Bernal:** Writing – original draft. **Mahmoud Abdelnabi:** Supervision.

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CONFLICT OF INTEREST STATEMENT

None declared.

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DATA AVAILABILITY STATEMENT

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CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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