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SUPERIOR SULCUS TUMOR MASQUERADING AS CERVICAL SPONDYLOSIS COMPLICATED BY PARANEOPLASTIC SIADH

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ABSTRACT **Background:** Superior sulcus tumors are an uncommon presentation of non small cell lung cancer (NSCLC) and frequently manifest with non pulmonary symptoms due to involvement of the thoracic inlet. Such atypical presentations often mimic degenerative or neurological disorders, resulting in delayed diagnosis. **Case presentation:** A 67 year old woman presented with progressive neck pain and right upper limb weakness initially attributed to cervical spondylosis on spinal imaging. Her clinical course was complicated by recurrent hyponatremia initially considered drug induced and later by altered sensorium and respiratory symptoms. Subsequent evaluation revealed severe hyponatremia consistent with syndrome of inappropriate antidiuretic hormone secretion (SIADH). Contrast enhanced computed tomography of the chest ultimately demonstrated a right apical lung mass with extensive thoracic inlet invasion, brachial plexus involvement, venous encasement with internal jugular vein thrombosis, and widespread metastatic disease. Histopathology and immunohistochemistry confirmed metastatic poorly differentiated carcinoma of lung origin, consistent with advanced stage NSCLC. **Conclusion:** This case highlights how superior sulcus NSCLC can evade early detection through a convergence of misleading clinical features, including degenerative cervical spine findings and presumed drug induced hyponatremia. Progressive upper limb neurological deficits, recurrent or unexplained SIADH, and evidence of thoracic inlet vascular involvement should prompt early thoracic imaging, even in the absence of initial respiratory symptoms, to avoid diagnostic delay and advanced disease at presentation.

KEYWORDS : Non small cell lung cancer; superior sulcus (Pancoast) tumor; cervical radiculopathy mimic; paraneoplastic SIADH; venous thrombosis; diagnostic delay

INTRODUCTION

Lung cancer remains the leading cause of cancer related mortality worldwide, largely because many patients continue to present with advanced-stage disease at the time of diagnosis despite improvements in screening strategies.[1] Non small cell lung cancer (NSCLC) constitutes the majority of lung cancer cases and is responsible for most lung cancer related morbidity and mortality, particularly when diagnosis occurs at an advanced stage.[2]

Superior sulcus tumors are a rare subset of bronchogenic carcinomas arising from the lung apex and account for less than 5% of lung cancers.[3] Due to their anatomical location, these tumors frequently invade structures of the thoracic inlet, including the brachial plexus, ribs, vertebral bodies, and subclavian vessels, resulting in distinctive non pulmonary clinical manifestations.[3] Patients therefore commonly present with shoulder pain, neck pain, or upper limb neurological deficits rather than respiratory symptoms, which contributes to delayed recognition of the underlying malignancy.[3]

Misdiagnosis as degenerative cervical spine disease is a well-described cause of diagnostic delay in superior sulcus tumors, as the neurological features closely mimic cervical radiculopathy and often lead to initial evaluation by orthopedic or neurological services.[4]

Furthermore, while paraneoplastic syndrome of inappropriate antidiuretic hormone secretion (SIADH) is well recognized in small cell lung cancer occurring in 10-45% of cases it is remarkably rare in non-small cell lung cancer (NSCLC), with an incidence of only approximately 1%. Crucially, such paraneoplastic markers may precede the radiological detection of the primary tumor, serving as an initial clinical sign that can further obscure the diagnosis.[5,6]

We present a case of advanced NSCLC with superior sulcus involvement, initially masquerading as cervical spondylosis with presumed drug-induced hyponatremia, later complicated by paraneoplastic SIADH and extensive venous thrombosis, illustrating multiple mechanisms of diagnostic delay.

Case Presentation

A 67-year-old woman with a history of hypothyroidism and systemic hypertension initially presented with a six-month history of right-sided neck pain radiating to the right upper limb. There were no associated constitutional or respiratory symptoms at onset. Magnetic resonance imaging of the cervical spine demonstrated degenerative changes consistent with cervical spondylosis, and she was treated conservatively with gabapentin and oxcarbazepine.

Over the next few months, she developed gradually progressive weakness of the right upper limb, eventually progressing to complete monoplegia. The absence of bowel or bladder involvement, cranial nerve deficits, or sensory level reinforced the initial diagnosis of degenerative cervical radiculopathy.

She later developed subacute altered sensorium characterized by confusion, agitation, and irritability. Non-contrast CT brain revealed calcified granulomas in the right cerebellar and temporal regions without perilesional edema, considered incidental. Six days later, she developed new-onset cough and progressive dyspnea. Chest radiography showed right upper and lower zone homogeneous opacities with air bronchograms and right rib fractures suggestive of pneumonia for which empirical intravenous antibiotics were started, she was admitted for further evaluation.

On admission, she was hypoxic and encephalopathic. Neurological examination showed she demonstrated 0/5 power across all muscle groups of the right upper extremity with absent deep tendon reflexes, while power and reflexes in left upper limb and bilateral lower limbs remained normal, consistent with a lower motor neuron-type brachial plexopathy. Notably, there were no clinical features of Horner's syndrome (ptosis, miosis, or anhidrosis). No bowel or bladder involvement or cranial nerve deficits.

Laboratory investigations revealed severe hyponatremia (117 mEq/L) with otherwise normal hematological, renal, and hepatic parameters. Cultures and viral panels results returned negative. Given oxcarbazepine exposure, hyponatremia was initially attributed to the drug, and hypertonic saline was initiated. Thyroid function tests and serum cortisol levels were normal.

Ultrasound abdomen showed increased hepatic echotexture with multiple hepatic cysts and bilaterally increased renal cortical echotexture with a right simple renal cyst. Echocardiography showed preserved systolic function (ejection fraction 53%). Despite correction of sodium, agitation persisted.

Contrast-enhanced CT chest revealed a large right upper lobe mass encasing the right main bronchus with cervical, supraclavicular, and axillary lymphadenopathy and lytic rib and vertebral lesions. CT chest angiography demonstrated extensive thoracic inlet invasion with encasement of the brachiocephalic, internal jugular, and subclavian veins, internal jugular vein thrombosis, right subclavian and vertebral arterial encasement, right hemi diaphragmatic palsy, and widespread metastatic disease (liver, adrenal, bone), consistent with stage T4N3M1c disease.

An ultrasound-guided biopsy of a right supraclavicular lymph node showed a poorly differentiated malignant neoplasm with giant cell features. IHC showed CK7, pan-cytokeratin (AE1/AE3), CK19, vimentin, GATA3, and CD10 positivity and negativity for CK20, TTF-1, Napsin A, synaptophysin, CK5/6, mammaglobin, GCDFP-15, WT-1, PAX8, CDX2, HepPar-1, CA19-9, and HER2/neu. The GATA3 and CD10 positivity necessitated the exclusion of breast and urothelial primaries; however, the lack of site-specific markers (mammaglobin, GCDFP-15) and the radiographic presence of a massive apical lung mass led to a diagnosis of metastatic poorly differentiated non–small cell lung carcinoma favoring a lung primary with giant cell features on clinic radiological correlation.

During hospitalization, she developed **hypercapnic respiratory failure**, requiring non-invasive ventilation, with **recurrent hyponatremia**, low serum osmolality with inappropriately high urine osmolality and sodium excretion whilst being euvolemic after which SIADH was reclassified as **paraneoplastic**. She improved with supportive care and was discharged on **high-flow nasal oxygen**, with plans for further oncological management.

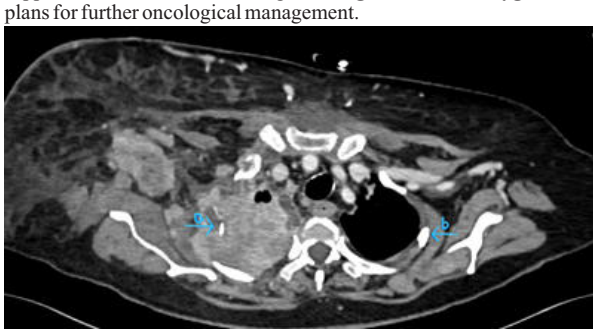

Figure 1: Axial CECT image at the thoracic inlet level demonstrating a large right-sided superior sulcus mass. Arrow (a) indicates lytic destruction of the right rib and vertebral body due to direct tumor invasion, contrasted with the intact contralateral bone (arrow b). Note the significant diffuse subcutaneous edema in the right upper limb and neck, consistent with secondary lymphedema from venous and lymphatic obstruction.

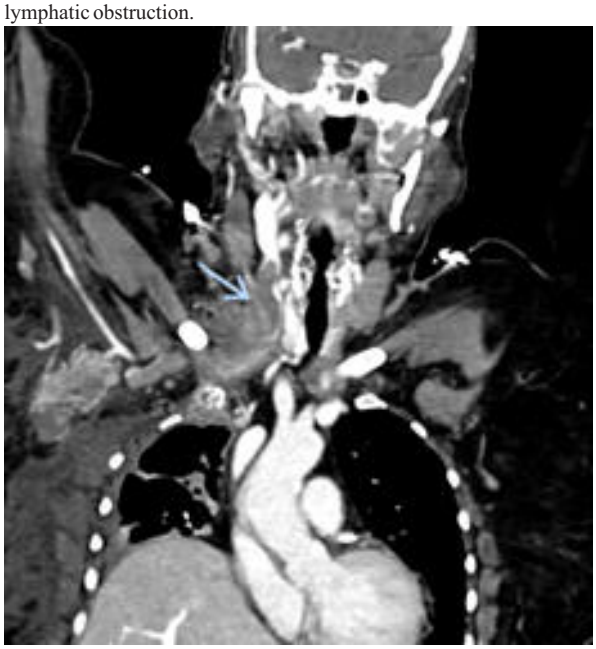

Figure 2: Coronal CT angiography reconstruction showing the right apical mass. The blue arrow indicates a hypodense thrombus within the right internal jugular vein resulting from tumor encasement. Note the significantly elevated right hemidiaphragm, consistent with right phrenic nerve involvement and subsequent palsy

DISCUSSION

This case highlights several well recognized but frequently underappreciated causes of delayed diagnosis in superior sulcus tumors. Because of their apical location, these tumors commonly invade the brachial plexus and adjacent bony structures, producing shoulder pain, neck pain, and progressive upper limb weakness that closely resemble cervical radiculopathy.[3] As demonstrated in clinical series, such patients are often initially managed for degenerative cervical spine disease, leading to prolonged diagnostic latency before appropriate thoracic imaging is obtained. [4]

The absence of early pulmonary symptoms further compounds this delay. Superior sulcus tumors may remain clinically silent from a respiratory perspective until significant local invasion has occurred, a feature consistently emphasized in major reviews of these tumors.[3] Consequently, diagnosis is often made when the disease is already locally advanced or metastatic.[7]

A major diagnostic confounder in this case was that SIADH clinically preceded radiological detection of the lung malignancy. This underscores that SIADH can be the initial manifestation of NSCLC, even before respiratory symptoms appear. Given its rare association with NSCLC (~1% incidence, compared with up to 45% in SCLC), the coexistence of SIADH with a hyponatremia-inducing drug such as oxcarbazepine increases the risk of misattribution and delays in oncologic evaluation.[5,6]

The recurrent and symptomatic nature of hyponatremia in our patient, despite correction, and withdrawal of potential offending drugs, low serum osmolality with inappropriately high urine osmolalityand sodium excretion fulfilling the Schwartz and Bartter criteria was ultimately consistent with paraneoplastic SIADH. Paraneoplastic endocrine syndromes may fluctuate with tumor burden and are associated with advanced disease and adverse prognosis in lung cancer.[5,6]

Vascular involvement provided an additional clue to the underlying diagnosis. Superior sulcus tumors frequently encase or obstruct subclavian and jugular veins, resulting in upper limb edema and venous thrombosis.[3] Such findings indicate extensive thoracic inlet involvement and are characteristic of advanced disease, particularly in cases with delayed diagnosis.[3]

Together, cervical radiculopathy-like symptoms, misleading metabolic abnormalities, and delayed chest imaging created a cascade of diagnostic anchoring that culminated in advanced-stage NSCLC at presentation, a pattern that has been well described in superior sulcus tumors.[3]

CONCLUSION

Superior sulcus tumors represent an uncommon but clinically important presentation of non small cell lung cancer, frequently manifesting with non pulmonary symptoms that mimic degenerative cervical spine disease. This case underscores how progressive upper limb neurological deficits, recurrent hyponatremia, and venous thrombosis particularly when evolving despite appropriate treatment should prompt reconsideration of the initial diagnosis and early thoracic imaging. Awareness of these atypical presentations is essential to avoid diagnostic delay and advanced-stage disease at presentation.

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