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Severe Hyponatremia Due to Paraneoplastic SIADH Revealing Occult Small Cell Lung Carcinoma

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Abstract: ***This case study aims to highlight the diagnostic complexity and clinical management of a comatose patient with hyponatremia, ultimately diagnosed with paraneoplastic SIADH secondary to small-cell lung carcinoma. A 52-year-old male chronic smoker presented in a comatose state due to severe hyponatremia, with serum sodium measured at 109 mmol/L. Initial neuroimaging excluded intracranial pathology, while thoracic CT and bronchoscopy revealed an infiltrative lesion in the right upper lobe. Histopathology confirmed small cell lung carcinoma (SCLC) with neuroendocrine features. Laboratory findings supported a diagnosis of syndrome of inappropriate antidiuretic hormone secretion (SIADH). Initial treatment with hypertonic saline failed to improve sodium levels. A modified protocol involving fluid restriction, oral sodium supplementation, loop diuretics, and desmopressin led to controlled correction. This case underscores the importance of recognizing paraneoplastic SIADH as a potential initial presentation of SCLC and highlights the value of a multidisciplinary approach to diagnosis and management.***

Keywords: small cell lung carcinoma, SIADH, severe hyponatremia, desmopressin therapy, paraneoplastic syndrome

1. Case Presentation

A 52-year-old man, a heavy smoker with hypertension and other chronic medical conditions, presented to the emergency department comatose. Serum sodium was 109 mmol/L. He was admitted to the ICU for stabilization and management of severe hyponatremia.

Investigations: Head CT was negative for acute intracranial hemorrhage. Chest CT demonstrated an infiltrative lesion in the right upper lobe, localized to segments 2 and 3 of the right upper lobe. Bronchoscopy confirmed an infiltrative lesion in the right upper lobe (segments 2–3), and histopathology revealed small cell carcinoma with neuroendocrine features, supported by chromogranin and TTF1 positivity.

2. Diagnosis

Upon laboratory findings of low serum osmolality, high urine osmolality, high urine sodium levels, and clinical euvolemia, and in the absence of renal or pituitary dysfunction and adrenal insufficiency, syndrome of inappropriate antidiuretic hormone secretion (SIADH)was diagnosed.

3. Management and Course

Initial treatment in the ICU included hypertonic 3% NaCl for severe symptomatic hyponatremia. As this had no effect, therapy was adjusted to fluid restriction, oral sodium supplementation, loop diuretic administration, and desmopressin therapy, which resulted in correction of the hyponatremia. During active correction, controlled sodium increase was targeted at ~0.5–1 mEq/L per hour with a goal total correction ≤8 mEq/L in 24 hours unless acute neurologic deterioration necessitated faster initial correction. Desmopressin was administered intranasally (dosed and titrated clinically; intranasal regimens commonly ranged from 10 to 20 µg, with frequency individualized) to prevent or manage overly rapid sodium rises. Serum sodium levels and neurologic function were monitored at 2 to 4-hour intervals during active management. The patient was referred for oncologic staging and treatment.

4. Conclusion

This case underscores the clinical value of early recognition of paraneoplastic syndromes in facilitating timely cancer diagnosis, particularly in patients presenting with non-specific neurological symptoms and electrolyte imbalances. Severe hyponatremia causing coma may be the initial manifestation of SCLC due to paraneoplastic SIADH. Rapid recognition, appropriate diagnostic exclusion of other causes, controlled sodium correction (~0.5–1 mEq/L/hr with ≤8 mEq/L/day limit), use of desmopressin to prevent overcorrection, and prompt oncologic management are critical.

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