

Beyond SIADH: Persistent Encephalopathy Unmasking Sinonasal Small Cell Neuroendocrine Carcinoma

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INTRODUCTION

Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) is a well-recognized paraneoplastic manifestation of small cell neuroendocrine carcinomas and represents an important cause of euvolemic hypotonic hyponatremia. Although correction of hyponatremia often results in neurological recovery, persistent or worsening encephalopathy despite normalization of serum sodium should prompt clinicians to investigate alternative diagnoses, including intracranial tumor extension, paraneoplastic neurological syndromes, and central nervous system infections. Sinonasal small cell neuroendocrine carcinoma is a rare, highly aggressive malignancy that may initially present with paraneoplastic SIADH, masking the underlying disease. This case highlights the importance of reassessing the diagnosis when the clinical course is discordant with biochemical improvement and emphasizes the need for early neuroimaging to identify structural pathology and facilitate timely oncological treatment.

CASE PRESENTATION

A 41-year-old man presented with a 2–3-month history of progressive left-sided neck swelling and 13-kg weight loss. Excisional biopsy of a left supraclavicular lymph node revealed metastatic small cell neuroendocrine carcinoma. He subsequently developed severe hypotonic hyponatremia consistent with SIADH, with persistent confusion and agitation despite sodium correction. MRI brain demonstrated an enhancing mass involving the bilateral ethmoid and left sphenoid sinuses, with nasal septal erosion, early cribriform plate involvement, and adjacent dural enhancement. CSF studies were negative for infection. He was treated with carboplatin–etoposide chemotherapy followed by concurrent radiotherapy, with subsequent improvement in sodium levels and neurological status.

FIGURE 1: CT BRAIN 26/3/26

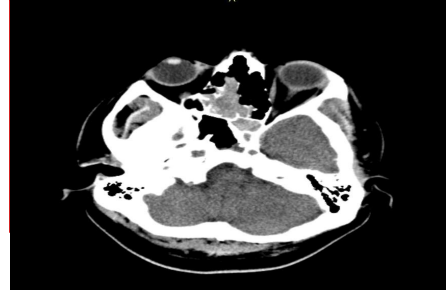


FIGURE 2: CT NCAP 27/3/26



TIMELINE OF KEY EVENTS

March 5, 2026

Left supraclavicular node biopsy confirms metastatic small cell neuroendocrine carcinoma.

March 16, 2026

Presents to EAR with urinary retention; severe hyponatremia (Na 116 mmol/L) noted. Patient leaves against medical advice.

March 24, 2026

Readmitted from endocrinology clinic with acute confusion and disorientation; diagnosed with SIADH

March 25, 2026

Shifted to ICU for worsening altered mental status and hyponatremia (Na 114 mmol/L); managed with 3% hypertonic saline.

March 26, 2026

LP performed, Empiric IV Acyclovir started. MRI Brain reveals ethmoid/sphenoid mass eroding the nasal septum and infiltrating the cribriform plate and adjacent dura.

March 28, 2026

Stepped down from ICU; Cycle 1 of Carboplatin and Etoposide chemotherapy initiated

April 3, 2026

Discharged in stable condition with normalized sodium (Na 147 mmol/L) and improved mentation.

FIGURE 3: MRI BRAIN 26/3/26



METHODS

This descriptive case study provides an in-depth examination of a single patient's experience with sinonasal neuroendocrine small cell tumor. The study focuses on the clinical presentation, diagnostic challenges, and treatment strategies employed. Detailed patient history, diagnostic imaging, histopathological evaluations, and therapeutic interventions were meticulously documented and analyzed to offer comprehensive insights into the management of this rare malignancy.

CLINICAL TAKEAWAYS

When hyponatremia corrects but the mind doesn't clear, look past SIADH. Persistent encephalopathy neurological function, and the patient continued after adequate sodium correction is a red flag for structural disease condition.

-image the brain early.

RESULT

The patient was diagnosed with syndrome of inappropriate antidiuretic hormone secretion (SIADH) secondary to metastatic sinonasal small cell neuroendocrine carcinoma, based on severe hypotonic euvolemic hyponatremia with inappropriately concentrated urine and elevated urine sodium. Despite gradual correction of serum sodium with hypertonic saline, the patient's neurological status remained impaired, prompting further diagnostic evaluation. Contrast-enhanced magnetic resonance imaging identified an ethmoidal and sphenoidal mass with erosion of the cribriform plate and adjacent dural involvement, explaining the persistent encephalopathy. Cerebrospinal fluid analysis excluded infectious causes. Following initiation of carboplatin–etoposide chemotherapy, the patient demonstrated progressive clinical improvement with normalization of serum sodium, resolution of altered mental status, and stabilization of neurological function. He was discharged in stable condition and subsequently continued outpatient chemotherapy and concurrent radiotherapy.

