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AN INTERESTING CASE OF ALTERED SENSORIUM DUE TO HYPONATREMIA: A DIAGNOSTIC AND THERAPEUTIC CHALLENGE OF SIADH IN AN ELDERLY PATIENT

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ABSTRACT

Background: Hyponatremia, defined as serum sodium below 135 mEq/L, is among the most prevalent electrolyte disturbances in clinical medicine and a leading cause of hospital-acquired neurological deterioration. The Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) is a frequently under-recognized yet eminently treatable cause of euvolemic hyponatremia. **Case Summary:** We describe a 74-year-old male presenting with a two-day history of progressive altered sensorium and oliguria. Laboratory evaluation demonstrated severe hyponatremia (serum sodium 110 mmol/L), serum hypotonicity, and inappropriately concentrated urine — fulfilling all Bartter and Schwartz criteria for SIADH. Imaging and endocrine evaluation excluded structural, renal, adrenal, and thyroid etiologies. The patient was managed with fluid restriction, hypokalemia correction, and cautiously titrated hypertonic saline, achieving full neurological and biochemical recovery. **Conclusion:** This case highlights SIADH as a critical and reversible cause of altered mental status in the elderly. A systematic biochemical approach and adherence to safe sodium correction protocols are essential to prevent osmotic demyelination and fatal outcomes.

KEYWORDS: SIADH; Hyponatremia; Altered sensorium; Euvolemia; Bartter and Schwartz criteria; Osmotic demyelination syndrome; Elderly.

1. INTRODUCTION

Hyponatremia (serum sodium <135 mEq/L) is the most common electrolyte disorder in hospitalized patients, with an estimated prevalence of 15–30% in medical wards.[1] Its clinical manifestations range from subclinical cognitive slowing to life-threatening cerebral edema, seizures, and coma. Among the diverse etiologies of hyponatremia, the Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) stands out as a major, often underappreciated, cause of euvolemic hyponatremia — particularly in elderly patients presenting with nonspecific neurological decline.

SIADH is characterized by the non-osmotic secretion of arginine vasopressin (AVP), leading to impaired free water excretion, dilutional hyponatremia, and inappropriately concentrated urine in the absence of volume depletion, renal failure, or endocrine insufficiency.[2] Despite its reversibility, SIADH is frequently missed in elderly patients whose neurological symptoms are often attributed prematurely to stroke, dementia, or metabolic encephalopathy.

We present a clinically instructive case of severe SIADH (serum sodium 110 mmol/L) in a 74-year-old male, in whom meticulous application of the Bartter and Schwartz diagnostic criteria guided safe and effective management, resulting in complete neurological recovery.

2. CASE PRESENTATION

2.1 Patient History

A 74-year-old male farmer with a background of chronic alcoholism and tobacco smoking presented to the

emergency department with a two-day history of progressively worsening altered sensorium and a one-day history of oliguria. The onset of confusion was gradual and non-fluctuating. There was no preceding history of fever, headache, seizures, head trauma, focal neurological deficit, vomiting, or recent initiation of diuretics, antipsychotics, or antidepressants.

2.2 Clinical Examination

On admission, the patient was confused and only partially responsive to verbal commands, with a Glasgow Coma Scale (GCS) score of 11 (E3V3M5). Vital signs were stable: heart rate 84 bpm, blood pressure 118/76 mmHg, respiratory rate 16/min, SpO_2 97% on room air, and afebrile. General examination revealed no peripheral edema, ascites, or signs of dehydration, consistent with a euvolemic state. Neurological examination demonstrated diminished deep tendon reflexes bilaterally but preserved motor responses to painful stimuli. No meningeal signs, papilloedema, or focal deficits were elicited.

2.3 Investigations

Biochemical investigation revealed severe hyponatremia and concurrent hypokalemia. The diagnostic workup systematically fulfilled the Bartter and Schwartz criteria for SIADH, as summarized in Table 1.

Table 1: Laboratory parameters at presentation and at discharge.

Parameter	Initial Value	Final Value	Reference / Interpretation
Serum Sodium (mmol/L)	110	135	Severe hyponatremia (<135)
Serum Potassium (mmol/L)	2.8	3.8	Hypokalemia (<3.5)
Serum Osmolality (mOsm/kg)	264	—	Hypotonic (<275)
Urine Osmolality (mOsm/kg)	438	—	Inappropriately high (>100)
Urine Sodium (mEq/L)	176	—	Elevated (>40)
Serum Cortisol / TFT	Normal	—	Adrenal/thyroid causes excluded
Blood Urea (mg/dL)	17.2	—	Normal
Serum Creatinine (mg/dL)	0.6	—	Normal renal function
Volume Status	Euvolemic	—	No edema or ascites
ABG pH / HCO_3	7.2 / 21	—	Metabolic acidosis

TFT = Thyroid Function Test; ABG = Arterial Blood Gas; HCO_3 = Bicarbonate.

2.4 Imaging and Additional Studies

- CT Brain (non-contrast): No acute infarction, hemorrhage, or mass lesion.
- Ultrasonography of the abdomen: Normal hepatic, renal, and splenic morphology; no free fluid.
- Chest radiograph: No consolidation, effusion, or mediastinal widening.
- Arterial Blood Gas: pH 7.2, pCO_2 24 mmHg, pO_2 125 mmHg, HCO_3 21 mEq/L — consistent with high-anion-gap metabolic acidosis, likely related to chronic alcoholism.

3. TREATMENT AND CLINICAL COURSE

On establishing the diagnosis of SIADH, a structured management plan was initiated in consultation with the Nephrology and Neurology teams.

Fluid restriction to 800–1000 mL per 24 hours was implemented as the primary intervention. Given the severity of hyponatremia (110 mmol/L) and the presence of neurological symptoms, 3% hypertonic saline was cautiously administered intravenously, targeting a correction rate of no more than 8–10 mEq/L in the first 24 hours and 18 mEq/L over 48 hours to minimize the risk of osmotic demyelination syndrome (ODS).[3]

Concurrent hypokalemia (2.8 mmol/L) was corrected with intravenous potassium chloride supplementation. Serum electrolytes were monitored every 4–6 hours during the acute phase. The patient showed progressive clinical improvement, with sensorium normalizing over 3–4 days in parallel with biochemical correction, achieving a discharge sodium of 135 mmol/L. No evidence of ODS or neurological sequelae was observed on follow-up.

4. DISCUSSION

This case illustrates the diagnostic and therapeutic challenges inherent to severe SIADH presenting as encephalopathy in the elderly. Three aspects merit detailed discussion: the diagnostic approach, the pathophysiology in the context of chronic alcoholism, and the critical importance of safe sodium correction.

4.1 Diagnostic Approach: Applying the Bartter and Schwartz Criteria

The Bartter and Schwartz criteria, first described in 1957, remain the foundational diagnostic framework for SIADH.[4] This patient met all five criteria: (i) serum hypo-osmolality (<275 mOsm/kg); (ii) urine osmolality inappropriately concentrated relative to serum (>100 mOsm/kg; here 438 mOsm/kg); (iii) elevated urinary sodium excretion (>40 mEq/L; here 176 mEq/L) in the absence of diuretic use; (iv) clinical euvolemia without edema or volume depletion; and (v) normal thyroid, adrenal, and renal function. The systematic exclusion of hypothyroidism (TFT normal) and adrenal insufficiency (morning cortisol normal) was essential, as these are obligatory steps before confirming SIADH.[2]

4.2 Pathophysiological Considerations in the Elderly Alcoholic Patient

Elderly patients with chronic alcoholism represent a particularly vulnerable population for SIADH and its

complications. Chronic alcohol use is associated with non-osmotic AVP stimulation, as well as nutritional deficiencies — notably thiamine, folate, and magnesium — that compound neurological vulnerability.[1] The gradual onset of hyponatremia in this patient (sensorium change over two days rather than hours) suggests a chronic or subacute course, during which astrocytic adaptation through organic osmolyte extrusion partially attenuated cerebral edema. This chronicity, while protective in one sense, heightens the risk of ODS upon overly rapid correction, as the brain has become volume-depleted relative to the extracellular space.[3]

The concurrent metabolic acidosis on ABG is noteworthy and is attributable to the combined effects of alcoholic ketoacidosis and possible lactic acidosis from poor nutritional status, rather than to SIADH itself. This underscores the importance of a systematic multi-system evaluation even when a unifying diagnosis is established.

4.3 Management Principles and Prevention of Osmotic Demyelination

The treatment of severe symptomatic hyponatremia requires a fine balance between adequate correction to relieve cerebral edema and sufficiently slow correction to prevent ODS — a potentially irreversible pontine or extrapontine myelinolysis.[3] Current guidelines recommend an initial target correction of 1–2 mEq/L per hour for the first 2–3 hours in patients with severe symptoms, followed by a controlled rate not exceeding 8–10 mEq/L per 24 hours thereafter.[1,2] Hypertonic saline (3% NaCl) remains the agent of choice in symptomatic patients and was used effectively in this case.

Fluid restriction alone is appropriate for mild-to-moderate asymptomatic SIADH but is insufficient as monotherapy when neurological symptoms are present. The concurrent correction of hypokalemia is equally important, as potassium administration has an additive effect on serum sodium concentration.[1]

5. CONCLUSION

SIADH is a reversible yet potentially fatal cause of altered mental status in elderly patients that demands systematic biochemical evaluation and judicious management. This case reinforces the value of applying the Bartter and Schwartz criteria in any patient presenting with euvolemic hyponatremia and neurological symptoms. The therapeutic paradox of SIADH — where both under-treatment and over-treatment carry serious risks — necessitates close electrolyte monitoring, a controlled correction protocol, and multidisciplinary input. Heightened clinical awareness of SIADH in elderly patients with unexplained encephalopathy can prevent delayed diagnosis and its potentially irreversible consequences.

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