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Cerebral Salt Wasting Syndrome in an Elderly Patient With Traumatic Brain Injury: Diagnostic Challenges. A case report

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Abstract

OBJECTIVE: Cerebral salt-wasting syndrome (CSWS) is an underdiagnosed cause of hyponatremia following intracranial injuries. This case highlights the diagnostic challenges of CSWS in elderly patients, particularly when typical volume depletion signs are absent.

CASE: We report an 80-year-old male with multiple comorbidities who developed CSWS following motor vehicle accident-related subarachnoid hemorrhage. The patient was confused, with a Glasgow Coma Scale (GCS) score ranging from eight to ten. Serum sodium declined significantly from 145 to 117 mmol/L approximately 20 days. The patient remained euvolemic with normal urine output and did not respond to fluid restriction. Elevated 24-hour urine sodium (563 mEq), and brain natriuretic peptide (154 pg/mL) confirmed CSWS. The patient initially responded to treatment; however, on the seventh day after discharge, he presented to the emergency department with a seizure. The patient died with a serum sodium level of 109 mmol/L.

CONCLUSIONS: CSWS can present without volume depletion signs. Differentiation from SIADH is pivotal, as the therapeutic approaches vary considerably. In this group of patients with intracranial trauma, the prognosis may be worse due to the risk of recurrent severe hyponatremia. Despite treatment, fatal recurrence highlights the need for intensive outpatient follow-up and frequent sodium monitoring.

CASE REPORT

INTRODUCTION

Hyponatremia is the most common electrolyte disorder in hospitalized patients and is linked with increased mortality and length of hospital stay (Spasovski 2024). It affects about 5% of all people and 35% of patients who are hospitalized (Adrogué *et al.* 2022). Hyponatraemia has also been reported in 50% of patients with subarachnoid haemorrhage in neurosurgical units (Hannon *et al.* 2012).

Cerebral salt wasting syndrome (CSWS) is characterized by renal sodium loss linked with intracranial disorders, resulting in hyponatremia and reduced extracellular fluid volume (Harrigan 2001). Although the etiology of CSWS is not fully understood, it is most commonly seen after central nervous system (CNS) injuries. The most frequently reported event is aneurysmal subarachnoid hemorrhage (Hall *et al.* 2025).

The exact mechanism by which intracranial disease leads to CSW remains incompletely understood. In recent years, multiple studies have aimed to identify a causal link between natriuretic peptides and CSWS. Among the different natriuretic peptides, BNP might be the leading candidate responsible for mediating CSWS (Berendes *et al.* 1997).

The onset of this disease usually occurs within the first ten days after a neurosurgical procedure or an intracranial disorder such as a subarachnoid hemorrhage (SAH) or stroke (Diringer *et al.* 2006).

Both cerebral salt wasting syndrome (CSWS) and syndrome of inappropriate antidiuretic hormone secretion (SIADH) may occur following cerebral injury and are accompanied by hyponatremia (Cui *et al.* 2019). Although it is difficult to distinguish between cerebral salt-wasting syndrome (CSWS) and syndrome of inappropriate secretion of antidiuretic hormone (SIADH), correct diagnosis is of great importance because the fluid therapy applied in these two conditions is completely opposite (Tanaka *et al.* 2014) Therefore, careful clinical and laboratory assesment is essential to reach a correct diagnosis.

Patients with CSWS often exhibit significant signs of volume depletion. Symptomatic hyponatremia, high urinary sodium excretion, and increased urine volume are the primary diagnostic features. Neurological symptoms range from mild confusion to profound encephalopathy. In addition, seizures may occur in severe hyponatremia (Hall *et al.* 2025). Although CSWS is not encountered as frequently as SIADH in daily practice, it is very important to keep it in mind in patients with cerebral events and hyponatremia in terms of early initiation of treatment and management of symptoms.

We report CSWS in an 80-year-old polytrauma patient with three notable features: (1) absence

of classical volume depletion despite CSWS diagnosis; (2) fatal recurrence of severe hyponatremia 7 days post-discharge despite documented treatment response; and (3) management challenges in elderly multimorbid patients.

CASE PRESENTATION

An 80-year-old male with a medical history of type two diabetes mellitus, hypertension, and chronic obstructive pulmonary disease (COPD) was admitted to the emergency department following a traffic accident inside a vehicle.

Cranial computed tomography revealed a subdural hemorrhage and subarachnoid hemorrhage (SAH). The neurosurgery department recommended close monitoring, and oral anticoagulant therapy was discontinued in preparation for potential surgery.

Additionally, a liver laceration was identified. A complete blood count and close abdominal examination were advised. Rib fractures were also noted; however, the thoracic surgery department did not schedule the patient for surgery. Based on the multidisciplinary evaluations, the patient was admitted to the intensive care unit (ICU). After stabilization and no longer requiring intensive care, he was transferred to the palliative care unit. The patient was conscious but confused, with a Glasgow Coma Scale (GCS) score ranging between eight and ten. No lateralizing neurological signs were observed, and there were no seizure episodes during the clinical course. There was no need for antiepileptic or anticholinergic medication during follow-up.

Upon evaluation in the palliative care unit, the patient's general condition was moderately poor. Approximately 20 days post-trauma, the patient's serum sodium level had decreased significantly, dropping from 145 mmol/L to 117 mmol/L. No abnormality was observed in the patient's urine output. Plasma osmolality was 246 mOsm/kg and urine osmolality was 410 mosmol/L. A spot urine sodium test revealed a level of 96.9 mmol/L. A 24-hour urine sodium level was also measured, and it was 563 meq. Fractional excretion of uric acid could not be calculated. Other serum electrolyte levels remained within normal limits, and there was no evidence of prior or ongoing diuretic therapy. The patient's brain natriuretic peptide (BNP) level was 154 pg/mL, and central venous pressure (CVP) was within normal limits. Fluid restriction was initiated; however, no improvement in serum sodium levels was observed. The patient was re-evaluated by a neurosurgeon. No additional pathology was observed on follow-up cranial imaging. In the patient who did not respond to fluid restriction, CSWS was assumed to be a more likely pathological condition and 8/10 g daily salt supplementation was added to fluid replacement. Given the presence of hyponatremia, increased renal sodium loss, absence of diuretic use, and a history of cerebral hemorrhage, a diagnosis of CSWS was established. The patient, whose serum sodium level was 131 mmol/L, was discharged from the palliative care unit with the

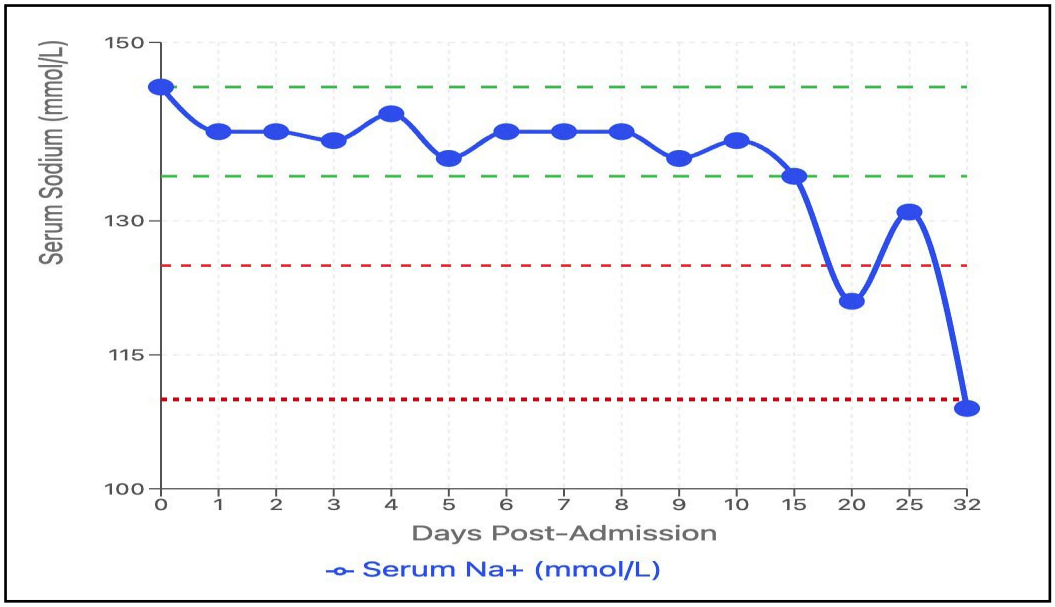

Figure 1. Serum sodium trajectory in an 80-year-old male with cerebral salt wasting syndrome following traumatic subarachnoid hemorrhage.

Serum sodium concentration (mmol/L) is plotted against days from initial trauma (Day 0). Three clinical phases are delineated: **(1)** an initial stable phase (Days 0–5) with sodium within the normal range; **(2)** a progressive decline phase (Days 5–22) during which sodium fell from 145 to 117 mmol/L (rate ≈ 0.88 mmol/L/day), culminating in severe symptomatic hyponatremia; and **(3)** a treatment and post-discharge phase (Days 22–32) in which salt supplementation (8–10 g/day) partially corrected sodium to 131 mmol/L at discharge (Day 25), followed by a rapid fatal decline to 109 mmol/L on Day 32 (rate ≈ 3.1 mmol/L/day).

The **green** dashed lines indicate the normal serum sodium level (145 mmol/L) and the hyponatremia threshold (135 mmol/L). The **red dashed** line marks the symptomatic hyponatremia zone (126 mmol/L). The **red dotted** line denotes the critical severe hyponatremia level (112 mmol/L).

Key clinical events are annotated: **CSWS** treatment initiation (Day 22, sodium 117 mmol/L), **discharge** from the palliative care unit (Day 25, sodium 131 mmol/L), and emergency **readmission** with seizure and subsequent death (Day 32, sodium 109 mmol/L). **Abbreviation**: CSWS, cerebral salt wasting syndrome.

recommendation to consume 8-10 grams of salt per day. The patient was transferred to home care services. Serum sodium levels measured post-discharge by home care services were also 131 mmol/L. The patient was readmitted to the emergency room with a seizure on the seventh day after discharge, and the patient died with a serum sodium value of 109 mmol/L. Figure 1. Serum Sodium Trajectory. Decline began day 5 post-trauma, reaching severe hyponatremia (117.2 mmol/L) on day 22 when CSWS treatment initiated. Recovery to 131 mmol/L by discharge (day 25). Despite continued treatment, terminal decline to 109 mmol/L on day 32. Green dashed lines: normal (145 mmol/L) and hyponatremia threshold (135 mmol/L). Red dashed line: symptomatic zone (126 mmol/L). Black dotted line: critical severe hyponatremia (112 mmol/L).

DISCUSSION

The fatal recurrence (sodium 131→109 mmol/L in 7 days; rate 3.1 mmol/L/day - 3.5× faster than the initial 0.88 mmol/L/day decline) indicates pathophysiologic change. Possible explanations might include non-compliance with 8-10 g/day salt intake or concomitant development of SIADH. Critically, the identical 8-10 g/day salt regimen that previously corrected hyponatremia now failed, suggesting superimposed SIADH rather than simple non-compliance. This dual-mechanism hypothesis (CSWS + SIADH coexistence) may explain both the atypical euvolemic presentation and treatment failure. Frequent sodium monitoring after discharge, followed by regular follow-ups and prophylactic fludrocortisone use should be considered.

Cerebral salt-wasting syndrome (CSWS) is a common reason of hyponatremia in neurosurgical patients (Kawa *et al.* 2024). Various brain pathologies have been defined as potential causes of CSWS; notably, subarachnoid hemorrhage accounts for the majority of cases (Cerdà-Esteve *et al.* 2008). Yet, the literature on this topic is limited. Additionally, the few studies investigating CSWS following traumatic brain injury have identified traffic accidents as the primary cause (Taylor *et al.* 2017, Rojas-Urrea *et al.* 2025).

In this case, a patient was brought to the emergency room after a vehicle accident and was diagnosed with subdural and subarachnoid hemorrhage. Hyponatremia developed during follow-up. Despite their limited number, certain observational studies indicate that CSWS could be more frequently observed than SIADH (Bracco *et al.* 2001). The difference between CSWS and SIADH could be very challenging as both conditions may present in the same patient group. A key difference

between SIADH and CSWS is that CSWS involves renal salt loss leading to hyponatremia and volume depletion, while SIADH is a euvolemic or hypervolemic state. Therefore, it is important to pay attention to volume status in patients with hyponatremia. Additionally, in such intricate cases, natriuretic peptides may facilitate establishing the diagnosis (Cerdà-Esteve *et al.* 2008). In our case, the patient's brain natriuretic peptide (BNP) level was 154 pg/mL, CVP was within normal limits. The absence of volume depletion despite high urine sodium represents a diagnostic challenge in elderly polytrauma patients. The therapeutic response to salt supplementation (sodium improvement from 117 to 131 mmol/L, Figure 1) retrospectively supports the CSWS diagnosis, as SIADH typically worsens with salt loading. This case illustrates that clinicians should maintain CSWS suspicion despite absent classical volume depletion signs and pursue comprehensive diagnostics.

In a prospective observation of 49 patients suffering from SAH, plasma atrial natriuretic peptide (ANP) concentration was unchanged, while BNP levels elevated, particularly in patients with ruptured anterior communicant aneurysm. There was a tendency for hyponatremia in patients with persistent increases in plasma BNP concentration over a week after SAH (Sviri *et al.* 2003). However, the results of the studies are controversial, and the mechanism by which BNP increases in SAH are not fully understood. Furthermore, the increase in natriuretic peptides cannot be the only reason for CSWS (Cerdà-Esteve *et al.* 2008).

Treatment of CSWS aims to correct both hypovolemia and hyponatremia. To maintain cerebral perfusion pressure, the primary goals in the treatment are to preserve normal intravascular volume and sodium concentration (Rabinstein *et al.* 2011). The first line of management involves fluid replacement with isotonic or hypertonic saline, depending on the severity of symptoms. Sodium correction should be gradual, avoiding an increase of more than 10 mmol/L within the first 24 hours (Bouchlarhem *et al.* 2022). Since patients with underlying central nervous system pathology have a higher risk of developing symptoms, hyponatremia should be prevented whenever possible and treated promptly when detected (Yee *et al.* 2010). In addition, it has been reported that treating CSWS may prevent vasospastic cerebral ischemia after SAH. Therefore, intravenous fluid therapy, usually a combination of crystalloids and colloids, should be continued in this patient group (Cerdà-Esteve *et al.* 2008). The use of fludrocortisone, a mineralocorticoid, to increase sodium retention and support volume status in patients with CSWS is also advocated by some clinicians (Bouchlarhem *et al.* 2022).

Hyponatremia in CSWS may persist for weeks to months following the initial CNS event, necessitating ongoing monitoring and management. Throughout treatment, frequent assessment of the GCS and neurological examination is critical to detect any signs of clinical deterioration (Rahman *et al.* 2009). Research results by Chendrasekhar *et al.* demonstrated that individuals with traumatic brain injury who developed CSWS had more severe injuries, spent more time in the hospital and ICU, and required longer ventilator support compared to those without CSWS. Notably, survival to hospital discharge was lower in patients with CSWS (88%) than those without the condition (99%) (Chendrasekhar *et al.* 2020).

Early diagnosis and appropriate fluid and sodium replacement are crucial to reducing the impact of complications and optimizing patient care. Delayed or incorrect treatment can lead to worsening neurological function, prolonged hospitalization, and potentially life-threatening outcomes. Prognosis depends on the underlying neurological condition and the timeliness of treatment. Potential complications include severe hyponatremia, hypovolemia, increased morbidity and mortality, and management challenges due to the need to differentiate from SIADH (Hall *et al.* 2025).

LIMITATIONS

Generalizability is limited due to it being a single case in an elderly patient with multiple traumas. The retrospective design prevents determining whether the fatal outcome was due to treatment non-compliance, disease progression, or concomitant development of SIADH.

CONCLUSION

CSWS can occur in elderly polytrauma patients without the classic signs of volume depletion, making differential diagnosis from SIADH difficult. BNP levels may be normal or slightly elevated, limiting diagnostic utility. Differentiation from SIADH is pivotal, as the therapeutic approaches vary considerably. Early diagnosis and appropriate sodium replacement are crucial, and long-term outpatient monitoring is emphasized to prevent fatal recurrence.

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AUTHOR CONTRIBUTIONS

Concept and Design: Y.K.G., N.Y.Ç.; Writing: original draft preparation: Y.K.G., N.Y.Ç; Review and editing: Y.K.G., N.Y.Ç; Resources: Y.K.G.; Supervision: N.Y.Ç.

PATIENT CONSENT

Written informed consent was obtained from next-of-kin for publication. All identifying information has been removed. This report adheres to CARE guidelines.

ETHICS APPROVAL

This case adheres to CARE guidelines.

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