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Cerebral salt wasting in myelin oligodendrocyte glycoprotein antibody–associated disease: avoiding the syndrome of inappropriate antidiuretic hormone secretion fluid-restriction trap in the emergency department. A case report

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Key words: cerebral salt wasting; MOGAD; hyponatremia; emergency medicine; neurocritical care; fludrocortisone.

Abstract

Hyponatremia is a frequent metabolic emergency in patients with acute neurological disease and may precipitate seizures, cerebral edema, and hemodynamic collapse. Distinguishing cerebral salt wasting from syndrome of inappropriate antidiuretic hormone secretion is clinically challenging because both share similar laboratory features but require opposite management strategies. We report a 66-year-old man with myelin oligodendrocyte glycoprotein antibody–associated disease presenting with generalized seizure and profound hyponatremia. Bedside ultrasound demonstrated hypovolemia, guiding early resuscitation. Serum and urine osmolality, together with marked natriuresis and concentrated urine, supported the diagnosis of cerebral salt wasting. Treatment with hypertonic saline, aggressive isotonic replacement, and short-course fludrocortisone resulted in rapid correction of sodium and hemodynamic stabilization. This case highlights the emergency danger of reflex fluid restriction in neuro-hyponatremia and

emphasizes structured volume assessment to differentiate cerebral salt wasting from syndrome of inappropriate antidiuretic hormone secretion.

Introduction

Hyponatremia is one of the most common metabolic abnormalities encountered in emergency departments and occurs in a substantial proportion of patients presenting with acute neurological disease. Severe hyponatremia may precipitate seizures, cerebral edema, and secondary neuronal injury, making rapid identification of the underlying mechanism essential for safe treatment.[1]

Two entities dominate the differential diagnosis in neuro-hyponatremia: Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) and Cerebral Salt Wasting (CSW). Both conditions produce hypotonic hyponatremia with elevated urine sodium and concentrated urine, yet their pathophysiology and management are fundamentally opposed.[2] SIADH represents euvolemic water retention driven by persistent vasopressin activity, whereas CSW results from renal sodium loss leading to true extracellular volume depletion.[3] Treating CSW with fluid restriction — appropriate therapy for SIADH — may precipitate circulatory collapse, worsen cerebral perfusion, and increase mortality.[1]

Clinical assessment of intravascular volume is unreliable in critically ill patients. Physical findings such as skin turgor, mucous membrane dryness, and blood pressure variability lack sensitivity. Inferior vena cava ultrasound offers a rapid bedside method for estimating volume status, with marked inspiratory collapsibility suggesting hypovolemia.[4] Objective assessment is particularly important in inflammatory neurological disorders where metabolic complications are easily overlooked.

Myelin Oligodendrocyte Glycoprotein Antibody–Associated Disease (MOGAD) is an autoimmune demyelinating disorder affecting optic pathways, brainstem, and periventricular regions. These anatomical areas regulate autonomic and endocrine homeostasis and therefore influence sodium balance.[5] Although neurological manifestations of MOGAD are well characterized, associated electrolyte disturbances remain poorly described. We present a case of life-threatening CSW in MOGAD to highlight an emergency diagnostic framework that prevents the SIADH fluid-restriction trap.

Case Report

A 66-year-old man with established MOG antibody–associated disease presented after a witnessed generalized tonic–clonic seizure followed by confusion. He had no prior epilepsy, diuretic use, gastrointestinal losses, or excessive water intake.

On arrival, Glasgow Coma Scale score was 14. Vital signs demonstrated tachycardia and hypotension requiring vasopressor support. Physical examination revealed dry mucous membranes and reduced skin turgor, suggesting intravascular depletion.

Bedside inferior vena cava ultrasound demonstrated a small diameter vessel with marked inspiratory collapse consistent with hypovolemia.

Laboratory evaluation revealed severe hypotonic hyponatremia with inappropriately concentrated urine and marked urinary sodium excretion. Serum potassium was normal. Endocrine testing excluded adrenal crisis. The seizure was attributed to acute symptomatic hyponatremia.

Immediate treatment included hypertonic saline bolus followed by aggressive isotonic fluid replacement guided by urine output and hemodynamics. Despite large replacement volumes, natriuresis persisted and fludrocortisone therapy was initiated. Serum sodium corrected gradually with parallel hemodynamic stabilization. Urinary indices normalized within 48 hours. Neurological status returned to baseline and the patient was discharged without deficit. The time course of biochemical and hemodynamic parameters during treatment is summarized in Table 1, while serial changes in serum and urine sodium are illustrated in Figure 1, and associated fluid balance and hemodynamic response are shown in Figure 2.

Discussion

CSW represents one of the most dangerous mimics of SIADH in emergency neurocritical care because laboratory findings are nearly indistinguishable while management is opposite.[2] Reflex fluid restriction — a common response to hyponatremia — may exacerbate hypovolemia and cerebral hypoperfusion in CSW.[1] Notably, severe hyponatremia is an independent predictor of mortality in hospitalised patients, and inappropriate or delayed therapy — for example, misclassifying CSW as SIADH and imposing fluid restriction — significantly worsens patient outcomes.

The central mechanism of CSW involves renal sodium wasting driven by neurohumoral factors released after brain injury. Natriuretic peptides suppress renin–angiotensin–aldosterone signalling, promoting sodium loss and extracellular volume depletion.[6] Secondary antidiuretic hormone release produces biochemical similarity to SIADH despite fundamentally different physiology.

Dynamic urinary response to therapy is a key discriminator. In SIADH, urine sodium remains elevated despite isotonic saline because vasopressin activity persists. In CSW, restoration of intravascular volume suppresses natriuretic drive, leading to rapid decline in urine sodium and osmolality.[3] The marked biochemical reversal in this patient confirmed CSW physiology.

Ideally, the diagnostic workup of hyponatremia includes serum osmolality, urine osmolality, and urine sodium, but these laboratory tests are not always rapidly available in the emergency setting.[11] In contrast, point-of-care ultrasound offers a quick, bedside assessment of volume status, which can guide management while awaiting laboratory results.

Bedside ultrasound of the inferior vena cava provided immediate objective evidence of hypovolemia, supporting aggressive resuscitation.[4] Ultrasound-guided volume assessment has demonstrated superior reliability compared with clinical examination alone and is increasingly incorporated into emergency hemodynamic algorithms. A structured emergency diagnostic approach integrating bedside volume assessment is illustrated in Figure 3. A thorough personal and medication history is also crucial; review of chronic medications such as thiazide diuretics or SSRIs, and comorbid conditions, often provides important clues to the underlying cause of hyponatremia and should inform diagnostic reasoning.

MOGAD's clinical spectrum, including brainstem and diencephalic involvement that can precipitate sodium dysregulation, has been well characterized.[12] Brainstem involvement may disrupt autonomic regulation of renal sodium handling.[5,7] Neuroinflammation may also stimulate natriuretic peptide release similar to mechanisms described in subarachnoid hemorrhage.[8]

Management differs fundamentally between entities. CSW requires isotonic saline replacement and hypertonic saline for symptomatic hyponatremia, while mineralocorticoid therapy is recommended when natriuresis persists.[9] Fludrocortisone enhances distal tubular sodium reabsorption and expands intravascular volume. Short-term therapy has demonstrated safety and effectiveness in neurocritical CSW.[9,10]

Safe correction limits must be observed to prevent osmotic demyelination. Careful monitoring of sodium trajectory and urine output is essential during high-volume resuscitation.[1]

This case reinforces a critical emergency principle: hyponatremia in neurological disease should never trigger automatic fluid restriction without structured volume assessment. In practice, this means initially obtaining serum and urine osmolality alongside urine sodium to classify the hyponatremic state, while concurrently assessing total blood volume — via clinical examination or point-of-care ultrasound — before deciding on fluid management.

Conclusions

Cerebral salt wasting should be actively considered in neuroinflammatory patients presenting with hyponatremia and hypovolemia. Objective volume assessment combined with urinary indices enables early differentiation from SIADH. Prompt recognition allows life-saving volume resuscitation and targeted mineralocorticoid therapy. Emergency physicians must recognize that reflex fluid restriction can worsen outcomes when CSW is misclassified as SIADH [1–3].

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Table 1. Timeline of biochemical and hemodynamic parameters during treatment.

Parameter	Admission	24 h	48 h	Units
Serum sodium	109	118	130	mmol/L
Serum osmolality	230	245	265	mOsm/kg
Urine sodium	152	85	20.8	mmol/L
Urine osmolality	348.5	210	71.9	mOsm/kg
Serum potassium	4.1	4.2	4.3	mmol/L
Mean arterial pressure	53	82	88	mmHg
Urine output	~5	3.2	1.8	L/24 h

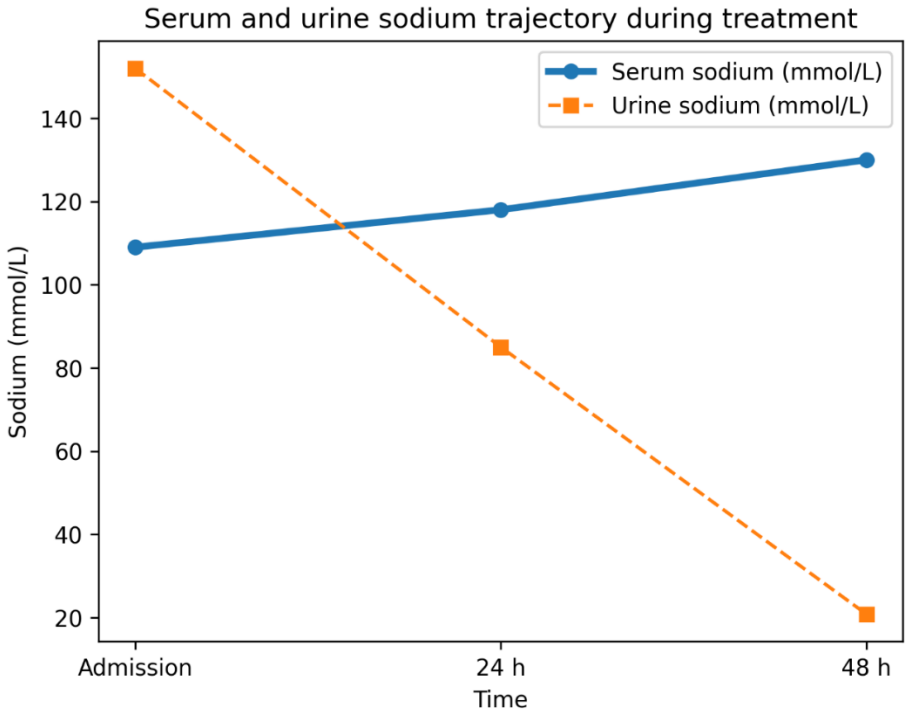

Figure 1. Serum and urine sodium trajectory during treatment.

Graph demonstrating rise in serum sodium and decline in urine sodium over 48 hours following volume resuscitation and fludrocortisone therapy.

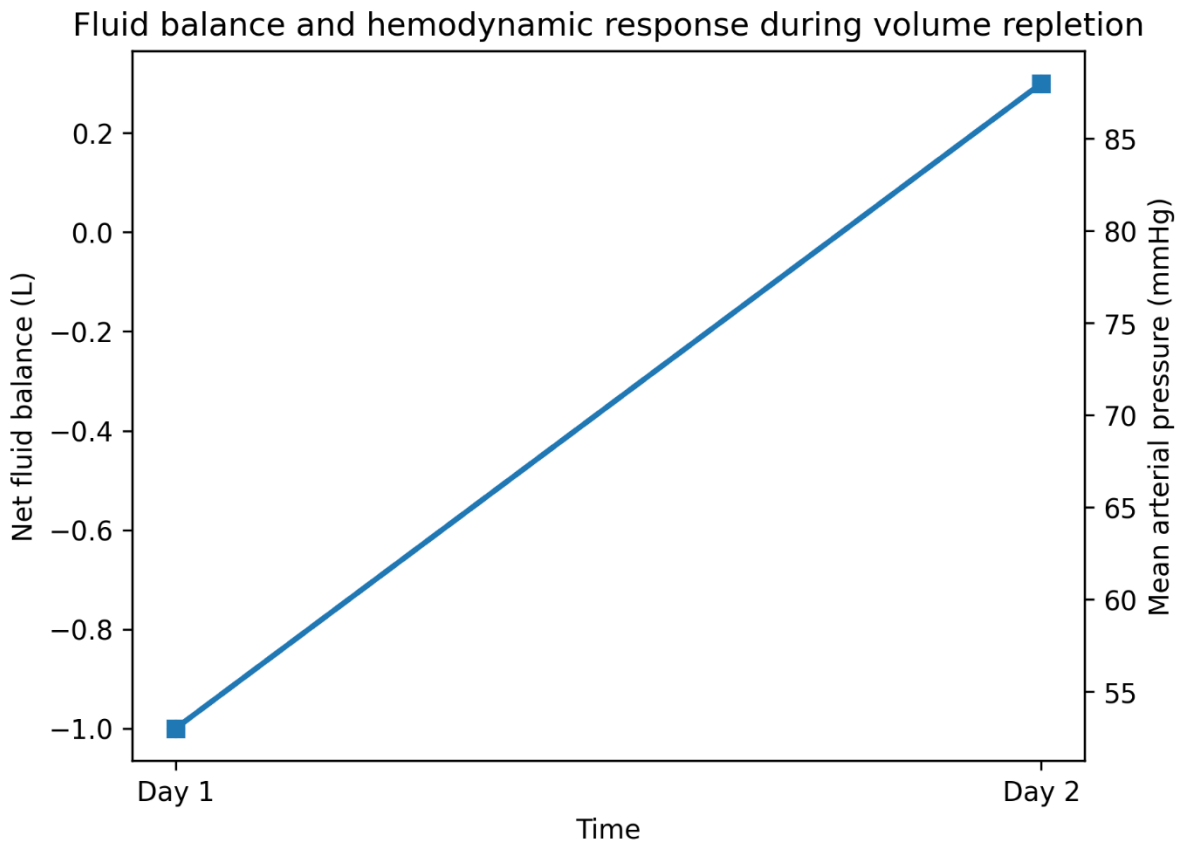

Figure 2. Fluid balance and hemodynamic response.

Net fluid balance changes correlated with improvement in mean arterial pressure during treatment.

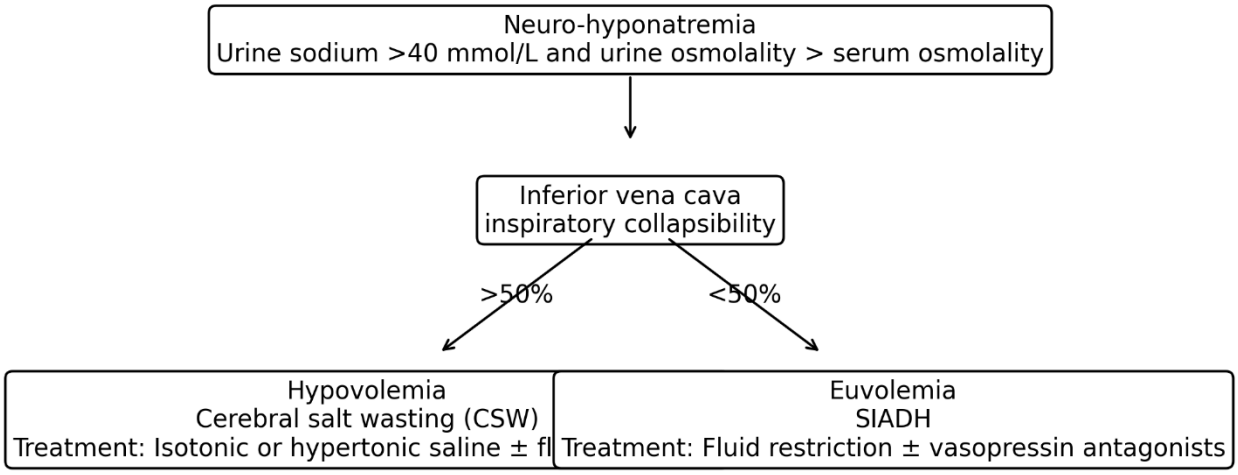

Figure 3. Emergency department diagnostic algorithm for neuro-hyponatremia.
Flow diagram illustrating structured assessment distinguishing cerebral salt wasting from SIADH using urinary indices and bedside volume evaluation.

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Ethics approval: no ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals.

Consent to participate: written informed consent was obtained from the patient (and, given his post-ictal confusion at presentation, additionally confirmed with his legally authorized representative) for publication of this case report and any accompanying clinical details. Participation did not affect the patient's treatment. All data were anonymized and handled in accordance with institutional and ethical guidelines to ensure confidentiality.

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Availability of data and materials: all data underlying the findings are fully available.

AI tools declaration: no AI tools were used in the preparation of this manuscript.