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Cerebral Salt Wasting Syndrome in Traumatic Epidural Hematoma and Subarachnoid Hemorrhage: A Case Report

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ABSTRACT

Introduction: Electrolyte imbalance is common in patients with neurologic disease, including traumatic brain injury. The most common type is hyponatremia, a condition in which serum sodium <135 mmol/L. High morbidity and mortality rates are associated with hyponatremia, especially if the underlying causes are misdiagnosed and improperly treated. One such condition that comes to mind is cerebral salt wasting syndrome (CSWS), which is treated primarily with volume resuscitation and sodium replacements and is characterized by hyponatremia, elevated urine sodium, and hypovolemia. **Case:** A 21-year-old man was hospitalized for a traumatic brain injury due to a traffic accident. He suffered from an epidural hematoma (EDH) and a subarachnoid hemorrhage (SAH). He underwent an emergency decompression craniotomy and hematoma evacuation. The operation went well, as expected. During hospitalization, the patient developed agitation, restlessness, and polyuria with hyponatremia in serum but elevated in urine. We identified that CSWS was the underlying cause and administered isotonic, hypertonic, and hydrocortisone treatments to the patient. The patient showed progressive improvement. His condition tends to be stable, and he has a normal electrolyte level. **Conclusion:** Cerebral salt wasting syndrome (CSWS) is an important but underrecognized cause in hyponatremic patients with central nervous system disorders.

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INTRODUCTION

Hyponatremia is the most common electrolyte imbalance in hospitalized patients with neurological disease.[1,2] Hyponatremia is defined as serum sodium <135 mmol/L.[1] Hyponatremia is frequently associated with patients with acute neurological disorders, but it can also become a clinical dilemma when a patient with an uncertain volume status is hospitalized.

In the hospital ward, 38% to 54% of patients had hyponatremia.[1] The incidence of hyponatremia in patients with subarachnoid hemorrhage (SAH) has been reported to be between 30–55%, ranging from 9.6% to 51% in cases of traumatic brain injury (TBI).[3,4] Hyponatremia is associated with a higher morbidity and mortality rate,[4] especially if the underlying cause is misdiagnosed and not properly treated.

The differential diagnosis for hyponatremia in the presence of central nervous system damage, especially brain injury, includes fluid volume depletion, side effects of certain medications, cerebral salt wasting syndrome (CSWS), syndrome of inappropriate antidiuretic hormone (SIADH), and diabetes insipidus (DI).[5,6] Blood sodium level, urine sodium level, and the patient's volume status can be used to distinguish between them. It is important to have an accurate diagnosis because there are different treatment strategies.

CSWS is characterized by hyponatremia caused by loss of sodium from the renal system, elevated urine sodium, and hypovolemia.[2,5] CSWS is a rare case of hyponatremia in neurotrauma.[7] The pathogenesis of this disorder is still not fully understood.[5] Sympathetic responses, such as natriuretic factors, play an important role in this syndrome.[5] CSWS management requires adequate volume and the maintenance of a salt balance.[2,3]

In this report, we explain the case of a patient with hyponatremia caused by CSWS from a moderate TBI that we treated with isotonic saline, hypertonic saline, and hydrocortisone.

CASE

A 21-year-old man was admitted to the emergency room 10 minutes after being involved in a single motorcycle accident. He was riding his motorcycle when he fell to the left side. He did not wear a helmet. He was then rendered unconscious and taken to the hospital.

At the hospital admission, the primary survey revealed that the airway breathing and circulation were clear; blood pressure was 140/80 mmHg; pulse was 72 beats per minute; respiratory rate was 22 times per minute; the Glasgow Coma Scale (GCS) score was 10 (E2, V3, M5); isochore pupils with diameters of 3 mm; there was no sign of lateralization; and he had some bruises on his head. On the secondary survey, he had no allergies, was not taking any regular medications, or had any medical history before, and his last meal was unknown. A comprehensive head-to-toe assessment was done; there were bruises on his right head, minimal bleeding from his right ear and nose, and blisters on his hands and legs. There were no neck, chest, abdomen, pelvis, or spine abnormalities.

CT brain trauma revealed subarachnoid hemorrhage (SAH) on the right temporal region and the left frontotemporal region; epidural hematoma (EDH) in the right parietal region with a thickness of +/- 0.2 cm; fracture of the os calvaria in the right parietal and right occipital region; a blood-density hyperdense lesion filling the right mastoid; bilateral ethmoidal and sphenoidal hemosinus with suspected cranial base fracture; subgaleal hematoma and emphysema subcutis in the right parieto-occipital region (Figure 1 and 2). The chest x-ray was normal. The laboratory panel showed leukocytosis (leu 20.710/mm$^{3)}$ and hypokalemia (3.06 mmol/L).

This patient was then treated initially with head up 30°, oxygen supplementation with a non-rebreathing mask at 12 liters per minute, securing the neck with a cervical collar, and also receiving fluids and drugs such as ringer acetate, ceftriaxone, ketorolac, omeprazole, tranexamic acid, vitamin K, mannitol, mecobalamin, and citicoline. An emergency craniotomy for hematoma evacuation was scheduled for the patient. During the operation, the patient lost 500 cc of blood and 1000 cc of urine, with fluid input of 500 cc of gelafusal, 230 cc of PRC, and 1000 cc of NS. The blood clot was identified and removed, and his hemodynamics were stable. The patient was then transferred to the ICU room.

In the first 24 hours, vital signs were stable (Figure 3), with adequate contact and a GCS score of E3, Vett, and M6. A 7-cc drain with a reddish color was installed on his head. His urine color was clear at 1.750 cc/24 hours, and his fluid balance per 24 hours was (+) 1.206.5cc. After being corrected, the patient's laboratory panel showed a normal potassium level of 3.53 mmol/L (Table 1).

The patient experienced agitation, restlessness, and another hypokalemia of 3.08 mmol/L three days after being in the ICU, with urine/24 hours of 4110 cc/24 hours and fluid balance/24 hours of (-) 1084 cc. He was given 100 meq/24 hours of KCl. The next day, the patient mumbled and pulled out his CVC line. He had hyponatremia with a sodium level of 127 mmol/L, hypochloremia with a chloride level of 94.7 mmol/L, and a normal potassium level of 3.31 mmol/L, with 2250 cc of urine per 24 hours and (-) 26 cc of fluid balance per 24 hours. The current treatment consists of fluid replacement of 0.9% NS and KCL at 75 mEq/24 hours.

On the sixth day, he was still periodically restless. He developed another hyponatremia, with the lowest value on the seventh day after admission: 117 mmol/L with 4124 ccs of urine per 24 hours, (+)514 cc of fluid balance per 24 hours, and diuresis at 2.4 cc/kg/hour. He was given additional therapy with 3% NS. Considering the patient's clinical and laboratory findings, more investigations were required to identify the underlying cause of hyponatremia and polyuria.

In the urinary electrolytes panel, hypernatremia and hyperchloremia were found to have the same value of 607 mmol/L. The final diagnosis was CSWS, and he was given adequate fluids based on his fluid balance, sodium replacement, and hydrocortisone for his condition. After that, the agitation was slowly reduced, and the patient tended to be stable. The electrolyte andwater balances were followed up after ICU discharge on day 18. On the 21st day of hospitalization, he was discharged with his sodium levels at 136.5 mmol/L, potassium at 3.5 mmol/L, and chloride at 104.1 mmol/L, with a fluid balance per 24 hours of (-)625 cc, 3325 ccs of urine per 24 hours, and diuresis at 2.3 cc/kg BW/hour. The chronology of this case can be seen in Figure 4.

DISCUSSION

We discovered in our case that following surgery, the patient became agitated with polyuria and hyponatremia. Previous studies have suggested that 27–41% of TBI patients develop hyponatremia. Moreover, 51% of TBI patients develop mild hyponatremia, whereas 20% develop moderate and severe hyponatremia.[8] We made a differential diagnosis between CSWS, SIADH, and DI based on the patient's signs and symptoms.[6,9,10] It was important to differentiate them in patients with severe neurologic injuries because their therapeutic strategies are distinct.[6,10]

CSWS is characterized by a hypovolemic condition with low serum osmolality, polyuria, and increased sodium excreted through urine.[7,9] CSWS is not yet clearly defined. It is hypothesized that CSWS develops with increasing natriuretic peptide (NP) levels and changes in the sympathetic nervous system.[1,5,7,11] While SIADH indicates fluid overload with oliguria and high urine sodium due to increasing antidiuretic hormone (ADH) secretion, DI patients also have ADH abnormalities in either decreased release or response to ADH. DI is a hypovolemic condition with low serum osmolality and low urinary sodium. The management of CSWS involves volume resuscitation and sodium replacement, contrary to the management of patients with SIADH,[9,12] where SIADH patients are emphasized for fluid restriction, sodium replacements, diuretics, and treating the underlying causes. In DI patients, we treated them with volume resuscitation and vasopressin.[1,5–8,13]

The main treatment for CSWS is volume replacement and the correction of hyponatremia and natriuresis. Fluid should be administered with normal saline, and 3% NS or sodium supplements may be required.[1,2,13] For additional therapy, using a mineralocorticoid, fludrocortisone, can help normalize serum sodium levels in patients with TBI who experience CSWS.[14–17] In our patient, we administered fluid based on the theory, and instead of using mineralocorticoids, we used hydrocortisone, a glucocorticoid. Hydrocortisone is used due to the absence of fludrocortisone, and it is selected in comparison with other types of glucocorticoids because it has the highest mineralocorticoid effect.[18–20]

Monitoring of adequate and balanced fluid administration in CSWS patients is needed to ensure that the amount of fluidis given appropriately. We can monitor patient awareness, vital signs, urine output, and electrolyte levels periodically to know that the patient hasbeen given the correct therapy.

CONCLUSION

CSWS should be considered in patients with neurological disease, although it is rare. A systematic and comprehensive physical examination and laboratory test should become the primary keys to evaluating the patient's condition since the correct diagnosis is essential for appropriate treatment. A correct and timely diagnosis is important to obtain a good outcome because different conditions are treated differently. CSWS is self-limiting and resolves within a few weeks to months; long-term treatment is not commonly required.

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Author Contribution
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Analysis and interpretation of data: CN-M and SA-PK.
Revision of the manuscript: CN-M, SA-PK, HI, KS-K

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TABLES AND FIGURES

Table 1. Patient electrolyte level (days 1-11 and day 21); serum sodium levels immediately increased and improved after getting therapy

Day	1	2	3	4	5	6	7	8	9	10	11	21
Na	137.5	138.6		138	127	117.4	117	124	128	135	135	136.5
K	3.06	3.53		3.08	3.31	3.85	3.73	4.06	39.5	4.16	3.87	3.5
Cl	103.2	105.6		103	94.7	85.1	86	93.1	95.7	101.7	99	104.1
Na (urine)									607			
K (urine)									57			
Cl (urine)									607			

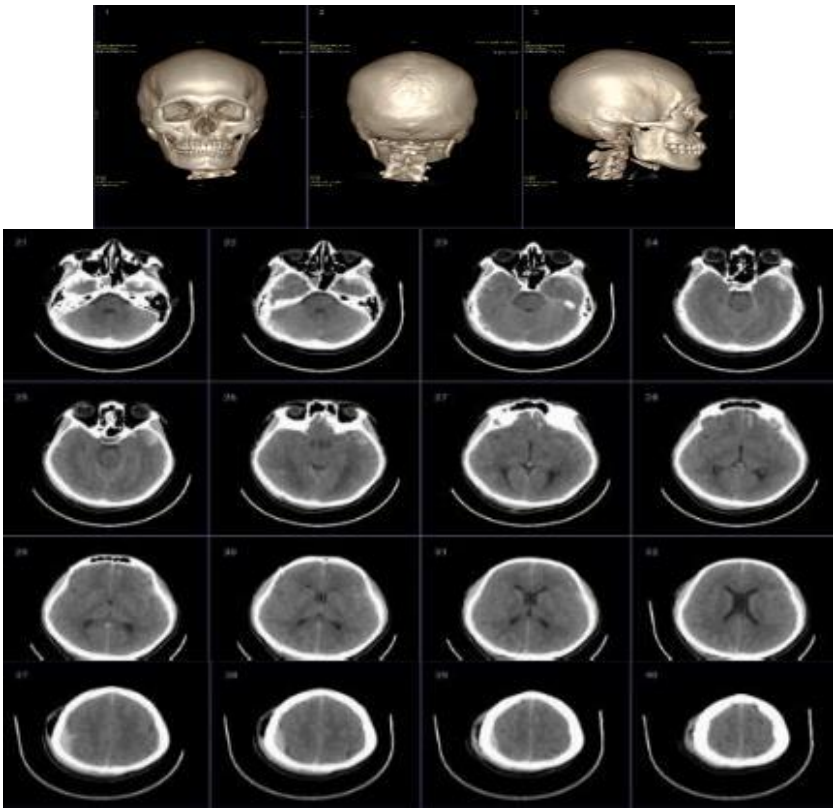

Figure 1. CT Scan Brain Trauma; CT brain trauma shows right temporal and left fronto-temporal SAH, right parietal EDH, right mastoid and bilateral ethmoidal and sphenoidal hemosinus, subgaleal hematoma and emphysema subcutis in the right parieto-occipital region

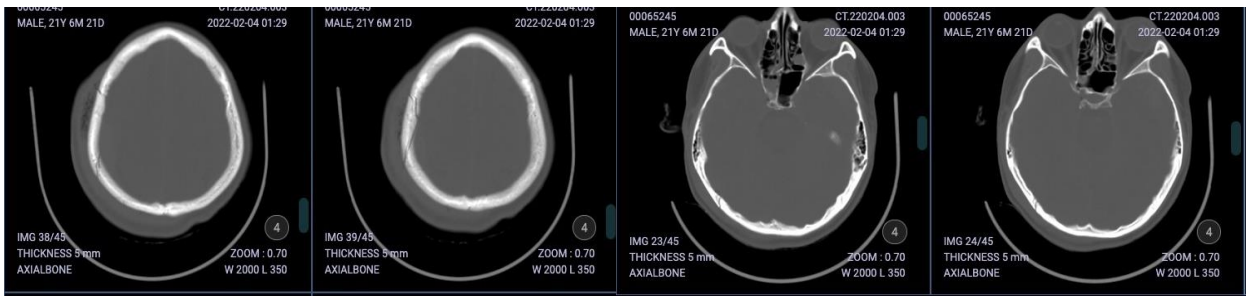

Figure 2. CT Scan Bone Window; CT scan bone window shows fracture of the os calvaria in the right parietal and right occipital region

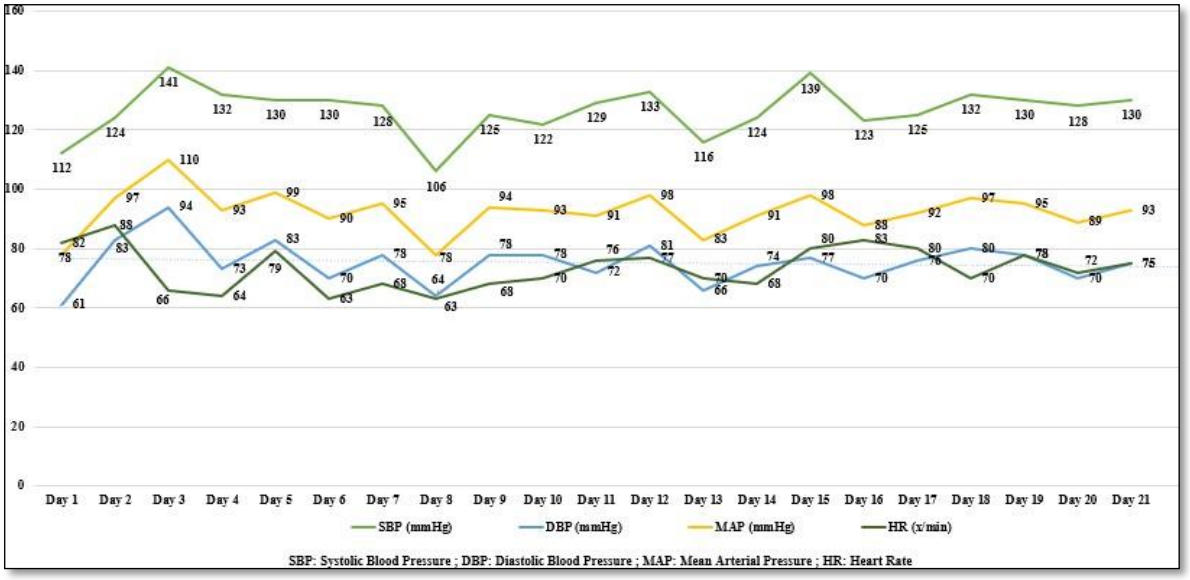

Figure 3. Graph of Patient Blood Pressure, Mean Arterial Pressure, and Heart Rate. The patient's vital signs were still within the normal range even though the patient's condition was initially unstable

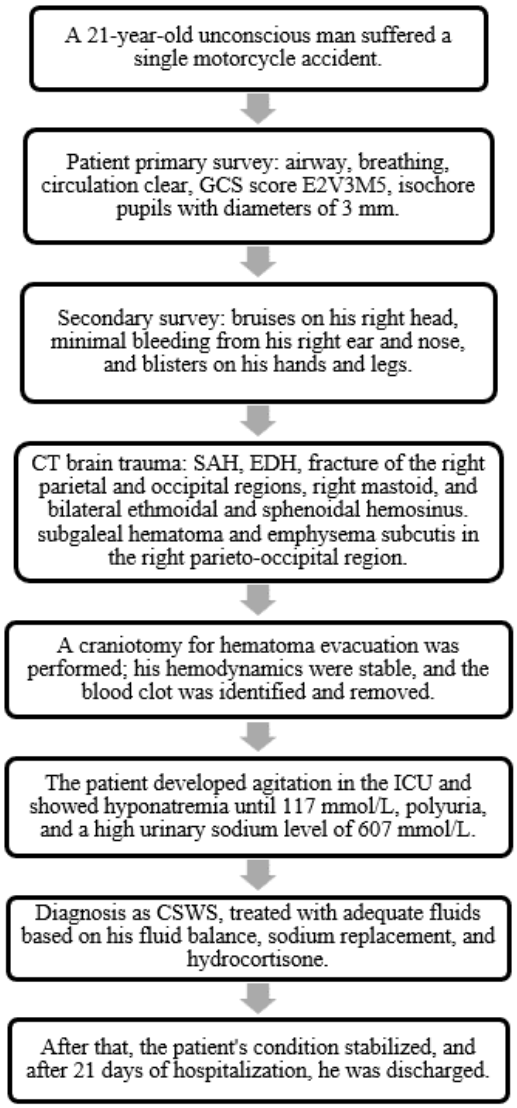

Figure 4. Patient Case Chronology