INTRODUCTION

Hyponatremia is a decrease in serum sodium of less than 135 mEq/L; it is the most common electrolyte imbalance, usually found as a complication of other medical conditions (e.g., heart failure, liver failure, renal failure, pneumonia, and cancer). Critically ill patients admitted to the hospital with neurological disorders frequently also presented with hyponatremia.¹

Cerebral Salt Wasting Syndrome (CSWS) is extracellular volume depletion caused by impaired renal sodium transport in patients with intracranial disease or trauma with normal kidney and thyroid function. In addition to symptomatic acute hyponatremia and dehydration, CSWS is also associated with symptomatic acute hyponatremia. This syndrome is still poorly understood, but it is known that defects in renal sodium transport led to decreased extracellular volume and cascade changes. The abnormalities in the proximal tubule cause excessive sodium loss.²

We reported and discussed the diagnostic approach and the management of a 66-year-old woman who experienced severe hyponatremia with a history of head trauma and was treated at Udayana University Hospital.

CASE DESCRIPTION

A 66-year-old female patient came to the hospital an hour after a sudden loss of consciousness. The patient had previously complained of severe headaches and frequent urination. Before being transported to the hospital, the patient complained of rigidity in her right and left hands, and her whole body shook violently for approximately 20 seconds. Her medical history included a history of head trauma 14 days before admission and vomiting once the next day.

On physical examination, there was a decrease in consciousness with GCS E2V2M5, with the poor orientation of people, place, and time. Blood pressure was 90/60 mmHg, heart rate was 100 beats per minute, respiratory rate was 20 beats per minute, patient’s temperature was 36.1°C, SpO2 was 98% at room air, and random blood sugar was 123 mg/dL. Positive neck stiffness was revealed during the head examination. Heart, lung, and abdomen examinations were within normal parameters. The patient’s neurological status confirmed positive neck stiffness without lateralization, normal motor strength in both extremities (value of 5), normal muscle tone, normal physiological reflexes, and absence of pathological reflexes.

Laboratory results during patient admission were the following: white blood cell count (WBC) of 17.11 x10³/µL, hemoglobin (Hb) of 10.7 g/dL, hematocrit (HCT) of 30.6%, platelets (PLT) of 298 x10³/µL, the random glucose level of 96 mg/dL, aspartate transaminase (AST) of 22 units/L, alanine transaminase (ALT) of 25 units/L, creatinine of 0.9 mg/dL, total bilirubin of 1.0 mg/dL, total protein of 6.6 g/dL, sodium of 117 mEq/L, potassium of 5.2 mEq/L, chloride of 98 mEq/L, and bicarbonate of 19 mEq/L.

On physical examination, the patient had a temperature of 36.5°C, respiratory rate of 18 breaths per minute, heart rate of 80 beats per minute, blood pressure of 120/80 mmHg, and SpO2 of 98% at room air. The patient was alert and oriented to time and place. The pupils were equal and reactive to light. The neck was hyperextended with no signs of neck stiffness. The heart sounds were normal with no murmurs, rubs, or gallops. The lungs were clear to auscultation. The abdomen was soft with normal bowel sounds. The extremities were warm to touch with normal muscle tone and strength. The patient complained of severe headaches and frequent urination.

On laboratory testing, the patient had a sodium level of 117 mEq/L, potassium level of 5.2 mEq/L, chloride level of 98 mEq/L, and bicarbonate level of 19 mEq/L. Blood glucose was 96 mg/dL. Hemoglobin was 10.7 g/dL, and hematocrit was 30.6%. Platelets were 298 x10³/µL. Creatinine was 0.9 mg/dL, and total bilirubin was 1.0 mg/dL. Total protein was 6.6 g/dL.

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Non-enhanced head CT scan revealed motion artifacts during the examination (Figure 2). The cerebral sulci and fissure of Sylvii were not widened, and the ventricular system and cisterns were not dilated. There were intact bone structures with no midline shift or abnormalities of the sella and para sellar. The conclusion gave the impression that there was no infarction, bleeding, edema, or fracture of the skull.

Initial diagnoses included severe hyponatremia, bacterial pneumonia, hypokalemia, and mild hypochromic microcytic anemia. The decreased consciousness and polyuria in this patient were suspected due to CSWS. She was admitted to the intensive care unit (ICU). The fluid of 0.9% sodium chloride and 3% sodium chloride in two separate lines was initiated, with the first IV line for normal 0.9% sodium chloride 1000 ml in one hour followed by 50 meq potassium chloride in the 500 ml 0.9% sodium chloride and the second line for 3% sodium chloride 12 drops per minute.

Therapy consists of cefoperazone 1 gram every 12 hours intravenously (iv), levofloxacin 750 grams iv, and hydrocortisone 100 mg every 12 hours iv initiated. The neurology department was consulted about the patient due to general motor tonic-clonic seizures. The neurologist administered citicoline 250 mg every 12 hours iv, paracetamol 1 gram every 8 hours via NGT, and diazepam 10 mg iv if needed with a rate < 5 mg/min.

On the second day of treatment, contact was inadequate with normal vital signs. She received normal saline 1500 ml in 24 hours, and the 3% sodium chloride infusion was discontinued. Potassium supplementation and other therapy were continued. The production of urine output on the second day was 3380 ml and a 24-hour urine sodium examination showed an increased urinary sodium level (410 mmoL/24 hours). The sodium plasma level was 111 mmoL/L (Table 1).

Her general condition on the third day of treatment was improved, with adequate contact. She could follow orders but was not able to speak. The vital signs were within normal limits. The urine output was 4770 ml in 24 hours. Potassium supplementation and 0.9% sodium chloride were discontinued on the third day. The patient got intravenous fluid of ringer’s lactate 1500 ml in 24 hours.

On the fourth day of treatment, contact was adequate, and she could engage in small communication. The vital signs were within normal limits. The amount of urine output was 5310 ml in 24 hours. On the fifth day of treatment, the patient’s condition was good, with adequate contact. The urine production was 7771 ml in 24 hours. Her general condition on the sixth day of treatment was good, with normal vital signs with a urine output of 9270 ml in 24 hours. 500 mg sodium salt tablets every 8 hours was initiated.

The patient was transferred from the ICU to the medical ward on the ninth day of admission. Contact was adequate, with normal vital signs. The urine output was 4570 ml in 24 hours. Antibiotics were discontinued. Treatment consisted of ringer’s lactate 1500 ml, and oral sodium supplementation was continued. The patient was suggested to drink 2 L of water per day. The urine output on day 14th was 3500 ml and decreased to 2080 ml in 24 hours on the twentieth day of admission. The serial electrolyte results were sodium of 134 mmoL/L and potassium of 3.0 mmoL/L, and the patient was discharged on the 21st day of the admission.

**DISCUSSION**

CSWS is defined as renal salt loss due to intracranial diseases that induce hyponatremia and decreased extracellular fluid volume. Alexandria Rudolph et al. showed that excessive urine output, hyponatremia, and natriuresis are the features of CSWS. Head trauma may cause changes in the hypothalamic-renal pathway, an imbalance in sympathetic output with reduced renal sympathetic activity, and probable direct injury to the anterior and posterior pituitaries.

According to the study by John K. Masaka et al., the primary treatment for CSWS is replacing water and sodium lost due to diuresis and natriuresis. For mild hyponatremia, 0.9% sodium chloride should be sufficient, and 3% sodium chloride should be administered for severe hyponatremia. Due to severe hyponatremia, we treat this patient with normal saline and 3% sodium chloride.
### Table 1. Serial blood gas analyses and electrolytes examination.

<table>
<thead>
<tr>
<th>Examination</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
<th>Day 8</th>
<th>Day 9</th>
<th>Day 10</th>
<th>Day 11</th>
<th>Day 12</th>
<th>Day 13</th>
<th>Day 14</th>
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<tbody>
<tr>
<td>Sodium (mmol/L)</td>
<td>127.7</td>
<td>127.7</td>
<td>130.5</td>
<td>134.4</td>
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<td>Potassium (mmol/L)</td>
<td>3.0</td>
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<td>Chloride (mmol/L)</td>
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<td>HCO3 (mmol/L)</td>
<td>111</td>
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<tr>
<td>Urine output (ml in 24 hours)</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
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Through 2 different IV lines in the hope that sodium can be corrected and euvolemia ensues. A study by Amine B et al. showed that administering mineralocorticoids/fludrocortisone at a twice-daily dose of 0.05-0.1 mg is associated with positive outcomes. Increased sodium reabsorption is caused by fludrocortisone’s direct action on renal tubules. Fludrocortisone on hyponatremia remains evident given the physiology of Na+ regulation by fludrocortisone’s direct action on renal tubules. Fludrocortisone on hyponatremia remains evident given the physiology of Na+ regulation. 

The management protocol was set to maintain blood sodium at >140 mmol/L, and 71 patients (placebo group: n=36, hydrocortisone group: n=35) entered the trial with various background variables. From day 0 through day 10, 300 mg of hydrocortisone or placebo was administered intravenously every six hours, every twelve hours on days 11 and 12, and days 13 and 14. The result is sodium excretion, and urine volume was significantly decreased in the hydrocortisone group; plasma osmolarity decreased to <280 mOsm/kg, whereas it stayed at around 290 mOsm/kg in the hydrocortisone group. Serum sodium remained >140 mmol/L in the hydrocortisone group and <140 mmol/L in the placebo group.

A case report by Tomotaka Tanaka et al. presented 79 years old Japanese woman with CSWS after hemorrhagic brain infarction. She showed hyponatremia in concert with polyuria complicated at the same time with the progress of hemorrhagic transformation on day 14 after admission, with serum sodium 134 mmol/L, urine osmolarity 454 Osm/kg H2O. The management was fluid balance control and substitution of sodium chloride. Extracellular volume assessment is a key factor in differentiating CSWS from SIADH. After treatment for 52 days, the serum Na level normalized in serum sodium 143 mmol/L, urine osmolarity 266 osm/kg H2O.

### CONCLUSION

We reported a case of a 66-year-old female with CSWS, the mechanism by which intracranial disease causes CSWS is still not fully understood. The management mainly focuses on the correction of intravascular volume depletion using 0.9% sodium chloride and hyponatremia, as well as on the replacement of ongoing urinary sodium loss, usually with IV hypertonic saline solutions. Fludrocortisone/mineralocorticoid that directly acts on the renal tubules to increase sodium reabsorption showed a favorable response in CSWS.

### CONFLICT OF INTEREST

We declare that there were no conflicts of interest in this study.
FUNDING

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AUTHOR CONTRIBUTION

All of the authors equally contributed to the study.

REFERENCES


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