SALT WASTING SYNDROME: SERIAL CASES

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ABSTRACT

Hyponatremia is one of the electrolyte disturbances that can be found in patients with central nervous system disorders, the most cases of hyponatremia are the Syndrome of Inappropriate Antidiuretic Hormone/SIADH compared to hyponatremia in Cerebral Salt Wasting Syndrome/CSWS and it is not uncommon to also occur in patients undergoing chemotherapy treatment without accompanying head abnormalities are referred to as Renal Salt Wasting Syndrome (RSWS). There is difficulty differentiating these two conditions due to almost the same clinical symptoms. Evaluation of volume status and monitoring of urate excretion fraction can help differentiate SIADH from RSWS and CSWS. Management requiring prompt diagnosis is essential to prevent severe hyponatremia from substantially causing cerebral edema and thereby reducing the risk of seizures. Fluid resuscitation therapy and sodium restoration are important. This case series report injured 2 CSWS patients after head trauma with different treatments and hyponatremia due to cisplatin induced RSWS.

KEYWORDS

Hyponatremia, CSWS, RSWS, SIADH

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INTRODUCTION

The electrolyte imbalance hyponatremia, characterized by a sodium level below 135 mEq/L, is common worldwide, affecting 10% of hospitalized patients and serving as an independent risk factor for morbidity and mortality. The mechanisms underlying hyponatremia are highly complex, causing confusion and misunderstanding among healthcare providers dealing with electrolyte imbalances.
Comprehensive understanding of the pathophysiology is crucial for initiating successful treatment, as treatment for hyponatremia is driven by its underlying mechanisms (Rudolph & Gantioque, 2018).

The prevalence of hyponatremia in head injuries and other neurological diseases such as SAH, SDH, EDH, or intracranial infections is 15-30% (Rudolph & Gantioque, 2018). The incidence of CSWS in head injuries ranges from 0.8% to 34.6% (Hoai et al., 2020). Hyponatremia is observed in cancer patients, varying from 4% to 44%, depending on the type of cancer, pre-chemotherapy hyponatremia, and the chemotherapy regimens used (Ezoe et al., 2018). Platinum-based drugs are the most common cause of hyponatremia, accounting for 11.9%, compared to non-platinum-based drugs (Ezoe et al., 2018). The incidence of RSWS after cisplatin administration is <1%, and this condition may be caused by underdiagnosis or misdiagnosis, such as SIADH (Hamdi et al., 2010).

This condition is often challenging to diagnose because it relies on exclusion. SAIDH, CSWS, and RSWS share common features that make diagnosis difficult. Determining the fluid volume status is a crucial indicator to differentiate these conditions. A new approach is to determine the fractional excretion of urate. This result consistently and accurately distinguishes SIADH from CSWS and RSWS (Maesaka et al., 2007) (Maesaka et al., 2007). The goal of this case series is to differentiate the three aforementioned conditions with different salt-wasting causes, so that appropriate treatment can be applied.

RESEARCH METHOD

CASE

Case 1: A 61-year-old female with no chronic illnesses was referred from a previous hospital due to deteriorating consciousness and unresolved electrolyte disturbances. The patient had received treatment at the previous hospital for 21 days with the main complaints of seizures and during the treatment, the patient experienced polyuria, electrolyte disturbances, and worsening consciousness, prompting the referral. The patient had a history of a motor vehicle accident three days before the onset of seizures and had undergone surgery for a mandibular fracture. A non-contrast CT brain scan revealed mild cerebral edema.

Laboratory results showed normal blood counts, thyroid function, and kidney function. The serum sodium level was 121 mmol/L, serum potassium was 2.26 mmol/L, chloride was 68.8 mmol/L, corrected calcium was 7.4 mg/dL, random blood sugar was 164 mg/dL, and albumin was 2.43 g/dL. Arterial blood gas analysis revealed a pH of 7.69, pCO2 of 39 mmHg, pO2 of 240 mmHg, BEecf of 27 mmol/L, and HCO3 of 47 mmol/L. Serum uric acid was 1 mg/dL, and serum osmolality was 254 mOsm/kg. Urinalysis showed a urine pH of 8.6, a specific gravity of 1.003, the presence of uric acid crystals, and a urine osmolality of 240.84 mOsm/kg, with uric acid in urine at 7.74 mg/dL (normal range: 2.7-5.4 mg/dL) and urine creatinine at 7.28 mg/dL, with a urate fractional excretion of 44%. The laboratory course during treatment is attached (Table 1 and Figure 1). An EKG revealed sinus rhythm at 120 beats per minute. A bedside echo showed an estimated right atrial pressure of 3 mmHg. An MRI of the head without contrast, 26 days after the motor vehicle accident, revealed a non-contrast CT brain scan revealed mild cerebral edema.
accident, revealed multiple hyperintensities in the lateral periventricular area, deep white matter in the right and left corona radiata, and right and left frontal lobes, suggesting small vessel ischemic changes.

Table 1. Serum and Urine Osmolality Data

<table>
<thead>
<tr>
<th>Day **</th>
<th>11/2*</th>
<th>25/2</th>
<th>27/2</th>
<th>14/3</th>
<th>Refer value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>13</td>
<td>27</td>
<td>29</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>Serum osmolality</td>
<td>-</td>
<td>254</td>
<td>271</td>
<td>-</td>
<td>282-295 mOsm/kg</td>
</tr>
<tr>
<td>Urine osmolality</td>
<td>-</td>
<td>240.8</td>
<td>-</td>
<td>373.3</td>
<td>500-800 mOsm/kg</td>
</tr>
<tr>
<td>Temporary urine sodium</td>
<td>94</td>
<td>103.2</td>
<td>-</td>
<td>133</td>
<td>54-150 mmol/L</td>
</tr>
<tr>
<td>Urinary potassium</td>
<td>-</td>
<td>14.7</td>
<td>-</td>
<td>17.42</td>
<td>20-80 mmol/L</td>
</tr>
<tr>
<td>Urine sodium</td>
<td>329</td>
<td>641</td>
<td>-</td>
<td>-</td>
<td>30-300 mmol/24hours</td>
</tr>
<tr>
<td>Urine potassium</td>
<td>-</td>
<td>91.92</td>
<td>-</td>
<td>-</td>
<td>25-100 mmol/24hours</td>
</tr>
</tbody>
</table>

*Previous hospital
**Days post-accident

- No Data

Image 1: Urine Volume and Serum Sodium Graph during Treatment at Prof IGNG Ngoerah Hospital
The (X) marks indicate the initiation of hydrocortisone therapy.

Image 2: Brain MRI without Contrast

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The patient was diagnosed with CSWS, post mild head injury with post-operation miniplate for left mandibular fracture and small vessel ischemia. The first emergency therapy involved administering isotonic NaCl 0.9% at 30 cc/kg body weight, with the goal of maintaining euvolemia while monitoring central venous pressure (CVP) and eRAP (estimated of right atrial pressure). After hydration, the serum sodium levels improved from 120 mmol/L to 130 mmol/L. Additionally, the patient received concurrent therapy for severe hypokalemia, with a maximum dose of 100 meq/hour via central venous catheter (CVC). The target potassium range during treatment was 3.5 to 5.1 mmol/L. The patient was aimed to achieve euvolemia during the first 3 days of treatment, followed by fluid restriction, where the total fluid given was 80% of the 24-hour urine output. Fluid restriction continued for 11 days, but as the patient's polyuria did not improve, it was decided to provide mineralocorticoid therapy (fludrocortisone). Due to the unavailability of the medication, the patient received intravenous hydrocortisone at a dose of 100 mg every 8 hours for a week, with a tapering down of the dose on the 5th day of therapy, resulting in improvements in urine volume and controlled electrolyte and blood sugar levels.

Case 2: A 28-Year-Old Male with Headache and Excessive Urination

A 28-year-old male presented with complaints of headache, nausea, and vomiting. The patient had a motor vehicle accident 6 days ago. He had no history of seizures, fever, cough, or shortness of breath. The patient reported feeling weak and having trouble sleeping due to frequent urination and excessive thirst. He mentioned urinating 8-12 times a day, and his daily water intake, which was usually 3 liters, had increased to 6-7 liters. The patient had no known chronic illnesses.

Physical examination revealed a GCS score of 15, normal vital signs, complete blood count, and normal kidney function. His blood glucose level was 123, sodium was 112, potassium was 3.69, calcium was 7.6, chloride was 84.2, and albumin was 3.54. Serum osmolality was measured at 233 mOsm/kg. Urine osmolarity 11 days after the accident was found to be 303.29 mOsm/kg H2O, with a serum sodium of 114.9 mmol/L, and a 24-hour urine sodium of 551.52 mmol. A CT scan of the brain conducted 6 days after the accident showed subdural hematoma in the posterior falx cerebri, a hypodense lesion with CSF density in the left frontal temporal region, associated with basal cistern suspected to be an arachnoid cyst or porencephaly, and cerebral edema.

The patient was diagnosed with mild head injury, interhemispheric subdural hematoma, post-concussion syndrome, and polyuria suggestive of CSW. The patient received fluid correction therapy with NaCl 0.9% at a volume of 80% of urine output. By the 6th day of treatment, the patient showed improvements in urine volume and serum sodium levels.

Table 2: Electrolyte Data History

<table>
<thead>
<tr>
<th>Day**</th>
<th>9/4*</th>
<th>15/4</th>
<th>16/4</th>
<th>17/4</th>
<th>18/4</th>
<th>19/4</th>
<th>21/4</th>
<th>Refer value (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>142</td>
<td>112</td>
<td>113</td>
<td>108</td>
<td>109</td>
<td>111</td>
<td>126</td>
<td>137</td>
</tr>
</tbody>
</table>
Table 3: Serum and Urine Osmolarity Data

<table>
<thead>
<tr>
<th>Day**</th>
<th>9/4*</th>
<th>15/4</th>
<th>19/4</th>
<th>20/4</th>
<th>Refer value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum osmolarity (mOsm/kg H2O)</td>
<td>294</td>
<td>233</td>
<td>282</td>
<td>-</td>
<td>282-295</td>
</tr>
<tr>
<td>Urine osmolarity (mOsm/kg H2O)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>303,2</td>
<td>500-800</td>
</tr>
<tr>
<td>Current urine sodium (mmol/L)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>114</td>
<td>54-150</td>
</tr>
<tr>
<td>Current urine potassium (mmol/L)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>7.73</td>
<td>20-80</td>
</tr>
<tr>
<td>24 hour urine sodium (mmol/24 hour)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>551</td>
<td>30-300</td>
</tr>
<tr>
<td>24 hour urine potassium (mmol/24 hour)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>25-100</td>
</tr>
</tbody>
</table>

Explanation:
* The day the patient had the accident, and the patient was not hospitalized (the first day after the accident).
** Post-accident day

Image 3: Urine Volume and Serum Sodium Graph during Treatment at Prof IGNG Ngoerah Hospital
Case 3: A 34-Year-Old Male Undergoing Chemotherapy for Advanced Osteosarcoma

A 34-year-old male was undergoing chemotherapy with Cisplatin and Doxorubicin series I due to a diagnosis of advanced Osteosarcoma. During the chemotherapy, the patient did not complain of nausea or diarrhea. Three days after receiving Cisplatin, the patient reported feeling weak and experiencing auditory hallucinations. There were no seizures or changes in consciousness observed in the patient. The patient denied complaints of shortness of breath, chest pain, diarrhea, or vomiting. Vital signs were normal, and there were no neurological deficits.

In the laboratory, 3 days after chemotherapy, the patient's sodium level was measured at 114 mmol/L, compared to a pre-chemotherapy sodium level of 134 mmol/L. Complete blood count, kidney function, and electrolytes were all within normal limits.

Five days after chemotherapy, the urine output increased (5500 ml/24 hours), urine osmolarity was low (194.99 mOsm/kg), and urinary sodium concentration was 487.85 mmol/24 hours with a urate fraction of 18.7%, which had been preceded by polyuria 3 days earlier. (Image 5)

The patient was suspected of having RSWS (Renal Salt-Wasting Syndrome) and was administered 80% of the total urine production in NaCl 0.9% solution over 3 days. The patient did not experience shock during this time, and post-therapy evaluation showed an increase in sodium to 127 with a urine output of 2200 ml/24 hours. There was an improvement in urinary sodium concentration to 94.6 mmol/24 hours and a urate fraction of 16.2%, although urine osmolarity remained low but showed improvement, measuring 250.66 mOsm/kg. The patient also experienced hypokalemia after 1 day of polyuria and improved after being given KCL 25% therapy.
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Note:
X: Pre-Chemotherapy Sodium Level

Below is a table summarizing the course of the disease and therapy for the three cases.

<table>
<thead>
<tr>
<th>Case Onset</th>
<th>Symptoms and sign</th>
<th>Head CT/ MRI</th>
<th>Timing development of CSWS</th>
<th>Treatment</th>
<th>LOS</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>61 years old female: mandibula fracture and cerebral edema. 3 days after the motor accident the patient complained.</td>
<td>- Decreased of consciousness - Seizure - Hyponatremia - Hypokalemia - polyuria</td>
<td>MRI of the head without contrast 26 days after motor accident small vessel ischemic change.</td>
<td>3 days</td>
<td>Saline hydration and hydrocortisone IV</td>
<td>40 days (include previous hospital)</td>
<td>improved</td>
</tr>
<tr>
<td>28 year old male: 6 days after the accident symptoms appeared and he was hospitalized</td>
<td>- Seizure - Hyponatremia - Polyuria</td>
<td>CT scan: SDH, edema cerebri</td>
<td>6 days</td>
<td>Saline hydration</td>
<td>7 days</td>
<td>improved</td>
</tr>
<tr>
<td>34 years old Male: fatigue and hallucination 2 days after Chemotherapy</td>
<td>- hallucination - Hyponatremia - hypokalemia - polyuria</td>
<td>not evaluated</td>
<td>2 Days</td>
<td>Normal Saline Hydration</td>
<td>8 days</td>
<td>Improved</td>
</tr>
</tbody>
</table>
RESULT AND DISCUSSION

Hyponatremia is a common complication in central nervous system disorders, especially in cases of head injuries and cerebrovascular disorders. Dehydration is often associated with hyponatremia, which frequently occurs in the context of Cerebral Salt-Wasting Syndrome (CSWS) compared to Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) (Chaudhary et al., 2016) (Tanaka et al., 2014) (Burst, 2019). CSWS is a condition involving the disruption of sodium and water homeostasis, resulting in excessive polyuria and sodium excretion in the urine, often accompanied by hypovolemia and normal kidney function (Chaudhary et al., 2016) (Taylor et al., 2017). We can observe the CSWS condition in the first and second cases. In the third case, hyponatremia, polyuria occurred without head trauma, following cisplatin chemotherapy. The type of cancer and pre-chemotherapy sodium levels (<138 mEq/L) are independent risk factors for post-cisplatin hyponatremia. Hyponatremia incidents occur after cisplatin administration in Small Cell Lung Cancer (SCLC), prostate cancer, hepatocellular carcinoma, and pancreatic cancer. Cisplatin-induced hyponatremia not associated with brain disease is referred to as RSWS (Hatakeyama et al., 2019).

Cisplatin has dose-dependent nephrotoxic effects that cause electrolyte imbalance, renal tubular acidosis, acute kidney injury with glomerular filtration impairment, Fanconi syndrome, nephrogenic diabetes insipidus, and renal salt-wasting syndrome (RSWS). Cisplatin damages the proximal tubules, the primary site for sodium and water reabsorption, resulting in natriuresis with increased urinary sodium excretion. To prevent a decline in intravascular volume, antidiuretic hormone (ADH) is released along with aldosterone to conserve salt and water. Cisplatin also decreases the expression of aquaporin 2 and 3, which regulate water permeability in the distal nephron, and reduces the activity of ENaC, leading to natriuresis (Shimizu et al., 2019) (Oh et al., 2014). Cisplatin also binds to and inhibits the sodium transporter protein Na+/K+-ATPase, causing diuresis and salt excretion (Pham et al., 2017) (Soares et al., 2020).

The pathogenesis of CSWS, SIADH, and RSWS is quite distinct. Natriuretic peptides like atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), C-type natriuretic peptide (CNP), and dendroapsis natriuretic peptide (DNP) play roles in CSWS. To date, BNP is considered the primary mediator of CSWS. An increase in plasma volume triggers atrial wall dilation, stimulating a sympathetic response or an increase in angiotensin II or endothelin, which can lead to the release of these peptides. A direct relationship between ANP or BNP and intracranial pressure has been established, indicating that the release of these peptides is a compensatory mechanism, resulting in renal salt wasting to maintain constant pressure, reducing the extreme elevation of intracranial pressure and the tendency for vasospasm in cases such as subarachnoid hemorrhage (Cui et al., 2019).
In CSWS, sympathetic activity and dopamine release decrease, resulting in natriuresis and increased release of natriuretic peptides (BNP) from the hypothalamus due to brain damage and renin-aldosterone system dysfunction. An increase in BNP serves as a protective mechanism against elevated intracranial pressure, inhibiting sympathetic outflow, the renin-aldosterone system, and the production of vasoconstrictor peptides, which decreases aldosterone effectiveness and significantly impairs the kidney’s ability to reabsorb sodium. Thus, CSWS is characterized by high urinary sodium concentration with urinary sodium > 20 mmol/L, urinary osmolality ratio > 100 mOsm, polyuria, postural hypotension, and low central venous pressure, leading to extracellular fluid depletion (Taylor et al., 2017).18,19 (Figure 7)

Figure 6. Mechanism of CSWS (Yee et al., 2010)

In SIADH, there is an increased sensitivity of the kidneys to ADH or inappropriate ADH secretion, leading to dilutional hyponatremia. FEurate examination becomes important in differentiating between CSWS and SIADH in patients who have not undergone CVP placement for patient volume status determination. In CSWS, there is a high uric acid excretion in the urine, with a urate excretion fraction of > 10%, and once hyponatremia is resolved, FEurate remains high with serum uric acid levels less than 4 mg/dL. In contrast to SIADH, where urate excretion in urine decreases even if serum sodium is within normal limits (Cui et al., 2019) (Palmer, 2000).20
Below is a table that explains the differences between CSWS, SIADH, and RSWS since they have different diagnostic criteria. Diagnosis necessitates an assessment and monitoring of sodium, water loss, and extracellular volume. The main differences in these disorders lie in plasma volume, sodium excretion, and urate excretion fraction.19,21 (Table 2)

Table 2: Clinical and Laboratory Differences in SIADH, CSWS, and RSWS

<table>
<thead>
<tr>
<th></th>
<th>SIADH</th>
<th>CSWS</th>
<th>RSWS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td>Normal/increased</td>
<td>Hypovolemia</td>
<td>Hypovolemia</td>
</tr>
<tr>
<td>CVP</td>
<td>Normal/slightly increased</td>
<td>Decrease</td>
<td>Decrease</td>
</tr>
<tr>
<td>Plasma sodium</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Serum uric acid concentration</td>
<td>Decreased or normal</td>
<td>Decrease</td>
<td>Decrease</td>
</tr>
<tr>
<td>Urate excretion fraction</td>
<td>Decreases due to volume expansion</td>
<td>Increases due to urinary losses and will remain high after sodium correction</td>
<td>Same with CSWS</td>
</tr>
<tr>
<td>Urine volume</td>
<td>Normal/decreasing</td>
<td>Increase</td>
<td>Increase</td>
</tr>
<tr>
<td>Urine sodium concentration</td>
<td>&gt;30mmol/L</td>
<td>&gt;&gt; 30mmol/L</td>
<td>&gt;&gt;30mmol/L</td>
</tr>
</tbody>
</table>
Hyponatremia resulting from traumatic brain injury often occurs between day 3 and 2 weeks after the traumatic event (Palmer, 2000) (Cui et al., 2019). Skull fractures accompanied by cerebral contusion and subdural hematoma with fluid intake exceeding 9 liters between the first day and the third day are risk factors for the development of hyponatremia. Cerebral contusion and brain swelling due to trauma disrupt and damage the neuroendocrine function of the hypothalamus and pituitary system, leading to SIADH/ or CSWS and triggering central hyponatremia (Cui et al., 2019).

Before establishing a diagnosis of CSWS, RSWS, and SIADH, the causes of hyponatremia must first be ruled out, such as the use of diuretics, adrenal insufficiency, hypothyroidism, or congestive heart failure. Misra et al. consider that there should be at least 2 out of 4 criteria for hyponatremia in CSWS, including: (1) clinical hypovolemia such as hypotension, dry mucous membranes, tachycardia, or postural hypotension; (2) laboratory evidence of dehydration, such as an increase in hematocrit, hemoglobin, serum albumin, or blood urea nitrogen; (3) negative fluid balance as found by intake and/or weight loss charts; (4) CVP < 6 cmH2O (Cui et al., 2019). (Table 2) (Figure 7)
Rapid diagnosis of CSWS and RSWS is crucial in management as it requires volume resuscitation and sodium restoration, which is different from SIADH where fluid restriction is the key treatment. Correct diagnosis and correction are crucial to prevent severe hyponatremia, which can substantially worsen cerebral edema and increase the risk of seizures (Taylor et al., 2017). The primary therapy in managing CSWS and RSWS is the replacement of sodium and water lost due to natriuresis. Patients with CSWS often exhibit significant intravascular volume depletion, and the total sodium deficit is at least 2 mmol/kg body weight. In cases of hypovolemia, the initial strategy is to administer normal saline with the goal of achieving intravascular volume sufficiency. Sodium correction follows once euvolemia is achieved. Hypertonic saline therapy is administered if sodium levels remain below 125 mEq/L, but it should be done slowly, with a target increase of no more than 10 mmol/L within 24 hours to prevent pontine myelinolysis (Pratiwi & Mahadewa, 2019).
Another treatment option is the administration of mineralocorticoids aimed at increasing serum sodium and intravascular volume. One such option is fludrocortisone, which should be started when the diagnosis of CSWS is confirmed and continued until sodium concentration returns to normal and intravascular volume concentration stabilizes. This therapy is considered if fluid and sodium replacement therapy has been conducted effectively but has not yielded satisfactory results. Fludrocortisone works on the renal tubules to enhance sodium reabsorption but may have secondary effects such as hypokalemia, pulmonary edema, and long-term hypertension. Therefore, the use of fludrocortisone is only considered when fluid and salt replacement cannot manage excessive natriuresis (Pratiwi & Mahadewa, 2019).

In a randomized controlled trial involving 71 SAH patients, 36 patients received a placebo, while the rest received hydrocortisone for 10 days, followed by a gradual reduction in the dosage. This treatment can prevent natriuresis and hypovolemia in the short term and has no long-lasting side effects (Katayama et al., 2007). The treatment of RSWS is similar to that of CSWS patients, but the administration of antioxidants such as N-acetylcysteine, vitamin C, and vitamin E should be considered as renal protectors against cisplatin administration, although clinical trials in humans are limited (Russo et al., 2021).

Cisplatin has dose-dependent nephrotoxic effects that cause electrolyte disturbances, renal tubular acidosis, acute kidney injury with decreased glomerular filtration, Fanconi syndrome, nephrogenic diabetes insipidus, and renal salt-wasting syndrome (RSWS). Cisplatin damages the proximal tubules, the primary site for sodium and water reabsorption, leading to natriuresis with increased urinary volume and urinary sodium. To prevent intravascular volume reduction, ADH is secreted along with aldosterone in an effort to conserve salt and water. Cisplatin is also known to reduce the expression of aquaporin 2 and 3, which regulate water permeability in the distal nephron, and decrease the activity of ENaC, resulting in natriuresis (Shimizu et al., 2019) (Oh et al., 2014). Cisplatin also binds to and inhibits the sodium transporter protein Na+/K+-ATPase, causing diuresis and salt excretion (Pham et al., 2017) (Soares et al., 2020).

Two out of the three cases experienced hypokalemia (cases 1 and 3). According to the author, the hypokalemia in the first case can be explained by the decrease in mineralocorticoid production due to BNP response to the adrenal medulla, which leads to unstimulated aldosterone and renin production, resulting in excessive urine production and prolonged volume depletion that causes metabolic alkalosis and recurring hypokalemia in the first case. In the third case, hypokalemia can be attributed to the use of cisplatin chemotherapy, which causes potassium loss through the inhibition of the Na+/K+-ATPase pump, partly due to decreased magnesium concentration following platinum administration. Unfortunately, magnesium levels were not tested in this case.
CONCLUSION

Hyponatremia is a common electrolyte disorder found in patients with malignancies, with or without chemotherapy, as well as in head trauma cases. It is essential to identify the intravascular fluid volume status to classify the cause of hyponatremia properly and provide the right treatment. This report presented a series of salt-wasting syndrome cases with different etiologies, including head trauma and platinum-based chemotherapy. It is crucial to differentiate between CSWS, RSWS, and SIADH because an incorrect diagnosis of hyponatremia in these three cases can lead to erroneous treatment and result in more severe complications. Adequate intravascular volume and sodium concentration are the goals of managing patients with CSWS, which can be achieved through a combination of isotonic saline, hypertonic saline, mineralocorticoids, and, in the case of cisplatin, renal protective measures.

REFERENCES


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