Cerebral Salt Wasting Syndrome in a Patient with Subarachnoid Hemorrhage: 
A Case Report
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ABSTRACT

Hyponatremia is a common condition following acute brain injury, the two main causes of which are the Syndrome of Inappropriate Antidiuretic Hormone (SIADH) and the Cerebral Salt Wasting Syndrome (CSWS). Differentiating the two conditions is important due to the divergent management approach. We present a case of hyponatremia in a 36-year-old female attributed to CSWS after subarachnoid hemorrhage and endovascular coiling of cerebral aneurysm. There are no established guidelines for the diagnosis of CSWS thus, a high degree of suspicion and accurate assessment of the volume status with clinical and objective parameters is essential in differentiating it from SIADH to avoid catastrophic complications. Aggressive salt and fluid management, together with fludrocortisone administration, was successfully used to achieve hemodynamic stability and prevent further neurologic deterioration in this case.

Key words: hyponatremia, cerebral salt wasting, syndrome of inappropriate anti-diuretic hormone, fludrocortisone

INTRODUCTION

Hyponatremia is a common condition following acute brain disorders. It occurs in more than 50% of patients with subarachnoid hemorrhage (SAH), with differing onset during hospitalization. It is associated with an increased length of hospital stay and seizure occurrence.\(^1,2\)

Previous reports point to Cerebral Salt Wasting Syndrome (CSWS) as the most common cause of the electrolyte imbalance after SAH, however current retrospective studies have identified Syndrome of Inappropriate Antidiuretic Hormone release (SIADH) as the more common etiology of hyponatremia.\(^1,2\)

Differentiating these two conditions is important for the contrasting management; CSWS requires vigorous salt replacement to compensate for the renal salt wasting whereas SIADH needs fluid restriction as excessive antidiuretic hormone is the primary cause of the relative water excess.\(^3\)

Unfortunately, both conditions can present similarly in terms of clinical presentation, and laboratory features such as a low serum osmolality, high urine osmolality, and a high urine sodium level.\(^4,5\) The main difference is the extracellular fluid volume state; SIADH presents with either euvoletic or hypervolemic state, in contrast to the volume depleted state in CSWS.
We present a case of Cerebral Salt Wasting Syndrome in a 36-year-old female that occurred after coiling of cerebral aneurysm post subarachnoid hemorrhage event, highlighting the importance of differentiating it with SIADH.

**CASE PRESENTATION**

This is a case of a 36-year-old Filipino woman who presented at the Emergency room due to a transient, fifteen second loss of consciousness following a sudden onset of severe generalized headache with associated vomiting episodes. Initial examination showed: stable vital signs with unremarkable regional physical examination findings; neurologic examination - Glasgow coma score of 15, supple neck with no nuchal rigidity, and no sensory or motor deficits; laboratory examination revealed normal hemogram, blood electrolytes and other chemistries.

Cranial computerized tomography angiogram (CTA) was done after findings of subarachnoid hemorrhage on plain scan. CTA showed a saccular aneurysm in the left suprACLinoID internal carotid artery near its bifurcation with the MI segment of the left middle cerebral artery. The patient was started on medical decompression therapy while preparing for immediate cerebral endovascular intervention. Intraoperative findings noted a 1.8 mm x 1.2 mm left anterior choroidal artery aneurysm near the branching of the anterior choroidal artery from the left internal carotid artery. Coil embolization was performed, achieving occlusion of 95% of the aneurysm. The immediate postoperative period was generally uneventful; the patient was stable with no appreciated sensory or motor deficits but with a persistent generalized headache.

Succeeding days of hospitalization showed the patient having persistent polyuria, averaging more than five liters in a 24-hour period during the 6th hospital day, despite discontinuation of mannitol (Figures 1 and 2).

![Figure 1. Graph of serum sodium vs 24-hour sodium intake during hospitalization.](image-url)
Diabetes insipidus (DI) was initially considered as the possible cause of polyuria, however the presence of an inappropriately concentrated urine in the background of hypoosmolar, hyponatremic state is not consistent with a diagnosis of DI and is more compatible with both SIADH and CSWS. However, due to the apparent clinical euvoletic state of the patient, SIADH was initially entertained as the more appropriate diagnosis, thus fluid restriction was started.

During the 7th and 8th hospital days, the serum sodium started to increase in response to the reduced fluid load given to the patient. However, during the 9th hospital day, the patient developed increasing dysphasia and agnosia; she became hemodynamically unstable, with hypotensive episodes at 80/60 responsive to intravenous fluid resuscitation. Cranial non-contrast magnetic resonance imaging was done which showed an acute infarct in the left parietal lobe. A central venous line was also inserted revealing initial venous pressure readings of 3-4 cm. The diagnosis was reconsidered to a possible Cerebral Salt Wasting Syndrome due to the persistent polyuria and computed negative salt and fluid balance, the latter of which possibly producing the hemodynamic instability due to volume contraction.

The patient was then managed with aggressive intravenous saline infusion, targeted towards restoring salt and fluid balance; these measures stabilized the patient’s hemodynamics thereby preventing further progression of her neurologic condition. Thereafter, the salt and fluid balance was maintained by corresponding modification in oral sodium intake (diet and sodium chloride tablet supplementation) to match the estimated daily sodium loss. Due to persistent polyuria and increasing saline requirement, fludrocortisone was started on the 11th hospital day at an initial dose of 100 mcg/day and subsequently increased to 200 mcg/day on 13th hospital day. Succeeding days showed progressive decrease in urine output and sodium requirement, thus the dosage was tapered down and maintained at 100 mcg/day before discontinuing it after two weeks.

On her follow up, after a month of discontinuing fludrocortisone and sodium supplementation, the patient remained hemodynamically stable with no neurologic deficits; her salt and fluid balance was
also maintained with a regular diet.

**DISCUSSION**

In literature, the two most common causes of hyponatremia occurring after subarachnoid hemorrhage are SIADH and CSWS. Previously, studies done on hyponatremic patients post SAH identify CSWS as the more common etiology; however, two large recent prevalence studies made by Kao and Sherlock et al., demonstrate that SIADH is the most common cause of hyponatremia in SAH patients.\(^1,6\)

Distinguishing between these two conditions is therapeutically important due to the contrasting approach to management; fluid restriction is employed for SIADH while vigorous sodium and fluid replacement corrects the volume contraction and hyponatremia in CSWS.\(^4\) As demonstrated in our case, catastrophic complications such as cerebral vasospasm leading to cerebral infarction may occur after coiling; this condition can be triggered and or aggravated by hemodynamic instability produced by volume depletion. Correct identification and appropriate management of co-morbid conditions associated with brain injury problems like hyponatremia is therefore of paramount importance to prevent possible catastrophic events. Identifying one over the other may prove to be difficult due to the many similarities in the presentation of both SIADH and CSWS.

Currently, there are no consensus criteria for the diagnosis of CSWS.\(^3\) The only key difference between CSWS and SIADH is the presence of volume depletion, which is characteristically found in CSWS.\(^5\) Clinically, extracellular fluid volume loss can be manifested by symptoms such as weakness, light-headedness, and syncope; and physical findings which include orthostatic hypotension, dry mucous membranes, and tachycardia.\(^7\) However, these findings have both low sensitivity and specificity.\(^8\) Another option to determine volume status is via invasive hemodynamic monitoring, demonstrated as low pulmonary capillary wedge pressure or central venous pressure.\(^2,3,9\) During the first few days of hospitalization, our patient appeared clinically euvoletic, leading to an initial diagnosis of SIADH. However, as noted during the succeeding hospital days, the persistent polyuria, negative fluid balance, and episodes of hypotension together with low CVP values (<4), led us to reconsider the diagnosis which is more consistent with CSWS. Other proposed distinguishing characteristics are shown in Table 1.

### Table 1. Characteristics of the Syndrome of Inappropriate Antidiuretic Hormone (SIADH) and Cerebral Salt Wasting Syndrome (CSWS)

| Signs of dehydration on physical examination | Variably present | Absent |
| Central venous pressure | \(\downarrow\) | \(\uparrow\) or normal |
| Plasma volume | \(\downarrow\) | \(\downarrow\) or normal |
| Sodium balance | Negative | Variable |
| Fractional sodium excretion | \(\uparrow\uparrow\) | \(\uparrow\) or normal |
| Hematocrit\(^a\) | \(\uparrow\) | Unchanged |
| Plasma urea/creatinine ratio | \(\uparrow\) | Normal |
| Plasma potassium concentration | \(\uparrow\) or normal | \(\downarrow\) or normal |
| Serum uric acid concentration | \(\downarrow\) or normal | \(\downarrow\) |

| \(\uparrow\) Increase, \(\downarrow\) Decrease |
| \(^a\)Hematocrit does not differentiate postoperatively |

Delayed neurological deterioration is a common sequela after SAH; this includes cerebral vasospasm, hydrocephalus and seizures.\(^10\) Cerebral vasospasm complicates approximately two-thirds of patients who survive their initial episode or who were successfully treated surgically or endovascularly.\(^11\) Vasospasm can be asymptomatic; symptoms usually develop when cerebral blood flow falls below a certain ischemic threshold. This can be the net result of vasoconstriction, impaired autoregulation, and
inadequate intravascular volume. The condition usually develops four to nine days following SAH, often heralded by worsening headache and increasing blood pressure, eventually progressing into confusion and a decreased level of consciousness with focal neurologic deficits. In CSWS, the renal salt wasting often disturbs the delicate balance during the management of SAH; the decrease in plasma volume could potentially worsen cerebral blood flow. This was demonstrated in our patient while undergoing fluid restriction during the 7th to the 9th day of hospitalization; the ischemic event could have been precipitated by induced volume contraction. Subsequent repletion of the sodium load, and in effect the expansion of the extracellular fluid volume status, gradually improved the neurologic function of the patient.

The management in CSWS is characterized by aggressive sodium replacement to compensate for the salt wasting by using either an isotonic or hypertonic saline infusion. Frequent monitoring of sodium and fluid balances, as well as daily weight measurements are essential to estimate body fluid changes. Fluid restriction, the cornerstone of management in SIADH, is deleterious in CSWS as demonstrated in a study done by Wijdicks et al. Almost half of the study patients treated with fluid restriction developed cerebral infarction, most of which were clinically diagnosed with SIADH. Although volume status was not clearly defined in the study, the authors implied that if the study population were indeed hypovolemic, fluid restriction may have aggravated the decreased cerebral perfusion pressure and causing vasospasm. Other treatment strategies that can be employed involves administration of corticosteroids such as fludrocortisone, a potent mineralocorticoid that acts on the distal tubules, stimulating reabsorption of sodium and water, leading to extracellular fluid expansion. This decreases the daily sodium requirement needed to maintain balance and also acts to stop the natriuresis-induced polyuria. This strategy was highly effective in our case as fludrocortisone decreased the urine output and sodium requirement (Figures 1 and 2).

CONCLUSION
The differentiation of CSWS versus SIADH occurring after an acute brain disorder can be challenging due to significant overlap between them. An accurate assessment of the volume status with clinical and objective parameters is critical to make the correct diagnosis and thus avoid untoward clinical consequences.

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REFERENCES


