

Cerebral Salt Wasting Syndrome due to Subdural, Intraparenchymal and Subarachnoid Hemorrhage: A Case Report

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Abstract

Introduction: Cerebral salt wasting is one of the causes of hypovolemia and hyponatremia seen in cranial trauma, cranial tumor or post-surgery patients. We describe a patient with traumatic subdural hematoma developing cerebral salt wasting on the 12th day of follow up.

Case presentation: A 65 year's old drunk man is found to have fell from his balcony over 4 meters early in the morning. The initial neurological examination in emergency ward showed a coma state with no consciousness, right localizing motor response to noxious stimuli and bilateral isochoric pupils. CT (Computer tomography) scan showed broad right frontotemporal subdural hematoma.

Decompressive craniectomy, hematoma drainage and duraplasty was performed, patient was admitted to our ICU (Intensive Care Unit). During ICU stay the patient didn't show any improvement with a GCS (Glasgow Coma Scale) of 5, flexion to noxious stimuli. Patient started to show decreased turgor tonus and hypotension on the 12th day with elevated urine sodium concentration and urine osmolality, low serum uric acid levels and hyponatremia. He is diagnosed with cerebral salt wasting syndrome. Serum sodium levels and symptoms of hypovolemia recovered with proper fluid/electrolyte replacement. Though the patient is lost on the 27th day.

Conclusion: Diagnose of cerebral salt wasting syndrome may be elusive. It may also be challenging to distinguish from inappropriate secretion of antidiuretic hormone syndrome in cranial lesions. Careful assessment leads to proper diagnosis and correct therapy for the patients.

Keywords: Cerebral salt wasting; Hypovolemia; Hyponatremia; Hematoma; Antidiuretic hormone

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Introduction

Cerebral endocrine problems comprising syndrome of Diabetes insipidus, inappropriate secretion of antidiuretic hormone and cerebral salt wasting are commonly encountered except for cerebral salt wasting [1]. Cerebral salt wasting Syndrome (CSWS) is defined as renal loss of sodium caused by intracranial malfunctioning leading to hyponatremia and hypovolemia. It can be seen in cranial trauma, cranial tumor, infection or after surgery patients. In the setting of central nervous system disease it is more frequent to see SIADH than CSWS. Considering the debate about cerebral salt wasting being a real syndrome, it is important to differ from SIADH (Secretion of inappropriate Antidiuretic Hormone Syndrome) as they are treated by opposite

therapeutic policies [2,3]. CSWS generally resolves in weeks to months. In some cases it can remain chronic. We report a case of a man with subdural and intraparenchymal hemorrhage who developed cerebral salt wasting syndrome.

Case Presentation

A 65 years old man with no past medical history other than alcohol abuse is found to have fell from his balcony over 4 meters early in the morning. He was referred to our emergency ward. The initial neurological examination showed a coma state with no consciousness, right localizing motor response to noxious stimuli and bilateral isochoric pupils. CT scan showed broad right frontotemporal subdural hematoma.

Decompressive craniectomy, hematoma drainage and duraplasty was performed, patient was admitted to our ICU. During follow up on the 5th hour, neurologic examination revealed anisocoria and a control CT scan is performed. A new intracerebral bleeding was detected and the patient was instantly sent to the operating room for the second time for decompression and hemostasis (**Figure 1**). His blood analysis revealed no abnormal findings as hemoglobin 10 mg/dl, hematocrit 40%, sodium 137 meq/L, blood urea nitrogen 16 mg/dl, creatinine 1,1 mg/dl.

During follow up in critical care the patient is treated with simvastatin, vitamin b complex and antiepileptics. Patient didn't show any neurologic improvement with a best Glasgow Coma Scale of 5, flexion to noxious stimuli. Patient started to show decreased turgor tonus and hypotension and high inferior vena cava distensibility (30%) on the 12th day with elevated urine sodium concentration (182 mmol/L) and urine osmolality (612 mosm/kg), low serum uric acid levels (1,7 mg/dl) and hyponatremia (131 mmol/L). On the following days with saline infusion, sodium concentrations didn't improve. Patient is diagnosed with cerebral salt wasting syndrome. Patient is followed with normal and hypertonic salt replacement. Serum sodium levels improved after ten days (**Figure 2**).

Discussion

Our case consists of subdural, intraparenchymal and subarachnoid hemorrhage following each other. Cerebral salt wasting syndrome (CSW) is characterized with polyuria and excessive loss of sodium with increased urinary output [4]. CSW is most commonly seen in aneurysmal subarachnoid hemorrhage and can also be seen in any traumatic brain injury as well as in stroke patients [5]. A study by Kalita et al. showed that cerebral salt wasting is the most frequent cause of stroke-related hyponatremia. The study showed that 43% of 100 stroke patients enrolled in the study had hyponatremia while 44% of them had cerebral salt wasting [1,6,7].

CSW usually develops in the first week but it may be less considered or may not be diagnosed properly. In our case, hyponatremia started on the 12th day of critical care follow up.

It is treated with hypertonic saline replacement but correction of serum sodium rapidly may also lead to other neurological symptoms [7-9]. CSW usually resolves spontaneously after 3 to 4 weeks. In the presence of CNS infection, cerebrospinal fluid obstruction and tumor progression it can last for months [10,11]. CSWS must be considered in the presence of hyponatremia, elevated urine osmolality, high urine volume and urine Na concentration above 30 mEq/L. Low serum uric acid concentration is expected due to urate wasting in the urine. Orthostatic hypotension, tachycardia, decreased skin turgor, and low central venous pressure also support CSWS. Increased hematocrit and urea level indicate dehydration. There are many potential causes of hyponatremia after brain surgery such as hypertonic fluid overload, SIADH, CSWS, secondary adrenal insufficiency, central hypothyroidism, desmopressin intoxication, hyperglycemia and drugs. SIADH and CSWS occur typically one week after surgery or later. SIADH and hypothyroidism are associated with euvolemia and CSWS is associated with hypovolemia. Distinguishing SIADH and CSWS is not easy as these two syndromes overlap in many laboratory findings and it is also challenging to differ them with physical evaluation [12-14]. The most important criterion to differentiate CSWS from SIADH is extracellular fluid volume status, which is generally normal in SIADH and decreased in CSWS. Serum osmolality is decreased in SIADH and increased or normal in CSWS, serum protein levels are normal or high in CSWS, and hematocrit is low or normal in SIADH and increased in CSWS [11]. Treatment of CSWS involves water and salt supplementation which suppress the release of ADH, resulting in dilute urine and correction of hyponatremia. Low serum sodium concentrations are related with increased cerebral edema. We didn't send samples for brain natriuretic peptide levels.

In SIADH, isotonic saline often worsens hyponatremia as the salt is excreted and some of the water is retained, so treatment must be fluid restriction [7,11]. If hyponatremia is resistant to treatment despite hypertonic saline replacement addition of fludrocortisone to treatment may be helpful [3,12]. In our case the patient had already received decompressive craniectomy and cerebral edema is less expected. Correction of serum sodium rapidly may lead to central pontine myelinolysis [15]. Our patient

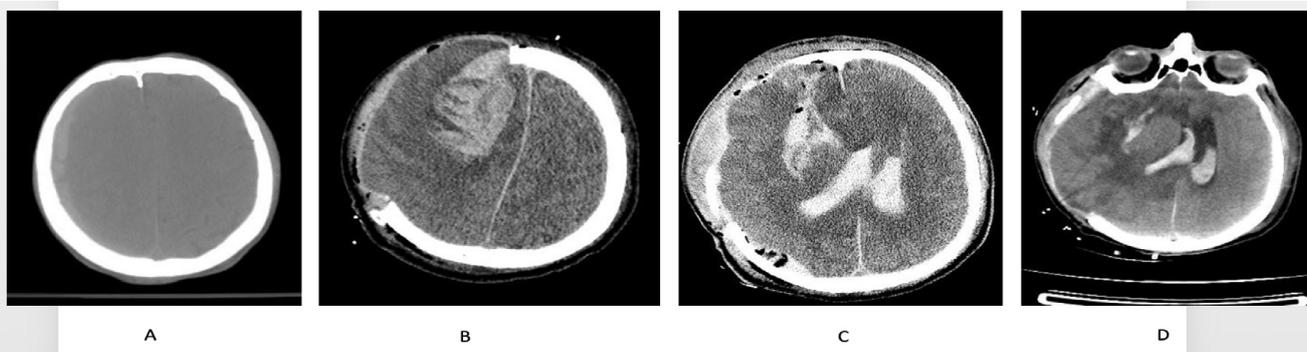


Figure 1 Subsequent Computer Tomographies of the patient.

(A) Subdural hematoma seen in emergency ward (B) Intracranial hematoma secondary to decompression surgery (C) After intraparenchymal hematoma decompression, Subarachnoidal hemorrhage (D) Subarachnoidal hematoma still seen in control tomography.

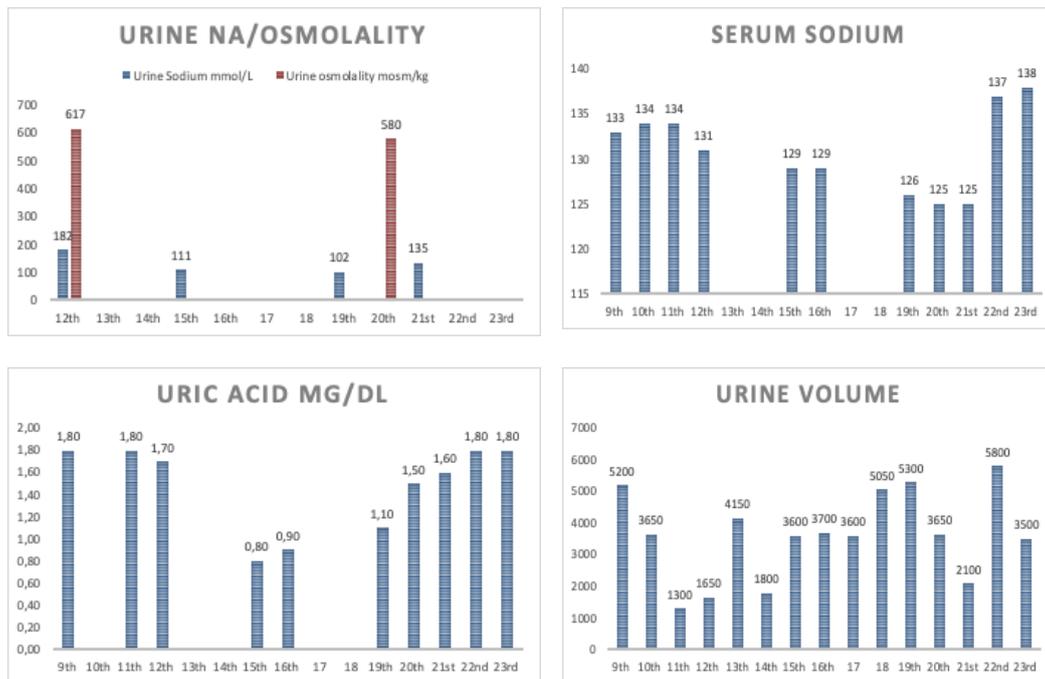


Figure 2 Laboratory Results of the Patient.

is treated with hypertonic saline after CSWS diagnosis and serum sodium levels improved immediately. Patient is lost later because of traumatic brain injury.

Conclusion

We reported a case of CSWS after subdural and intraparenchymal hemorrhage due to traumatic brain injury. Serum electrolyte disorders should be expected in patients with traumatic brain injury. The aim of this case presentation is to emphasize the importance of regular follow up of the patients clinical status, hydration, urine output, urine osmolality and sodium balance after brain surgery as correct diagnosis is essential in proper treatment.

Availability of Data and Material

Some of the data analyzed during this study are included in this published article. The remaining datasets generated during and

analyzed during the current case report are available from the corresponding author on reasonable request.

Author's Contributions

FSY wrote the manuscript. FSY and MŞ acquired patient data. HS and AE reviewed the case notes and were major contributors in writing the manuscript. All authors read and approved the final manuscript.

Acknowledgments

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Conflicts of Interest

The authors declare that they have no conflict of interest in this case report.

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