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Case Report

# Cerebral salt wasting syndrome in a patient who suffered a gunshot traumatic head injury: a case report

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## Abstract

Cerebral salt wasting syndrome is described as a hyponatremic condition in the context of a central nervous system injury. It is caused by a renal loss of sodium, and the primary distinguishing factor among similar conditions, such as the syndrome of inappropriate antidiuretic hormone secretion, is a reduction in the extracellular fluid volume. We describe here the case of a young adult who suffered brain injuries as a result of a gunshot and developed cerebral salt wasting syndrome during his in-hospital stay. Any type of cerebral attack can lead to hyponatraemic syndrome, but the most common are subarachnoid hemorrhages and neurological and meningeal tuberculosis infections. Treatment of the cerebral salt wasting syndrome leads to both hypovolemia and hyponatremia correction. The first line of management included hydric reposition with saline solution, either isotonic or hypertonic, depending on the severity of the symptoms.

## Keywords

brain injuries, cerebral hemorrhage, hyponatremia, gunshot wounds

## Introduction

Cerebral salt wasting (CSW) syndrome is characterized as a hyponatremic condition that arises from a central nervous system (CNS) injury.[1] It is caused by a renal loss of sodium, and the primary distinguishing factor among similar conditions is a reduction in the extracellular fluid volume.[2] The first description of CSW was made by Peters et al. in 1950.[3] Besides, CSW and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) are the most common causes of hyponatremia in neurological patients. Notice that hyponatremia has been described as having a major prevalence in neurological patients in comparison to other types.[4] There are two similar syndromes, and even some authors define the CSW as a subtype of SIADH.[1] Both CSW and SIADH present with hyponatremia and increased urine sodium levels. The principal difference is that the first syndrome has a decreased extracellular fluid volume, whereas the second syndrome has an increased extracellular fluid volume.[1,2] Accurate distinction between the two disorders is essential for approaching treatment properly.[1] We describe the case of a young adult who suffered brain injuries as a result of a gunshot and developed CSW during his in-hospital stay.

## Case report

*A 25-year-old male patient, migrant and homeless with a history of drug abuse, was brought to the emergency de-*

*partment after being found unconscious on the street with multiple craniofacial injuries. The patient's vital signs, as determined by physical examination, included a blood pressure of 112/78 mmHg, a heart rate of 106 beats per minute, a respiratory rate of 20 breaths per minute, and temperature of 36.0°C. The Glasgow Coma Scale score was 7/15 (eye opening one point, verbal response one point, and motor response five points). The patient had a circular injury on the left temporal region with moderate nasal and oral bleeding and a lineal injury with edema on the right zygomatic arch.*

*Due to his neurological status, the patient required invasive mechanical ventilation with an endotracheal tube and a right subclavian central venous catheter was placed. Cranial computed tomography (CT) showed multiple bullet shrapnel in the temporal lobe and left parietal lobe, compromise of the greater wing of the sphenoid, sphenoidal sinus and left maxillary sinus* ***(Fig. 1)****, associated with a diffuse subarachnoid hemorrhage (SAH)* ***(Fig. 2)****.*

*Additionally, he had a cerebral and neck CT angiography within normal parameters. Control cranial CT reported a decrease in SAH with an increase in cerebral edema* ***(Fig. 3)****. The neurosurgical team, in consensus with oral and maxil-*

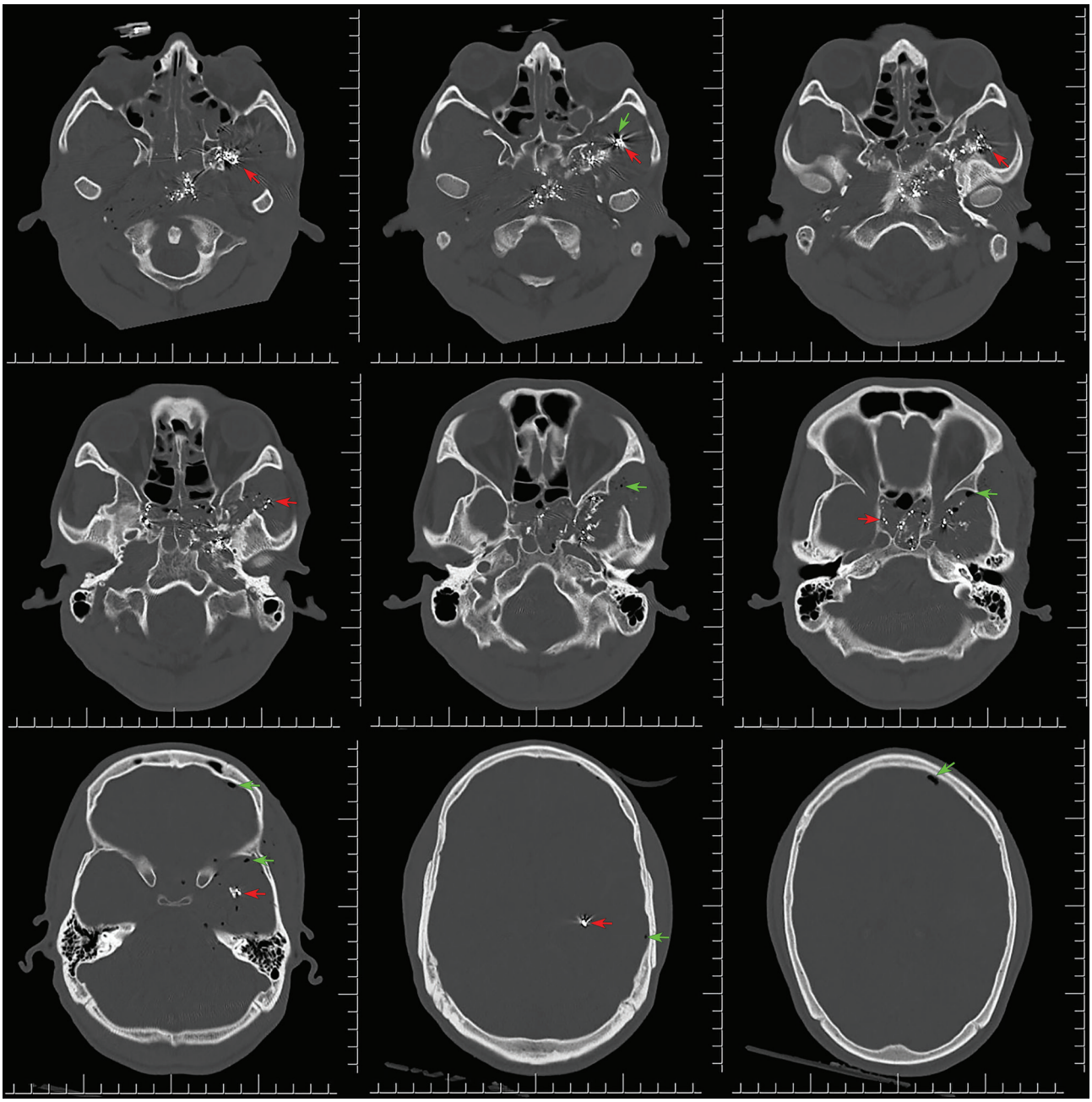

**Figure 1.** Axial slices of head CT without contrast in the bone window show air in the subdural space (green arrows), multiple metallic elements (2980 HU) no larger than 3 mm at the base of the skull and left temporal and parietal lobe (red arrows), loss of continuity of the greater wing of the left sphenoid with hypertensive liquid content in the sphenoidal sinus, and loss of continuity of the left lateral wall of the sphenoid sinus and of the lateral wall of the left maxillary sinus.

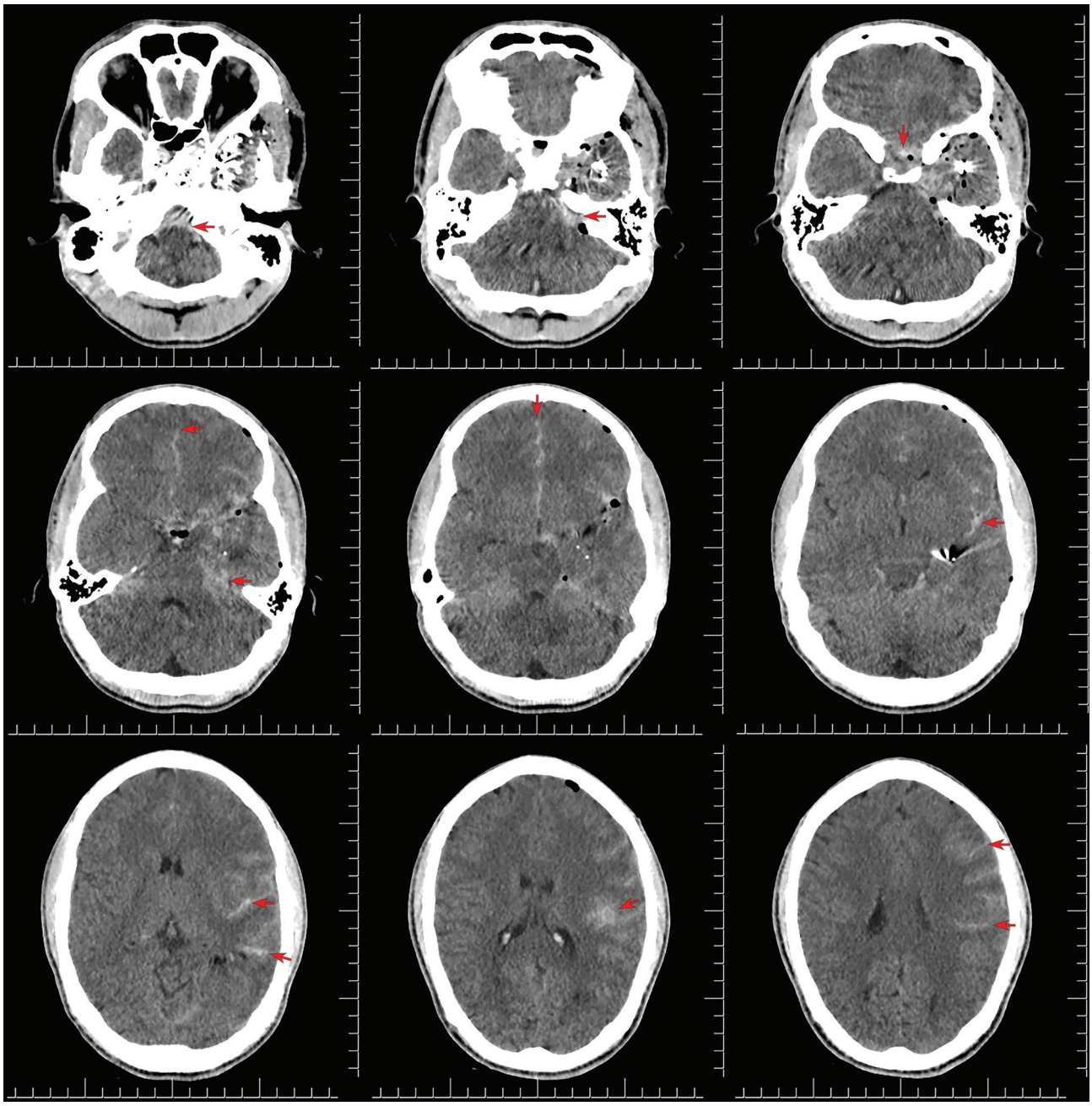

**Figure 2.** Axial slices of head CT without contrast in the brain window show bleeding in the frontal, parietal and left temporal subarachnoid spaces with extension to the sylvian fissure, interhemispheric space and base cisterns (red arrows).

*lofacial staff, decided that the patient was not a candidate for surgical intervention. The thoracic radiography revealed pneumothorax of grade II, as classified by the Argentine Society of Thoracic Surgeons. The patient was managed with a closed tube thoracostomy for a duration of 48 hours, after which the tube was removed.*

*The patient had a satisfactory clinical evolution, was successfully extubated three days later, and continued oxygen supplementation with a nasal cannula without the need for vasoactive agents. Despite the evident clinical improvement, the patient began to manifest hypokalemia* ***(Table 1)****. The initial treatment plan involved intravenous (IV) repositioning, initially with an infusion of potassium chloride (4 mEq per hour) and subsequently with hypertonic saline solution (3%), although without adequate response.*

*The patient continued with stationary clinical evolution, drowsiness with episodes of agitation and dysarthric, and vital signs within normal parameters, although fluid balance showed polyuria* ***(Table 2)*** *and persistent severe hyponatremia* ***(Table 1)****. Emergency staff considered the diagnosis of CSW syndrome due to the classic findings that include the polyuric state, which, without IV reposition, will lead to symptomatic hypovolemia. They decided to increase IV reposition with an infusion of hypertonic saline solution (7.5%) at 30 cc/hour and started management with desmopressin 15 mcg IV every day for three days and later decreased the*

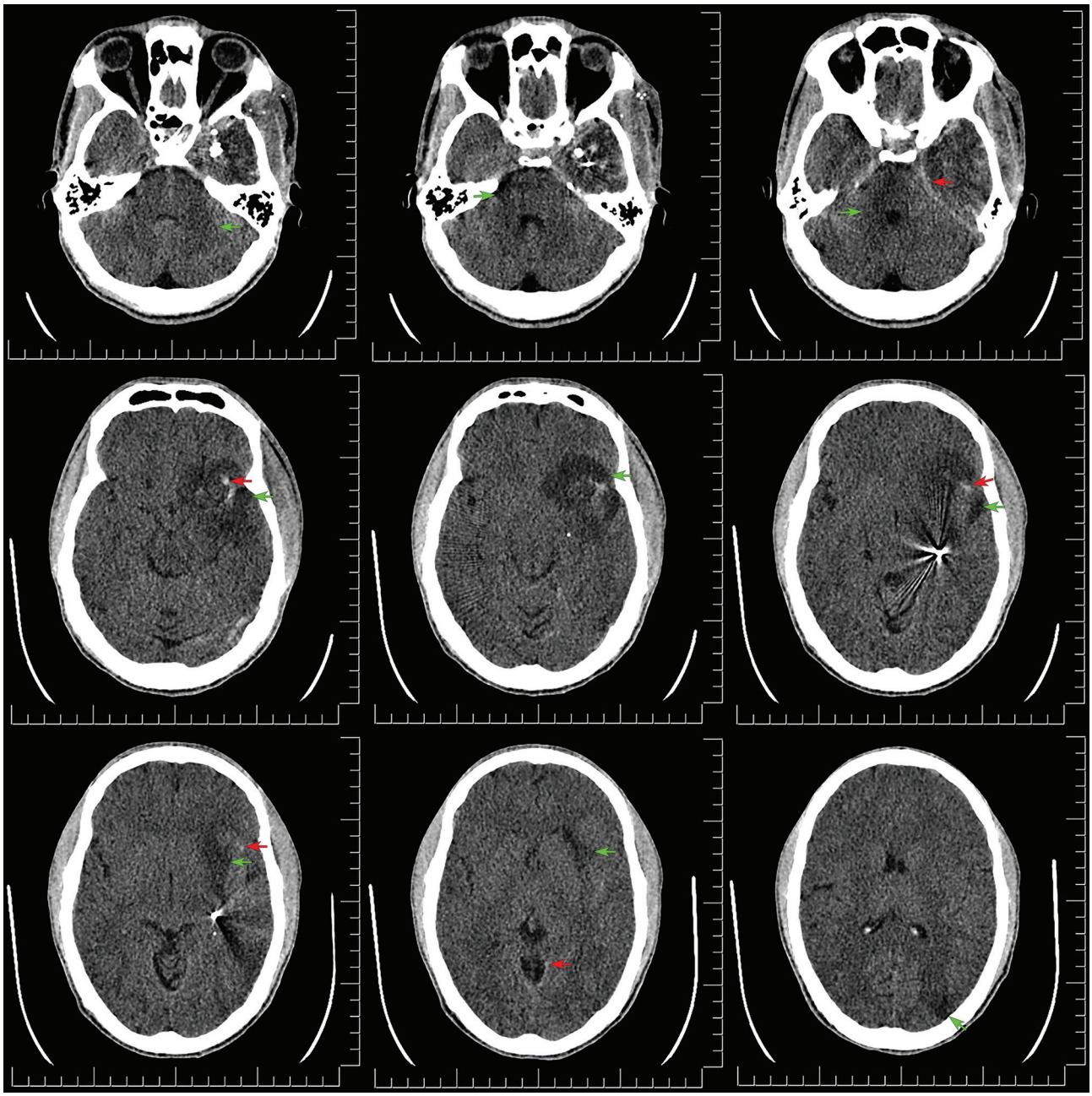

**Figure 3.** Axial slices of head CT without contrast in the brain window show a decrease in bleeding in the frontal, parietal and left temporal subarachnoid spaces (red arrows), with a notorious increase in vasogenic edema (green arrows) in comparison with the previous image.

*dose to 4 every 8 hours. Once the serum sodium increased, the desmopressin was replaced by tolvaptan (15 mg every day). During the hospital stay, the patient presented clinical symptoms compatible with withdrawal syndrome, so an appointment by the psychiatric department was requested and they decided to start management with benzodiazepines and typical antipsychotics. On day eleven of the hospital stay, there was no availability of desmopressin or tolvaptan; as a result, the patient continued only the sodium reposition with hypertonic saline solution 3% as treatment. The patient exhibited a substantial improvement in sodium levels, achieving a normal range, and was discharged with outpatient appointments.*

## Discussion

SIADH and CSW are hyponatremia syndromes derived from CNS injuries. They share most of the laboratory findings, in both SIADH and CSW, there is hyponatremia (<135 mEq/L), elevated urine sodium (>40 mEq/L), and elevated urine osmolarity (>100 mOSm/kg). In contrast, they presented different clinical findings. On the one hand, CSW syndrome can present hypotension, tachycardia, lack of skin turgor, dry mucous membrane and laboratory tests showing elevated hematocrit, elevated blood urea, elevated serum albumin, negative fluid balance or a decreased

**Table 1.** Laboratory reports

| | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Day 9 | Day 10 | Day 11 | Day 12 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| White blood cell (WBC) ($mm^3$) | 19.760 | 14.360 | 8.920 | 8.960 | 7.320 | 8.300 | 11.260 | 10.680 | 7.930 | 10.860 | | |
| Neutrophils (%) | 75 | 85.2 | 76.8 | 79.7 | 64.3 | 62.6 | 66.4 | 89.6 | 79.5 | 71.7 | | |
| Lymphocytes (%) | 15.1 | 8.7 | 15.4 | 12.6 | 21.5 | 18.3 | 12 | 6.4 | 11.4 | 17.7 | | |
| Hemoglobin (g/dL) | 13.5 | 8.9 | 9.5 | 11.1 | 10.1 | 9.2 | 11 | 9.8 | 9.9 | 9.3 | | |
| Hematocrit (%) | 39.6 | 26 | 27.8 | 32.3 | 29.7 | 26.8 | 33 | 28.9 | 28.7 | 26.9 | | |
| Platelets ($\times 10^3$ $mm^3$) | 307 | 155 | 175 | 227 | 219 | 246 | 315 | 325 | 365 | 342 | | |
| Glucose (mg/dL) | 127.6 | 96.7 | 90 | 90 | 104 | 86.2 | | 134 | | 99.2 | | |
| Creatinine (mg/dL) | 0.78 | 0.4 | 0.71 | 0.47 | 0.61 | 0.52 | 0.63 | 0.5 | 0.46 | 0.67 | | |
| Blood urea nitrogen (mg/dL) | 12.8 | 7.7 | 5.5 | 7.1 | 7.9 | 8.8 | | 7.3 | 11.6 | 8.8 | | |
| Sodium (mmol/L) | 140 | 131 | 122 | 119 | 125 | 128 | 125 | 121 | 122 | 124 | 126 | 137 |
| Potassium (mmol/L) | 4.1 | 3.2 | 3.9 | 3.6 | 3.8 | 4 | 4.1 | 3.9 | 3.7 | 3.2 | | |
| Osmolarity (mOSm/dL) | 291 | 270 | 251 | 245 | 259 | 263 | | 252 | | 256 | | |

Source: own elaboration

**Table 2.** Fluid balance

| | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Day 9 | Day 10 |
|---|---|---|---|---|---|---|---|
| Fluid input (cc) | 3410 | 3940 | 3900 | 3560 | 7603 | 3850 | 3690 |
| Fluid output (cc) | 6226 | 4786 | 6036 | 8036 | 6434 | 6236 | 4536 |
| Urine output (cc/kg/hr) | 3.3 | 2.4 | 3.2 | 4.5 | 3.5 | 3.3 | 2.3 |

Source: own elaboration

central venous pressure (CVP).[1,4,5] On the other hand, the Misra et al.[6] criteria necessitate the presence of at least two out of four findings to diagnose SIADH. The first criterion is the absence of hypovolemia signs, the second criterion is the absence of laboratory tests suggesting dehydration, the third criterion is a normal or positive fluid balance without any weight loss, and the fourth criterion is an elevated CVP (>6 cm of water).[4,6]

Any type of cerebral aggression has the potential to develop into hyponatremia syndrome, although the most commonly associated are subarachnoid hemorrhages (SAH) and neurological and meningeal tuberculosis infections.[7] Some of the theories that tried to explain the causality of the sodium loss in the CSW syndrome proposed fewer sympathetic stimulations of the juxta-glomerular apparatus, which finally reduced sodium, urate and water absorption; additionally, it has been associated with less secretion of renin and aldosterone.[7,8] The second assumption was correlated with high levels of brain natriuretic peptide (BNP) and atrial natriuretic peptide (ANP) in patients with SAH and sodium and water renal loss.[7] Normally, CNS injury is caused by SAH, chronic conditions or postoperative states after tumor or mass resections. Notice that CSW syndrome can also be caused by traumatic methods, and some writers have published works about cases of CSW syndrome after a serious brain injury. CSW syndrome can occur after a gunshot injury, but it is extremely unusual because head gunshot injuries have a high early death rate. There is no literature available on the subject, and the few studies that have looked at CSW syndrome after a traumatic brain injury have mostly found that traffic accidents are the main cause.[9-12] Our patient may be an exceptional case; he survived after a headshot, then the medical team confirmed CSW syndrome and the patient presented relatively low sequelae after this significant life-threatening condition.

Treatment of the CSW tried to correct both hypovolemia and hyponatremia; the first line of management included hydric reposition with saline solution, isotonic or hypertonic, depending on the severity of the symptoms. The sodium repository may be progressive, avoiding correcting more than 10 mmol/L in the first 24 hours.[7] Other medications that have been studied in cases of hyponatremia secondary to neurocritical conditions are fludrocortisone in doses of 0.1 to 0.4 mg because it has a direct effect on proximal renal tubules, increasing sodium reabsorption.[7,9,13] Besides, in cases of hypothalamic-pituitary region compromises, a refractory polyuria could manifest, where 1-deamino-8-D-arginine vasopressin (DDAVP) could be helpful, although its use is controversial because, despite a decrease in urine volume, it has a major production of natriuretic peptide, worsening hyponatremia.[8] Finding that could explain what happened to our patient, who during his hospitalization presented with hyponatremia and an important polyuria with a negative fluid balance. He was treated with hypertonic solutions and desmopressin without adequate response;

nevertheless, once the desmopressin was suspended, the sodium levels increased significantly.

Shah et al. evaluated the outcomes of patients with non-traumatic hemorrhagic stroke and hyponatremia, finding that this entity is a negative prognostic factor due to its longer hospitalizations, higher mortality rate and worse surviving outcomes.[14] Chendrasekhar et al. found comparable results, reporting major morbidity and mortality in patients suffering from CSW syndrome.[15] Our patient, despite having a severe brain injury, had a favorable neurological evolution, although their paraclinical findings determined greater in-hospital requirements.

## Conclusions

Even though CSW and SIADH are not common syndromes, it is important to consider them, particularly in patients who have suffered a brain injury, primarily after a SAH, without ignoring traumatic causes of several types, including penetrating wounds from gunshots. The distinction between both hyponatremic syndromes could be a challenge for healthcare practitioners, although it has enormous relevance due to each entity being treated differently. The goal of prompt management is to avoid the progression of hyponatremic and, in the case of CSW, worsening hypovolemic status.

## Author contributions

D.A.M. and A.R.U.: investigation, writing – original draft; L.G.A. and I.C.G.C.: supervision, writing – review and editing.

## Ethics

Written informed consent was obtained from the patient to publish the case report.

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## Competing interests

The authors have declared that no competing interests exist.

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