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***Case Report***

# Physiological aspects of cerebral salt wasting syndrome: A case report

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## Abstract

We want to report an interesting case of cerebral wasting syndrome presented with moderate brain injury and electric shock to provide a good physiological view and a better understanding of this syndrome. We have reported the case of a 39-year-old male patient with a history of recent electric shock and brain injury. This case report provides a unique opportunity to observe the physiological aspect of a patient diagnosed with cerebral salt wasting syndrome after electric shock and brain injury. Understanding the underlying pathophysiology of cerebral salt wasting syndrome is essential for effective diagnosis and treatment.

**Keywords:** Physiology, cerebral, salt wasting, hyponatremia.

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## Introduction

Cerebral salt-wasting syndrome (CSWS)is a development of extracellular volume depletion due to a renal sodium transport abnormality in patients with intracranial disease and normal adrenal and thyroid function.[1] It is still a mysterious entity characterized by hypotension that can lead to hypovolemic shock[2] and different from another syndrome (syndrome of inappropriate antidiuretic hormone secretion) by a distinctive feature of polyuria whereas in the syndrome of inappropriate antidiuretic hormone secretion(SIAH), the urine volume is normal or low.[3]

One of the greatest challenges is to explain the physiological aspect of CSWS with multiple lesions under study. However, the previous studies of CSWS have reported on cases with one lesion that may lead to the syndrome. Oshima and his colleagues[4] reported on a case of Kawasaki disease with CSWS, characterized by a negative balance of both Na and water with hypouricemia and high fractional excretion of uric acid. Bitew *et. al.* [5]reported a case with renal salt wasting had pneumonia without cerebral disease and showed increased plasma aldosterone and Fractional excretion phosphate, and two patients with syndrome of inappropriate antidiuretic hormone had increased blood volume, low plasma renin and aldosterone, and normal Fractional excretion phosphate. The patient with renal salt wasting responded to isotonic saline by excretion of dilute urine, prompt correction of hyponatremia, and a normal water loading test after volume repletion. Hypouricemia and increased Fractional excretion of urate persisted after correction of hyponatremia. Two patients with the syndrome of inappropriate antidiuretic hormone failed to dilute their urine and remained hyponatremic during 48 and 110 h of saline infusion.

Here, we reported and discussed the physiological aspects of a rare case with traumatic brain injury, electric shock and cerebral salt wasting case with severe hypotension.

## Case report:

We reported the case of a male 39-year-old patient without a history of chronic disease. He presented with a moderate burns on his right arm due to an electric shock, then fell from a height 9 meters onto a hard concrete surface, causing a severe brain injury in the right side of his head and eye, with multiple fractures in the face, ribs, leg, thigh, and knee.

The chest Computerized tomography scan (CT scan) reveals a moderate contusion on the right side and increased lung density in the posterior zone, accompanied by subpleural effusion. Following the shock, the patient experienced 17 days of unconsciousness in the intensive care unit, where he was hospitalized for emergency evacuation. He was later extubated as his neurological and physiological status improved. Upon regaining consciousness, he exhibited severe hypotension (as shown in Figure 1), abnormal body movements, seizures, and a coma. His medical history does not indicate any episodes of poorly controlled hypotension. After his admission to the emergency room due to worsening coma, abnormal movements, and severe hypotension, laboratory tests revealed excessive polyuria (approximately 6000 ml/day), a serum creatinine level of 40 mmol/L (within normal range), albumin at 3.5 g/dL (normal), and a decreased serum cortisol level of 13.9 pg/mL.

Other clinical tests, as reported in Table I and Figure 2, revealed that the mean levels were as follows: Packed Cell Volume (PCV) was 17.44±14.84 (l/l), glucose was 106±35.35 (mg/dl), blood urea was 42.9±30.97 (mg/dl), sodium was 132±1.41 (mmol/l), and potassium was 4.15±0.12 (mmol/l). All thyroid hormones were within normal levels (T3 was 84.3 nmol/L, T4 was 1.3 nmol/L, and TSH was 0.43 uIU/ml). The blood level of ACTH (adrenocorticotropic hormone) was 8.2 pg/mL, and the serum cortisol level was 13.9 ng/mL, while the urinary cortisol level was 984 mg/day, which is considered high. Treatment focused on correcting fluid and electrolyte imbalances using normal saline and Ringer's solution over three weeks to maintain a positive salt balance and prevent volume depletion by matching urinary output with fluid replenishment.

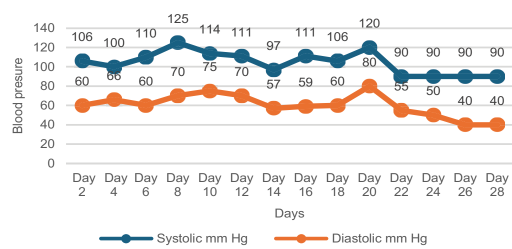


**Figure 1** : Monitoring blood pressure (mm Hg) changes following coma recovery

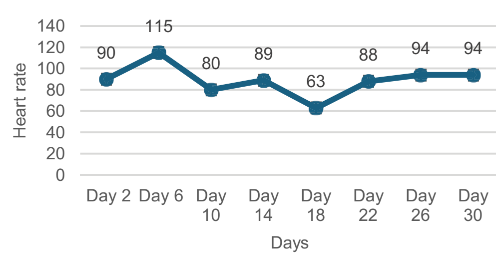


**Figure 2**: Heart rate monitoring following coma recovery

**Table I**: Patient Laboratory Findings Throughout Hospital Stay

| Clinical tests | Minimum | Maximum | Mean | SD | Number of readings |
|---|---|---|---|---|---|
| PCV (l/l) | 0.34 | 27.00 | 17.44 | 14.84 | 3 |
| Troponin (ng/ml) | 0.19 | 0.19 | 0.19 | . | 1 |
| Glucose (mg/dl) | 81.00 | 131.00 | 106.00 | 35.35 | 3 |
| Blood Urea (mg/dl) | 21.00 | 64.80 | 42.90 | 30.97 | 3 |
| Sodium (mmol/l) | 131.00 | 133.00 | 132.00 | 1.41 | 3 |
| Potassium (mmol/l) | 4.03 | 4.20 | 4.11 | 0.12 | 3 |
| Chloride (mmol/l) | 104.00 | 104.00 | 104.00 | . | 1 |
| Calcium (mg/dl) | 8.23 | 8.23 | 8.23 | . | 1 |
| Magnesium (mg/dl) | 1.96 | 1.96 | 1.96 | . | 1 |

**Table II**: Hormonal Test Results During Hospitalization

| Test | Results | Number of readings |
|---|---|---|
| Serum ACTH | 8.2 pg/ml | 1 |
| Serum cortisol | 13.9 ng/ml | 1 |
| Urinary cortisol | 984 mg/day | 1 |
| Total urinary volume | 6000 ml/day | 1 |
| T3 | 84.3 nmol/l | 1 |
| T4 | 1.3 nmol/l | 1 |
| TSH | 0.43 uIU/ml | 1 |

## Discussion

Cerebral salt wasting syndrome is a condition characterized by hyponatremia (low blood sodium levels) and excessive urinary sodium loss.[6] In our study, the mean sodium level was 132.00±1.41 mmol/l (Table I), with low blood pressure 90/50 mm/Hg (hypotension), with different hart rate levels between 63-115 per min.as illustrated in Figure 1 and 2.

The cerebral salt wasting usually occurs in patients with acute or chronic brain injuries, such as traumatic brain injury, subarachnoid hemorrhage, or brain tumors.[7-8] It leads to increased urine output (polyuria), which can result in dehydration and electrolyte imbalances.[9-10] Excessive sodium loss in the urine (natriuresis) is the hallmark feature of the condition. It results from a combination of hormonal and neural factors that disrupt normal renal function, impairing the kidneys' ability to reabsorb sodium and leading to its loss through polyuria.[11]

In our case report, the urine output exceeded 6000 mL/day, which is consistent with findings reported in previous study. Leonard *et al*.[1] mentioned that the diagnosis of CSWS in patients with very high urine output was made at volumes of approximately 6000 mL/day

Rajendran *et. al.*[3]revealed that CSWS can occur in patients with cerebral diseases despite normal adrenal and thyroid gland function, leading to hyponatremia and extracellular volume depletion. In our study, all thyroid and adrenal function tests were within normal limits (Table II)

The relationship between brain injury and cerebral salt waste:

Brain injury or head trauma can affect kidney function by triggering the release of specific proteins that interfere with normal renal processes.[1] It is the release of the natriuretic factors (brain natriuretic peptide) which then enter systemic circulation through a disrupted blood-brain barrier. The brain natriuretic peptide acts on collecting ducts of the renal tubules to inhibit sodium reabsorption as well as decrease the release of renin.[12]

To connect with the central nervous system, the kidneys are richly innervated by many nerves (renal efferent and afferent nerves)[13], each one have different functions. The renal efferent nerves release renin and prostaglandins as well as regulate glomerular filtration rate, tubular reabsorption of sodium and water and renal blood flow. All these factors help to regulate renal function. Renal afferent nerves complete the feedback loop via central autonomic nuclei, where the signals are processed and affect central sympathetic outflow, both types of neurons are critical components of the self-regulated Reno renal reflex loop.[13-14] The renal efferent nerves are predominantly adrenergic. Norepinephrine release mediates vasoconstriction of the renal vasculature, as well as sodium and water reabsorption at renal tubular epithelial cells, and renin release from the juxtaglomerular lar cells.[14] So, during brain injuries the sympathetic nervous activity increases and the sodium loss from the kidneys, by mechanisms the first one is increased the dopamine release due to brain injury, leading to elevated renal pressure–natriuresis response and urinary sodium loss. Another mechanism is probably due to the injured brain's release of brain natriuretic peptides.[3] Many hormones regulate the sodium hemostasis in the body; renin is a circulating enzyme produced and stored within the kidney and released in response to low systemic and renal arterial perfusion. Once released, it initiates a series of intricate sequential enzymatic steps involving the converting of angiotensin enzyme, the ultimate product of which is the formation of angiotensin II. This potent vasopressor agent immediately affects blood pressure by influencing the constrictive properties of peripheral vasculature, increasing sympathetic tone, and stimulating the release of Antidiuretic hormone.[15] We hypothesize that increased activity of parasympathetic neurons during cerebral salt wasting associated with brain injury leads to an elevated micturition rate through enhanced acetylcholine release, which stimulates detrusor muscle contraction and facilitates bladder emptying.

## Conclusion

This case report offers a valuable opportunity to explore the physiological manifestations in a patient diagnosed with CSWS following an electric shock and traumatic brain injury. The findings suggest that brain injury has immediate effects on renal function, mediated by enhanced parasympathetic activity, increased glomerular filtration rate, altered water and sodium reabsorption, and changes in blood pressure, driven by complex physiological mechanisms.

CSWS is a multifaceted condition marked by excessive renal sodium excretion, resulting in hyponatremia and hypovolemia. Commonly precipitated by brain injuries or tumors, CSWS is mediated by the release of natriuretic peptides that disrupt sodium homeostasis. The ensuing low serum sodium levels and dehydration present as polyuria, polydipsia, muscle cramps, and other clinical signs. Accurate differentiation of CSWS from similar syndromes, such as the syndrome of inappropriate antidiuretic hormone secretion (SIADH), is critical for effective management, which primarily involves fluid and sodium replacement. A clear understanding of the pathophysiology is essential for accurate diagnosis and appropriate therapeutic intervention.

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## Ethical aspects

Informed consent was taken from the patient to publish this study.

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