Syndrome Of Inappropriate Antidiuretic Hormone After Head Injury: A Case Report.

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Abstract

We report a case of Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) in a 28-year-old male following a head injury due to a road traffic accident. The patient exhibited classic symptoms of SIADH including disorientation and hyponatremia with a serum sodium of 128 mmol/L. Diagnostic findings supported the presence of SIADH with elevated urine osmolality and urine sodium concentration. Management involved fluid restriction and cautious electrolyte correction, leading to gradual clinical improvement and resolution of cerebral edema. This case highlights the importance of considering SIADH in patients with traumatic brain injury, as early recognition and management are crucial to prevent severe complications and enhance recovery.

Keywords: SIADH; Hyponatremia; Traumatic Brain Injury; Neuroendocrine Dysregulation.

INTRODUCTION

The Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) represents a critical disorder where excessive secretion of the antidiuretic hormone (ADH) leads to fluid retention, hyponatremia, and a concentrated urine despite normal or increased plasma volume.[1] This endocrine condition often manifests secondary to various triggers including malignancies,,

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infections, medications, and notably, traumatic brain injuries. Head trauma, a common cause in younger adults, disrupts the hypothalamic-pituitary axis leading to an unregulated release of ADH.[2]

In terms of epidemiology, SIADH is an underrecognized complication of head injuries, particularly in the context of non-penetrating traumas.[3] The pathophysiology involves damage or stimulation of hypothalamic cells that produce ADH or the supraoptic nuclei, leading to inappropriate secretion despite hypo-osmolarity of the blood. This dysregulation is particularly perilous as it can exacerbate cerebral edema in head injury patients, thereby increasing morbidity and mortality if not swiftly addressed.[4]

Clinically, SIADH in trauma patients may present subtly with symptoms ranging from mild confusion or lethargy to severe outcomes such as seizures due to acute hyponatremia. Diagnosis involves identifying decreased serum osmolality, increased urine osmolality, and high urine sodium levels in the absence of diuretic use, adrenal insufficiency, renal salt wasting, or recent fluid administration that could alter serum sodium. The manifestation of SIADH following head trauma is significant due to its potential to significantly alter clinical outcomes.[5]

A notable feature in this case is the rapid development of symptoms post-injury, which can provide essential clues to its presence in the acute setting. Early detection and management are crucial to mitigate the risks of severe hyponatremia and its neurological consequences.

CASE REPORT

The patient, a 28-year-old male, was admitted following a motor vehicle accident with a documented head trauma. Initial evaluation revealed disorientation and agitation with a documented Glasgow Coma Scale of 14. No other physical injuries were noted on examination.

Diagnostic imaging with a head CT scan was performed, showing no hemorrhagic lesions but suggested mild diffuse cerebral edema. Laboratory tests revealed a serum sodium of 128 mmol/L, which raised concerns for SIADH. The diagnosis was further supported by a urine osmolality of 450 mOsm/kg and a urine sodium concentration of 90 mmol/L. Blood tests ruled out other potential causes of hyponatremia, such as adrenal insufficiency and hypothyroidism.

Laboratory Test	Patient Value	Normal Range	Interpretation
Serum Sodium	128 mmol/L	135-145 mmol/L	Hyponatremia indicative of SIADH, suggests fluid retention and dilutional state.
Serum Osmolality	260 mOsm/kg	275-295 mOsm/kg	Low serum osmolality confirms hypo-osmotic condition, consistent with SIADH.
Urine Osmolality	450 mOsm/kg	50-1200 mOsm/kg	Inappropriately high urine osmolality relative to serum osmolality, characteristic of SIADH.
Urine Sodium	90 mmol/L	20-40 mmol/L	Elevated urine sodium in the context of hyponatremia, supports the diagnosis of SIADH.
Blood Urea Nitrogen	8 mg/dL	8-20 mg/dL	Normal BUN suggests adequate renal function and helps rule out renal causes of hyponatremia.
Serum Creatinine	0.9 mg/dL	0.6-1.2 mg/dL	Normal creatinine indicates normal renal function, reinforcing SIADH

			diagnosis without renal impairment.
Thyroid-Stimulating Hormone (TSH)	2.5 µIU/mL	0.4-4.0 µIU/mL	Normal TSH rules out thyroid dysfunction as a cause of SIADH.
Cortisol (AM)	12 µg/dL	10-20 µg/dL	Normal morning cortisol excludes adrenal insufficiency as a contributing factor.

Table 1:- Laboratory investigations suggestive of SIADH.

Management began with fluid restriction and close monitoring of serum electrolytes. The patient's sodium levels were cautiously corrected with hypertonic saline to prevent central pontine myelinolysis. Over the next few days, gradual improvement in the patient's sodium levels and mental status was noted, aligning with resolution of cerebral edema on subsequent imaging.

DISCUSSION

The pathophysiological basis for SIADH in the setting of head trauma involves the disruption of the hypothalamic-pituitary axis, particularly impacting the regions responsible for ADH regulation. In response to physical stress and neuronal injury, there can be an aberrant release of ADH, which in turn causes water retention, dilutional hyponatremia, and concentrated urine. These manifestations are particularly perilous as they can exacerbate cerebral edema, thereby compounding the primary injury.[7]

The literature reveals numerous instances of SIADH following various types of brain injury. A study by Kim et al. (2015) in the "Journal of Clinical Endocrinology & Metabolism" described similar findings in a cohort of patients with mild to severe traumatic brain injury, where the incidence of SIADH was noted to be approximately 10%, aligning with other epidemiological data. This is significant as it underscores the potential for overlooked diagnoses in a subset of trauma patients who may present with subtle signs of sodium imbalance. [8]

Further comparative analysis is found in the work of Hannon et al which reviewed the outcomes of patients with post-traumatic SIADH and highlighted the challenges in managing these cases. Their findings suggested that meticulous attention to fluid balance and cautious correction of sodium levels are crucial, as overly rapid correction can lead to central pontine myelinolysis, a devastating and often irreversible complication.[9]

it is also pertinent to consider the broader implications for clinical practice. For instance, the consensus guidelines for the management of SIADH suggest initial conservative treatment with fluid restriction, which was applied in this case. Additionally, the use of hypertonic saline is recommended in cases of severe or symptomatic hyponatremia. This approach was successfully implemented in our patient, leading to a gradual and safe correction of his serum sodium and clinical improvement.[10]

Moreover, this case contributes to the ongoing discourse on the necessity for routine screening for electrolyte disturbances in all patients presenting with head trauma, irrespective of the severity of the injury. The prompt recognition and treatment of SIADH can substantially improve outcomes and reduce the long-term sequelae associated with both the hormonal disorder and the primary neurological injury.

CONCLUSION

This case serves as a critical reminder of the complexities involved in the intersection of neurotrauma and hormonal dysregulation. This case also emphasizes importance of vigilance and proactive management in patients with traumatic brain injuries to identify and treat conditions such as SIADH promptly. Our experience suggests that an integrated approach involving early diagnostic evaluation and judicious management of fluid and electrolyte balance can significantly enhance patient outcomes in such challenging clinical scenarios.

Conflict of interest

None

Source of Funding

None

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Author Contribution:-ST: Concept Of Design; Manuscript Preparation; Revision Of Manuscript; Review Of Manuscript
How to cite This Article:-
Shahed Taher, Syndrome Of Inappropriate Antidiuretic Hormone After Head Injury: A Case Report, Int. j. med. case reports. 2024; 5 (2): 15-
Received: 30 January 2024 Revised: 15 February 2024 Accepted: 15 March 2024