



# A Diagnostic Dilemma in Combined Central Diabetes Insipidus and Cerebral Salt Wasting Syndrome

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J Neuroanaesthesiol Crit Care

Sodium imbalance is common after traumatic brain injury. It may develop due to cerebral salt wasting syndrome (CSWS); syndrome of inappropriate antidiuretic hormone secretion (SIADH); central diabetes insipidus (DI); or other causes such as hyperglycemia, administration of mannitol, and hypertonic saline.<sup>1</sup> However, the combined presentation of DI and CSWS is rarely described and may pose a diagnostic dilemma, affecting management.

A 20-year-old male polytrauma victim with no underlying systemic illness was conservatively managed in the neurointensive care unit for severe head injury. A provisional diagnosis of diffuse axonal injury (DAI) was made based on computed tomography (CT) scan of brain and a gradual improvement of neurological status was observed over days. Three weeks after admission, the patient developed polyuria with hypotension which was managed with crystalloid resuscitation while other possible causes were ruled out. The initial presentation was of hypernatremia with an elevated serum osmolality (339 mOsm/kg) which suggested the possibility of central DI. At the same time, the spot sodium level of 64 mEq/L with hypovolemia and raised blood urea (**Table 1**) pointed toward CSWS. The urine specific gravity (1.005) and

urine osmolality (499 mOsm/kg) were equivocal. Based on the high sodium levels, free water correction was started through enteral route which resulted in gradual reduction in serum Na<sup>+</sup> levels over the next 48 hours and normalization over the next 7 days. Polyuria persisted for 6 days during which hemodynamic parameters were maintained within normal limits by low-dose noradrenaline infusion, and positive fluid balance was maintained by enteral feeding or intravenous fluids as required. At the end of week, serum sodium level was 139 mEq/L, urine osmolality was 451 mOsm/kg, urine specific gravity was 1.003, and urine spot sodium was 56 mEq with urea level 47 mg/dL. The patient did not receive any osmotic agents prior to the development of this condition. Serum calcium (8.8 mg/dL) and daily blood sugar levels were normal (88–144 mg/dL). Renal ultrasonography was unremarkable. Patient became hemodynamically stable and no neurological deterioration was observed.

Our patient did not fit strictly in a picture of central DI as raised urine spot sodium and urine osmolality pointed toward probable concurrent cerebral salt wasting syndrome. The onset of such disturbances have been described between hours to many days after the trauma.<sup>2</sup> CSW is reported to

**Table 1** Trend of different values

Day	Serum sodium (mEq/L)	Urine osmolality (mOsm/kg)	Serum osmolality (mOsm/kg)	Urine spot sodium (mEq)	Urine specific gravity	Blood urea (mg/dL)	Blood glucose (mg/dL)
1	152		399			86	110
2	160	499		64	1.005	78	109
3	147					82	144
4	139	490	357	59	1.006	81	88
5	138					55	89
6	139					68	101
7	139	451		60	1.000	47	129



occur 2 to 8 days after the onset of central DI.<sup>3</sup> In our patient, both were noticed almost simultaneously and after 3 weeks of TBI with no known apparent inciting cause for such a delayed presentation. Investigations ruled out other less possible causes like triphasic response of DI-SIADH-DI, partial DI, or nephrogenic DI. Administration of desmopressin for central DI in a case of coexistent CSW, with the aim of suppressing polyuria, might have resulted in severe hyponatremia and, therefore, was avoided. Pharmacotherapy was not administered in our patient and fluid therapy was titrated based on the sodium levels and urine output.

Though less commonly reported, combined CSW and central DI may be an entity going unrecognized. Early suspicion; prompt diagnosis with serum and urine osmolality, and serum and urine sodium levels; along with appropriate treatment can reduce the high morbidity observed in this condition.

### Conflict of Interest

None declared.

### References

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