



## PROGNOSTICATION OF HYPONATREMIA IN TRAUMATIC BRAIN INJURY - A TERTIARY CENTRE BASED STUDY IN EASTERN INDIA

### Neurosurgery

**Dr. Arthelieu Sangma**

Mch Resident, Department of Neurosurgery, NRSMCH, Kolkata

**Dr. Kaushik Roy**

Professor, Department of Neurosurgery, NRSMCH, Kolkata

**Dr. Suniti Kumar Saha**

Professor, Department of Neurosurgery, NRSMCH, Kolkata

**Dr. Jiwesh Kumar\*** Mch Resident, Department of Neurosurgery, NRSMCH, Kolkata \*Corresponding Author

### ABSTRACT

**Introduction:** Road traffic accidents (RTA), the leading cause of TBI (around 60%), not only hits the most intellectual and economic class of 15-29 years but also causes loss to the Gross Domestic Product of the country to the tune of 1-3%. The most common electrolyte imbalance studied is that pertaining to sodium and that is hyponatremia. **Material and methods:** a prospective observational study comprising 300 patients with traumatic brain injury admitted at our centre were studied and their demographics, electrolyte imbalances, length of stay, were studied. **Results:** Among the 300 patients included in the study, 39 developed hyponatremia (13%), with 34 males (87.19%) and a mean age was 31.3 years. 6 patients had severe, 23 moderate and 10 had mild head injuries. The incidence of mild, moderate and severe hyponatremia was 26.7%, 51% and 22.3% respectively. Hyponatremia was associated with natriuresis in 38/39 patients (97.43%). The mean duration of stay for patients with hyponatremia was 23.7 days as opposed to 11.3 days for those without hyponatremia. **Conclusion:** the management of hyponatremia in traumatic brain injury is a critical aspect of patient care that demands a delicate balance between the correction of electrolyte imbalances and the prevention of osmotic demyelination syndrome. Thorough understanding of the underlying pathophysiology, regular monitoring of serum sodium levels and the understanding of judicious use of hypertonic alliance solutions are the key to successful management.

### KEYWORDS

Traumatic brain injury(TBI),Glasgow coma scale(GCS),Cerebral salt wasting( CSW), hyponatremia

#### INTRODUCTION:

Control and management of traumatic brain injury has become a challenge for the entire nation given the burden it ensues over the sufferer, the family and obviously the society. Given the increasing access to transportation and health care, we are now managing these patients in greater numbers and the results are encouraging. Road traffic accidents (RTA), the leading cause of TBI (around 60%), not only hits the most intellectual and economic class of 15-29 years but also causes loss to the Gross Domestic Product of the country to the tune of 1-3% [1].

During the course of management there often exists electrolyte imbalances, dyslipidemia, deranged renal functions etc. further delaying the recovery and contributing to the morbidity. These imbalances if not corrected within time, may lead to disability for life or even mortality.

The most common electrolyte imbalance studied is that pertaining to sodium, be it hyponatremia (defined as serum sodium < 135meq/L) or hypernatremia (>145meq/L). The other imbalances are those concerning potassium (<3.5meq/L and >5.5meq/L), magnesium, phosphate (hypo/hyper). The common causes of hyponatremia in TBI are CSW, SIADH, hypopituitarism and inadequate dietary intake of salt [2-5]. Most studies have demonstrated that timely institution of corrective measures/ continuous renal replacement therapy have greatly influenced the final outcome.

Our study, being one of the few from this part of India, aims to correlate the electrolyte imbalances in the traumatic brain injury patients being admitted in our department and the final outcome experienced on subsequent management.

#### AIMS:

- to determine the prevalence of electrolyte imbalance (sodium and potassium) in admitted patients of traumatic brain injury
  - to correlate this findings with the final outcome of the patients
- Materials and methods

**Study design:** prospective observational study

**Study place:** department of Neurosurgery, NRSMCH

**Study population:** all traumatic brain injury patients consenting to

participate (in case where patient was not able to give consent, the parent/guardian's consent taken)

#### Inclusion criteria:

age of patient > 1 year, diagnosed as any of traumatic brain injury (Intracerebral hemorrhage or ICH/ depressed fracture/ acute extradural hemorrhage or EDH/ acute subdural hemorrhage or SDH/ hemorrhagic contusion/ subarachnoid hemorrhage or SAH)

#### Exclusion criteria:

previous history of CKD/brain tumor /uncontrolled diabetes mellitus type 2 and patients with age < 1 year

**Study duration:** February 2023 to July 2023

**Sample size:** 180

#### Statistical Analysis:

Means and standard deviations were used to summarize all continuous variables; categorical variables were summarized using frequencies with percentages. Association between hyponatremia and other risk factors were assessed using chi-square tests. Results were presented as odds ratios with 95% confidence intervals (CI). All analyses were done using SPSS 22

#### RESULTS:

A total of 300 patients were admitted with a diagnosis of traumatic brain injury at our centre during the study period of whom 39 developed hyponatremia (13%), with 34 males (87.19%) and a mean age was 31.3 years. 6 patients had severe, 23 moderate and 10 had mild head injuries. The incidence of mild, moderate and severe hyponatremia was 26.7%, 51% and 22.3% respectively. Hyponatremia was associated with natriuresis in 38/39 patients (97.43%). Presence of intracerebral hemorrhage/hemorrhagic contusion on the admission CT scan of the brain was the commonest finding in these patients. The mean duration of stay for patients with hyponatremia was 23.7 days as opposed to 11.3 days for those without hyponatremia. The final outcome as assessed by our study reflected the appropriate and timely management of these patients as 35 patients were successfully discharged but 4 patients succumbed. Those who succumbed belonged to moderate and severe hyponatremia group (2 each). The causes were due to multiple comorbidities associated which could not be dealt successfully.

**Table 1. Results summarized**

Parameters	N	%
Hyponatremia	39/300	13
No. of males	34/39	87.19
Mean age at admission	31.3 years	
Head injury		
Mild	10	25.64
Moderate	23	58.97
Severe	6	15.38
Hyponatremia		
Mild	10	26
	20	51
	9	23
Moderate		
Severe		
Hyponatremia with natriuresis	38	97.43
Mean duration of stay (in days)		
With hyponatremia	23.7	
Without hyponatremia	11.3	
Final outcome		
Discharged	35	89.74
Dead	4	10.25
Mild hyponatremia	0	
Moderate hyponatremia	2	50%
Severe hyponatremia	2	50%

**DISCUSSION:**

Serum sodium (135-145 mEq/L) is tightly regulated in the body by many mechanisms including but not limited to the hypothalamic osmostat, renal control mechanisms, preserved thirst response and sweating. A change in the serum concentration of most ions in the body produces end organ effects due to the alteration in transmembrane potentials, such as the arrhythmias in hypokalemia or weakness in hypomagnesemia. Dysnatremias, in addition to altering transmembrane potentials, also cause alterations in cell volume due to changes in tonicity [2]. A fall in the serum sodium causes osmotic shift of water from the extracellular to the intracellular compartment, causing cellular swelling and an increase in intracranial pressure.

Many studies have reported hyponatremia in TBI patients, however the incidence varies widely in different studies. Sherlock et al reported an incidence of 9.6% [6], Moro et al in a retrospective analysis of 298 patients with TBI documented an incidence of 16.8% [1], Meng X et al found that one third of their patients with TBI had hyponatremia [7] and Yumoto et al have reported an incidence of 51% [8].

The incidence of hyponatremia was 13% in our study with the incidence of moderate hyponatremia being about 51% while 22.31% developed severe hyponatremia. The serum sodium was checked either daily or on alternate days during the acute phase of the admission. Intracerebral hematoma/ hemorrhagic contusion was the most common radiological abnormality observed in this cohort (63%). Syndrome of inappropriate ADH secretion (SIADH) and cerebral salt wasting (CSW) are often considered to be the likely causes for this situation, however no definite consensus has been reached in literature till date about which should be considered to be the most responsible for the same [1,9-11]. SIADH is often over diagnosed because fluid restriction does raise the serum sodium even in patients with hyponatremia due to other reasons, and this is taken as proof of the diagnosis.

Vingerhoets et al in their prospective study of 256 patients with TBI identified true SIADH as a cause of hyponatremia in only three out of the six suspected SIADH patients [12]. Nelson et al demonstrated reduced blood volumes in 10/12 patients with clinical SIADH emphasizing the fact that the primary problem is the failure of the kidneys to conserve sodium [13]. In a setting of TBI, the use of osmotic diuretics such as mannitol and the presence of an impaired hypothalamo-pituitary adrenal (HPA) axis, would compound the difficulty of diagnosing SIADH [14]. Diringier et al demonstrated that patients on fluid restriction for management of a presumed SIADH are at a higher risk of developing delayed ischaemic neurological deficits [15]. Wijdicks et al in their retrospective study of 134 patients with subarachnoid haemorrhage concluded that restricting fluids for correction of hyponatremia was potentially dangerous and resulted in cerebral infarction [16].

Since ours being a high volume centre for trauma care and having poor patients with limited resources, not much tests could be done to differentiate between the the above to arrive at a definite cause for hyponatremia.

Salt retaining therapy - an alternative approach: Since fluid restriction is potentially hazardous and hyponatremia in TBI is commonly associated with natriuresis, salt retaining therapy using an agent with mineralocorticoid properties would be a logical option in managing such patients. Moro et al recommended the use of salt retaining therapy in managing hyponatremia associated with TBI [1]. Mori et al in a prospective study of thirty patients with aneurysmal SAH demonstrated improved efficacy of hypervolemic therapy, when coupled with the inhibition of natriuresis using fludrocortisone [17]. R. Rajagopal et al, in 2017 published a similar cohort study where Fludrocortisone was used in 36.36% (72/198) of TBI patients with hyponatremia and they found that they had a significantly reduced hospital stay ( $p < 0.05$ ) [18]. This treatment therapy could be a savior and we hope to utilise this in our next batch of patients.

**Drawbacks:**

Apart from hyponatremia, other electrolytes like potassium, magnesium, calcium, parameters like serum albumin, hemoglobin also have an effect on overall recovery in such patients which we have not included in this study. More treatment options apart from oral replenishment and 3% saline therapy could be utilized and results be compared to further our knowledge for early recovery of the patients

**CONCLUSIONS:**

the management of hyponatremia in traumatic brain injury is a critical aspect of patient care that demands a delicate balance between the correction of electrolyte imbalances and the prevention of osmotic demyelination syndrome. As this article has highlighted a thorough understanding of the underlying pathophysiology, regular monitoring of serum sodium levels and the understanding of judicious use of hypertonic alliance solutions are the key to successful management. While the road of recovery for the TBI patients may be challenging, effective management of hyponatremia premium plays a pivotal role in improving their overall prognosis and quality of life. As research in this field continues to evolve, health care professionals must remain vigilant in their efforts leading to better outcomes for those affected by traumatic brain injury.

**REFERENCES:**

- Moro N, Katayama Y, Igarashi T, Mori T, Kawamata T, Kojima J. Hyponatremia in patients with traumatic brain injury: incidence, mechanism, and response to sodium supplementation or retention therapy with hydrocortisone. *Surg Neurol.* 2007 Oct;68(4):387-93.
- Adrogue HJ, Madias NE. Hyponatremia. *N Engl J Med.* 2000 May 25;342(21):1581-9.
- Born JD, Hans P, Smits S, Legros JJ, Kay S. Syndrome of inappropriate secretion of antidiuretic hormone after severe head injury. *Surg Neurol.* 1985 Apr;23(4):383-7. MANUSCRIPT ACCEPTED
- Dóczy T, Tarjányi J, Huszka E, Kiss J. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) after head injury. *Neurosurgery.* 1982 Jun;10(6 Pt 1):685-8.
- Kurokawa Y, Uede T, Ishiguro M, Honda O, Honnou O, Kato T, et al. Pathogenesis of hyponatremia following subarachnoid hemorrhage due to ruptured cerebral aneurysm. *Surg Neurol.* 1996 Nov;46(5):500-507-508.
- Sherlock M, O'Sullivan E, Agha A, Behan LA, Owens D, Finucane F, et al. Incidence and pathophysiology of severe hyponatraemia in neurosurgical patients. *Postgrad Med J.* 2009 Apr;85(1002):171-5.
- Meng X, Shi B. Traumatic Brain Injury Patients With a Glasgow Coma Scale Score of  $\leq 8$ , Cerebral Edema, and/or a Basal Skull Fracture are More Susceptible to Developing Hyponatremia. *J Neurosurg Anesthesiol.* 2016 Jan;28(1):21-6.
- Yumoto T, Sato K, Ugawa T, Ichiba S, Ujiie Y. Prevalence, risk factors, and short-term consequences of traumatic brain injury-associated hyponatremia. *Acta Med Okayama.* 2015;69(4):213-8.
- Lohani S, Devkota UP. Hyponatremia in Patients with Traumatic Brain Injury: Etiology, Incidence, and Severity Correlation. *World Neurosurg.* 2011 Sep;76(3-4):355-60.
- Sterns RH, Silver SM. Cerebral Salt Wasting Versus SIADH: What Difference? *J Am Soc Nephrol.* 2008 Feb 1
- Sivakumar V, Rajshekhar V, Chandy MJ. Management of neurosurgical patients with hyponatremia and natriuresis. *Neurosurgery.* 1994 Feb;34(2):269-274.
- Vingerhoets F, de Tribolet N. Hyponatremia hypo-osmolality in neurosurgical patients. "Appropriate secretion of ADH" and "cerebral salt wasting syndrome." *Acta Neurochir (Wien).* 1988;91(1-2):50-4.
- Nelson PB, Seif SM, Maroon JC, Robinson AG. Hyponatremia in intracranial disease: perhaps not the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). *J Neurosurg.* 1981 Dec;55(6):938-41.
- The Syndrome of Inappropriate Antidiuresis - NEJMcp066837 [Internet]. [cited 2016 Jul 8]. Available from: <http://www.nejm.org/doi/pdf/10.1056/NEJMcp066837>
- Diringier MN, Zazulia AR. Hyponatremia in neurologic patients: consequences and approaches to treatment. *The Neurologist.* 2006 May;12(3):117-26.
- Wijdicks EF, Vermeulen M, Hijdra A, van Gijn J. Hyponatremia and cerebral infarction in patients with ruptured intracranial aneurysms: is fluid restriction harmful? *Ann Neurol.* 1985 Feb;17(2):137-40.
- Mori T, Katayama Y, Kawamata T, Hirayama T. Improved efficiency of hypervolemic therapy with inhibition of natriuresis by fludrocortisone in patients with aneurysmal subarachnoid hemorrhage. *J Neurosurg.* 1999 Dec;91(6):947-52.
- [Rajagopal R, Swaminathan G, Nair S, Joseph M. Hyponatremia in Traumatic Brain Injury: A Practical Management Protocol. *World Neurosurg.* 2017 Dec;108:529-533