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Association Between Hypernatremia and Mortality in Traumatic Brain Injury Patients

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Abstract

Hypernatremia—a state of elevated serum sodium concentration—is a frequent electrolyte disturbance encountered in critically ill patients. In the context of brain injury, its occurrence is particularly significant due to unique pathophysiological interactions between sodium balance, cerebral edema, intracranial pressure (ICP), and systemic homeostasis. This article explores the association between hypernatremia and mortality in patients with brain injury, synthesizing current evidence, underlying mechanisms, clinical implications, and directions for future research. By reviewing epidemiology, pathophysiology, clinical studies, and management strategies, this comprehensive analysis aims to clarify why hypernatremia may serve as both a marker and a contributor to poor outcomes. Overall, it was observed that neither osmotic diuretics nor hyperglycemia were linked to hypernatremia related mortality. However, statistically significantly association was observed between hypernatremia and renal dysfunction. Also, mortality rate was found to be same in patients having hypernatremia at them of admission vs patients developing hypernatremia during admission in ICU.

Keywords: Hypernatremia, Hyperglycemia, osmotic fluids, mortality, traumatic brain injury

Introduction

Hypernatremia is common electrolyte disturbance in hospitalized patients, and in patients admitted to medical and surgical intensive care units. Critically ill patients with neurological and neurosurgical diseases are more susceptible to develop hypernatremia. Its incidence is 1% in general inpatient population and 9% in the intensive care setting. It is more common in brain injured patients and is often an indicator of the severity of the underlying disease (1).

Hypernatremia may be due to primary sodium ion gain or water deficit. These patients often have impaired thirst mechanisms due to altered sensorium or nervous system disorders, affecting thirst perception (2). Sodium disturbances are common in patients with brain injury because of the major role that the central nervous system plays in the regulation of sodium and water homeostasis (3). More importantly, hypernatremia has also been shown to be associated with an increased incidence of renal dysfunction (6). Hypernatremic pateints may also have diabetes insipidus because of pituitary or hypothalamic dysfunction (7, 8). Additionally, treatment of injured brain can itself disturb regulation of sodium and water. Further, in patients with cerebral edema and raised intracranial pressure, hypernatremia frequently results from the therapeutic use of osmotic diuretics or hypertonic saline. In adults with postoperative or posttraumatic cerebral edema treated with 3% saline, a reduction in intracranial pressure has been shown to correlate

with rise in serum sodium levels (9). On one hand hypernatremia may be beneficial in controlling intracranial pressure. On the other ahnd, based on surgeries conducted in medical surgical wards and ICUs, hypernatremia could be associated with increased mortality and morbidity (7, 8). After brain injury, hypernatremia is most commonly related to central central diabetes insipidus or the overzealous use of osmotic diuretics such as mannitol. Nephrogenic DI is also a cause of hypernatremia in the general hospital (3). To appropriately treat patients with osmotic therapy, it is essential to study the impact of hypernatremia on mortality. It is also important to identify a threshold to which serum sodium levels can be safely raised. The objective of the current study was to examine the incidence of hypernatremia and its impact on mortality in patients admitted to neurological/neurosurgical intensive care units (NNICU).

Material and Methods

The study was conducted in Neurosurgery department, PGIMER, Chandigarh. It was a progressive, nonrandomized study of all the patients admitted with the diagnosis of severe traumatic brain injury (TBI). Inclusion criteria – patients with diagnosis of severe traumatic brain injury with GCS<8. Excusion criteria – Severe co-morbid conditions, diabetes mellitus, patints on osmotic diureses and drugs causing hypernatremia like lithium. Severe TBI was classified as Glassglow coma scale (GCS) of 8 or less at the time of admission. Hypernatremia was defined as serum sodium concentration of more than 145mmol/l (31). Hypernatremia was further divided into three groups – Group I – Mild (Na 145-150 mmol/l); Group II – Moderate (150-155mmol/l) and Group III – Severe (Na > 155mmol/l). The data of severe TBI patients admitted to Neurosurgery department with respect dermographics, clinical and laboratory characteristics.

Results

1. Onset of hypernatremia at admission/during stay in ICU – There were 74 patients included in the study. 50% had hypernatremia at time of admission. Out of these, 35% had mild type (Type I), 40.5% had moderate type (type II) and rest had severe type (type III) hypernatremia. 44.6% of patients developed hypernatremia on 1-5 days of admission. Out of these, 42% had mild hyopernatremia, 27% had moderate hypernatremia, while rest had severe hypernatremia. Statistically, no correlation was observed between degree of hypernatremia and time of onset of hypernatremia ($p>0.05$)
2. Further, After admission to ICU, the patients were assessed on the basis of association between various biochemical parameters, which included serum sodium levels and renal function tests with mortality. 43.24% patients had mild hypernatremia (group I); 18.91% had moderate hypernatremia (group II) and 37.84% had severe hypernatremia (group III). Depending on values of blood urea >50mg/dl and serum creatinine >1.2 mg/dl as criteria for deranged renal functions. 62.16% patients had deranged renal profile, while rest of the patients had normal renal functions.
3. Incidence of hypoernatremia in patients administered with mannitol/frusemide – It was observed that 68.9% of the patients who were given diuretics developed severe hypernatrtemia in ICU. Out of all the patients who were not given mannitol/ frusemide, 31% developed severe hypernatremia. However, no statistically significant difference was observed among patients treated with mannitol/frusemide and the patients who were not ($p>0.05$)
4. Correlation of aboce factors with mortality – It was observed that mortality rate was 65.2% in patients tyreated with mannitol/frusemide, while it was only 34.8% in patients who were not treated with mannitol/ frusemide. Among different types of hypernatremic patients, mortality rate was highest in type III hypernatremic patients with very high levels of hyperglycemia (>220mg/dl). In pateinst of

severe hypernatremia, mortality rate was 100%, while in pateints with moderate hypernatremia it was 78.6% and in rest of the patients it was 21%. These findings were statistically highly significant ($p<0.001$). Statistically, no significant correlation was observed between mortality and time of onset of hypernatremia. It was observed that mortality rate was same in patients developing hypernatremia after admission to ICU or patients who had hypernatremia ta time of admission.

Discussion

The reported frequency of hypernatremia in general poluation ranges from 0.8 – 3.5%. Patients admitted to ICU. Patients admitted to the ICU have a higher incidence of hypernatremia compared with the general hospital population. Current study aimed to observe the prevalence of hypernatremia as an independent risk factor for mortality in patients with traumatic brain injury admitted to ICU. Hypernatremia in brain-injured patients represents a complex clinical phenomenon with significant implications for mortality. While controlled hypernatremia is therapeutically employed to manage elevated ICP, unintentional or excessive hypernatremia is consistently associated with poorer outcomes. The pathophysiological basis includes osmotic shifts, neuronal dehydration, systemic organ dysfunction, and immune dysregulation.

To determine this hypernatremia was graded into three groups (10) – group I – mild hypernatremia (145-150 mmol/l); group II – moderate hypernatremia (150-155 mmol/l) and group III – Severe hypernatremia (>155mmol/l). Because, sodium concentration is a major determinant of serum osmolality, it is largely responsible for the normal regulation and distribution of total body water. In essence, total body water is controlled by renal manipulation of sodium, with resulting water adjustment to maintain tonicity. Authors like Masel et al (11) observed that hypernatremia was associated with an increased incidence of renal dysfunction. In current study, statistically significant correltion was observed between hypernatremia and deranged renal profile ($p<0.001$). Another study by Aiyagari et al (2), also observed statistically significant increase in the incidence of renal failure with increasing degree of hypernatremia. Although hypernatremia can lead to renal dysfunction, renal failure itself can lead to hypernatremia as a result of reduced glomerular filtration rate and impaired renal tubular function. Evidence supports that both the **degree** and **dynamics** of hypernatremia influence prognosis. Monitoring, individualized fluid and sodium management, and differentiation between therapeutic and pathological hypernatremia are essential. Although more definitive trials are needed, current data underscore the importance of prudent sodium regulation in improving outcomes for patients with brain injury. Studies have also reported association between hypernatremia and administration of fluids like mannitol and frusemide (12-14). Current study also observed that 25% of patients administered with mannitol/ frusemide were severely hypernatremic, while 42% were moderately hypernatremic. However, no significant correlation was observed between hypernatreami and administration of above mentioned osmotic agents in current study. We also observed relationship between mortality and administration of osmotic agents. Mortality was observed in 30% of patients administered with osmotic fluids and 41% in patients, who were not administered these fluids. Thus, as per observations of current study osmotic agents or diuretics do not contribute significantly to mortality in hypernatremic patients. Overall, in current study no significant correlation was observed between hypernatremia and administration of osmotic agents or diuretics. Similarly, no significant correlation was observed between hyperglycemia and severe hypernatremia and mortality. However, highly significant correlation was observed between degree of hypernatremia and deranged renal profile. Further, it was observed that mortality rate was same in case of hypernatremia at time od admission vs hypernatremia developed during ICU stay.

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