

Evaluation of Serum Electrolytes in Traumatic Brain Injury Patients: Prospective Randomized Observational Study

Research Article

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Received: October 30, 2015 | **Published:** July 28, 2016**Abstract**

Background: Traumatic brain injuries (TBI) are the major public health problem and devastating condition, with significant mortality and morbidity. Electrolyte imbalance after resuscitation in TBI patients are common and further aggravate this condition. The main objectives are prevention and treatment of intracranial hypertension and secondary brain insults, preservation of cerebral perfusion pressure (CPP), and optimization of cerebral oxygenation. Proper in time detection followed by appropriate management not only improves neurological status but also decrease morbidity and mortality.

Aim: We evaluate the electrolyte derangement in traumatic brain injury patients 24 hour after resuscitation.

Method: After Institutional Ethical approval and written informed consent, all TBI patients meeting inclusion criteria were included in this prospective observational study. Serum electrolytes (serum sodium, potassium, calcium and phosphate) were measured at time of admission and 24 hour after resuscitation. All patients received standard treatment according to institutional protocol for TBI patients.

Result: Hyponatremia (27.30%) is the most common electrolyte abnormality followed by hyponatremia (18.73%), Hypokalemia (21.58%), Hyperkalemia (17.77%), hypocalcaemia (11.4%), Hyperphosphatemia (9.8%) followed hypophosphatemia (4.8%) within the first 24 hours after resuscitation.

Conclusion: Electrolyte imbalance following traumatic head injury is an important cause to look for in patient monitoring. Sodium is the chief electrolytes of concern. Serum potassium, calcium and Phosphate levels also under goes notable changes. Based on CT scan findings several traumatic brain injuries associated with various electrolytes derangements are of important concern especially with in first 24 hours after resuscitation.

Keywords: Serum Electrolytes; Traumatic Brain Injury

Introduction

Traumatic brain injuries (TBIs) is a leading cause of morbidity, mortality, disability and socio economic losses in India and in other developing countries. In India, over 100,000 people die due to road traffic accidents each year [1] and nearly 50-60% of them are hospitalized for brain injury [2]. Electrolyte derangements are common sequel of traumatic brain injury. Dyselectrolytemia is very common in head injuries patients and it is likely due to abnormality in serum sodium, potassium, calcium, phosphate. It may be due to use of intravenous fluids, diuretics, syndrome of inappropriate ADH secretion and cerebral salt washing. Serum Sodium is the most common and important electrolyte abnormality responsible among these electrolytes. Both hyponatremia and hypernatremia can result. More so changes in potassium chiefly Hypokalemia [3] and fluid content [4] are also encountered in clinical practice [5]. There are some different causes and among them most common being syndrome of inappropriate anti-diuretic hormone secretion (SIADH) [6], Cerebral salt wasting (CSW) [5] use of diuretics like Furosemide and Mannitol [4]. Age

is another important factor that also greatly affects morbidity and mortality. Advancing age has poor outcome [8] appropriate fluid management of patients with traumatic brain injury (TBI) presents a challenge in most part of the world [9] isotonic fluid can be given without significant fluid disturbances in body [10]. However patients may deteriorate after initial improvement even after a week due to electrolyte disturbances chiefly sodium [11]. So proper management of dyselectrolytemia in such patient following a head injury is most important [9]. Apart from Sodium and Potassium, Serum calcium is also important electrolyte abnormality associated with a variety of clinical manifestations in patients with traumatic brain injury [12]. Initially from the development of tetany [13] to seizures all can happen following derangement in serum calcium. Abnormal responses of neurons to stimulation secondary to accumulation of intracellular calcium in traumatic brain injury are responsible for these features [14]. Both serum hypocalcaemia and hypercalcaemia can occur [12]. Abnormality in serum phosphate following a traumatic brain injury is also been observed. So in our study we also observed the phosphorus level and as we know serum phosphate (Po42)

is also a major intracellular anion and plays an important role in many biochemical pathways relating to normal physiologic functions, especially in maintaining muscle tone [15,16]. As Hypophosphatemia has been shown to be associated with muscle weakness, including weakness of respiratory muscles [17,18], and with respiratory infection [18]. According to study by Aubier et al. [19] they concluded that hypophosphatemia impairs the contractile properties of the diaphragm in critically ill patients, leading to difficulties in weaning from the ventilator. A low Phosphate level may also be associated with decreased cardiac output and with ventricular tachycardia after myocardial infarction [20]. Patients with hypophosphatemia may be asymptomatic or may experience weakness, malaise, anorexia, bone pain, and respiratory arrest [16]. Hypophosphatemia is common in head injury and postoperative patients. Proper in time detection followed by appropriate treatment not only improves neurological status but also decrease morbidity and mortality. This study was conducted to know serum derangements of different electrolytes in patients with traumatic brain injury.

Methods

After Institutional Ethical approval and written informed consent, prospective observational study was conducted at multi-specialty Trauma Centre, Institute of Medical Science, Banaras Hindu University from October 2014 to January 2016. All TBI patients who were admitted in Trauma center Emergency during this period were included in the study. Patients with a history of end-organ dysfunction, hypertension, endocrine disorder, history of allergy, pregnancy and on chronic diuretic therapy were excluded. All TBI patients who were admitted in Trauma center Emergency during this period were included in this study. It included 315 patients in the age group of 15-65 yrs. The severity of head injury was assessed with Glasgow coma scale (GCS) (Table 1) and type of injury was assessed by head computed tomography (CT). Serum electrolytes (serum sodium, potassium, calcium and phosphate) were measured at time of admission in emergency before starting intravenous fluid and repeated at 24 hours after resuscitation. All patient received standard treatment as per institutional protocol for TBI. Serum sodium and Potassium were determined by flame photometer (Coleman). Phosphorus by molybdate reaction in technicon auto analyzer, calcium was determined by fluorometry. Normal reference range for sodium is 130 -145 mEq/L, for potassium is 3.5-5.5 mEq/L, for calcium 8.8-10.5 mg/dL and for Phosphate 2.5-4.5 mg/dL. Outside this range considered as electrolyte imbalance.

Statistical Analysis

A descriptive statistics were applied to express the results. The changes in the electrolyte levels in different types of brain injuries were expressed in percentage. The association between the electrolyte levels and level of consciousness was assessed by using Pearson's correlation coefficient.

Results

A total 315 traumatic brain injury patients got admitted to our multispecialty hospital during our study period from October 2014 to January 2016 and were managed in high dependency unit. Out of which 227 patients (71.5%) were male and 88 patients (27.7%) were females. A total of 280 patients belong to the age

group of 18-50 yrs (89%) and only 35 (11%) patients belongs to age more than 50 years. Among the mechanism of Head injuries the Road Traffic Accident constitutes majority of cases accounting 211 patients (67%) followed by Assault 53 patients (17%) and fall from height/slip which contributes 51 patients (16%) and they are the three leading cause of head injury in our study group. In our study most common NCCT finding at the time of admission were Subarachnoid Hemorrhage (SAH) 112 patients (35%) followed by Subdural Hematoma (SDH) 70 patients (22.53%), Extradural Hematoma (EDH) 52 patients (16.5%), Diffuse Axonal Injury 30 patients (9.5%), Intra cerebral Hemorrhage 20 patients (6.5%), Intra cerebral Hematoma 17 patients (5.5%), diffuse TBI 14 patients (4.5%) were least common (Table 1). Serum sodium was the major electrolyte that underwent significant change followed by Potassium, Calcium and Phosphate. Among 315 patients, Hyponatremia seen in 86 patients (27.30%) and Hyponatremia in 59 patients (18.73%) and in 170 patients no change were observed in Serum Sodium Level (Table 2).

Table 1: Demographic characteristics.

Parameters		Number of Patients (n = 315)	Percentage of Subjects
Age	18-50 years	280	89%
	>50 years	35	11%
Sex	Male	227	71.50%
	Female	88	27.70%
Mechanism of Injury	Road Traffic Accident	211	67%
	Assault	53	17%
	Fall from height/slip	51	16%
NCCT Findings	Sub arachnoid Hemorrhage	112	35%
	Sub dural Hemorrhage	70	22.50%
	Extra dural Hemorrhage	52	16.50%
	Diffuse Axonal Injury	30	9.50%
	Intra cerebral Hemorrhage	20	6.50%
	Intra cerebral Hematoma	17	5.50%
	Diffuse TBI	14	4.50%

Then Hypokalemia observed in 68 patients out of 315 patients (21.58%); Hyperkalemia in 56 patients (17.77%) and in 191 patients (60.16%) had static potassium levels (Table 3). Other electrolytes which we look for were serum Calcium and Phosphate. Hypocalcaemia were present in 36 patients out of 315 patients constituting (11.4%) followed by Hypercalcaemia in 18 Patients (5.7%) and in 261 patients no change in serum calcium level were observed (82.22%) (Table 4). Finally Hyperphosphatemia were found in 31 patients out of 315 patients (9.8%) followed

by Hypophosphatemia in 15 patients (4.8%) and in 269 patients static level of phosphorus were observed (84.73%) (Table 5). We also found a statistically significant ($p < 0.05$) association between observed electrolytes and Glasgow coma scale ($r = 0.639$). The positive correlation implies that lower the observed electrolyte level, lesser is the GCS. Out of total 315 patients 173 patients have Mild GCS (13-15) which constitutes 55 % followed by in 123 patients have Moderate GCS (9-12) (39%) and only 18 patients have severe GCS constitutes 6% (Table 6).

Table 2: Serum sodium (Na⁺) derangements following traumatic brain injury.

Changes in Electrolyte Level	Diagnosis	Number of Subjects (n=315)	Percentage of Subjects
Serum Sodium > 145 MEq/L	Diffuse TBI	12/14	27.30% (86/315)
	Diffuse Axonal Injury	26/30	
	Intracerebral hemorrhage	12/20	
	Extradural hematoma	8/52	
	Subdural hematoma	9/70	
	Subarachnoid Hemorrhage	9/112	
	Intra cerebral hematoma	8/17	
Serum Sodium < 130 MEq/L	Subdural hematoma	32/70	59/315 18.73%
	Subarachnoid Hemorrhage	4/112	
	Extradural Hematoma	16/52	
	Intracerebral hematoma	2/17	
	Diffuse axonal injury	3/30	
	Diffuse TBI	1/14	
No Change in Serum Sodium			(170/315) 53.55%

Discussion

Electrolyte derangements are common in patients with head injury [7]. Most common and notable electrolyte derangements are serum sodium and potassium levels following a head injury [23] Changes in fluid level secondary to resuscitative measures and pharmacological therapy (use of Furosemide and Mannitol) [24] are mainly responsible for these. Volume replacement with isotonic fluids not only is therapeutically of limited efficacy but may aggravate posttraumatic brain edema [25]. Same were the causes in our study and we also experienced difficulty in maintaining fluid balance. More so we also used isotonic saline and Hartman's solution as James HE et al. [10] and the main aim was to achieve normovolemia. To control intracranial pressure (ICP) is very pivotal in traumatic brain injury patients. According to study by Unterberg A et al. [11] they used different pharmacological agents like Mannitol and Furosemide [24]. In our study the incidence of sodium disorders was high (27.30%) which is in accordance with studies by Donati-Genet et al. [26] and Cole et al. [27]. The cause

for hypernatremia could be diabetes insipidus, as 15-30% of TBI patients have hypothalamic-pituitary dysfunction, particularly growth hormone deficiency, ACTH, TSH and gonadotrophin deficiency and diabetes insipidus. Hyponatremia may develop as a result of syndrome of inappropriate secretion of antidiuretic hormone characterized by dilution hyponatremia or cerebral salt-wasting syndrome featured by natriuresis. Similarly a study conducted by Audibert et al. [28] and by Harrigan et al. [29] concluded that ANP (Atrial natriuretic peptide) have a potential role in causing hyponatremia in patients with SAH which results in large amounts of sodium and fluid excretion. The increased excretion of urine occurs due to inhibition of reabsorption of sodium in the collecting duct as concluded by Palmer et al. [30]. Patients with severe head injury are at high risk for the development of Hypokalemia. Low potassium levels in these patients might be due to an increase in their urinary loss, caused by neurologic trauma. Potassium was the second most common electrolyte which underwent significant derangements followed by serum sodium levels. This is in accordance with the study conducted by Pomeranz S et al. [3]. In our study low serum potassium was in 21.58% of patients as compared to 17.77% who had high serum potassium levels. These changes were thought to be due to the large catecholamine discharge that is known to accompany severe head trauma, with resultant beta2-adrenergic stimulation of the Na⁺-K⁺ pump [3].

Table 3: Serum Potassium (K⁺) derangements following traumatic brain injury.

Changes in Electrolyte Level	Diagnosis	Number of Subjects (n=315)	Percentage of Subjects
Serum Potassium > 5 MEq/L	Diffuse TBI	3/14	17.77% (56/315)
	Diffuse Axonal Injury	11/30	
	Intracerebral hemorrhage	5/20	
	Extra dural hematoma	3/52	
	Subdural hematoma	7/70	
	Subarachnoid Hemorrhage	21/112	
	Intra cerebral hematoma	6/17	
Serum Potassium < 3.5 MEq/L	Diffuse TBI	8/14	21.58% (68/315)
	Diffuse Axonal Injury	10/30	
	Intracerebral hemorrhage	9/20	
	Extradural hematoma	8/52	
	Subdural hematoma	9/70	
	Subarachnoid Hemorrhage	15/112	
No Change in Serum Potassium			60.16% (191/315)

Table 4: Serum calcium (Ca²⁺) derangements following traumatic brain Injury.

Changes in Electrolyte Level	Diagnosis	Number of Subjects (n=315)	Percentage of Subjects
Serum Calcium > 10.5 mg/dl	Diffuse TBI	3/14	5.70% (18/315)
	Diffuse Axonal Injury	4/30	
	Intracerebral hemorrhage	3/20	
	Extra dural hematoma	1/52	
	Subdural hematoma	2/70	
	Subarachnoid Hemorrhage	3/112	
	Intra cerebral hematoma	2/17	
Serum Calcium < 8.8 mg/dl	Diffuse TBI	4/14	11.40% (36/315)
	Diffuse Axonal Injury	2/30	
	Intracerebral hemorrhage	8/20	
	Extradural hematoma	4/52	
	Subdural hematoma	5/70	
	Subarachnoid Hemorrhage	12/112	
	Intra cerebral hematoma	3/17	
No Change in Serum Calcium			82.22% (261/315)

Serum calcium changes render a variety of clinical manifestations in patients with traumatic brain injury [12]. Initially from development of tetany [13] to seizures all can happen. Abnormal responses of neurons to stimulation secondary to accumulation of intracellular calcium in traumatic brain injury are responsible for these features [14]. Both serum hypocalcaemia and hypercalcaemia can occur [12]. In our study we had more patients with hypocalcaemia 11.4% than hypercalcaemia 5.7%. We also measured serum phosphate following traumatic brain injury and we observed more Hyperphosphatemia (9.8%) than Hypophosphatemia (4.8%). Most common cause of Hyperphosphatemia is renal insufficiency though other causes include excessive Phosphorus administration, such as enteral and parenteral nutrition therapies, acidosis, hemolysis, Rhabdomyolysis, Tumor lysis syndrome and hypoparathyroidism which is may be associated in patient following traumatic brain injury [31,32]. Some of the established factors associated with Hypophosphatemia include nasogastric suction, liver disease, sepsis, alcoholism, and acidosis associated with diabetic ketoacidosis .Some drugs such as P-binding antacids, catecholamine, beta-adrenergic agonists, Sodium bicarbonate, acetazolamide, and (when administered via a gastro-tube) sucralfate can contribute significantly to the development of Hypophosphatemia [22]. Also, refeeding syndrome occurs due to Hypophosphatemia because of starvation for a period as short as

48 hrs occurs commonly in critically ill patients in the ICU [23]. Poor nutritional status predisposes to this syndrome. Polyuresis induced by cerebral injury increases this risk even further, as demonstrated by the results of our study. Hypophosphatemia is associated with weakness of respiratory muscles [17-19], respiratory infections [18], and ventricular tachycardia [20]. Thus, clinical outcome in ICU patients may be adversely affected by Hypophosphatemia. The mechanism through which patients with severe head injury could be put at risk for the development of electrolyte disorders is unclear. A shift of electrolytes from the extracellular to the intracellular compartment may have taken place; electrolyte loss through induction of polyuresis by cerebral injury may also have played a role. In most ICUs, Na and K are measured routinely at admission in all patients, including those with cerebral injury. However, calcium and phosphorus are not measured on a routine basis; therefore, deficiencies in levels of these electrolytes are likely to remain undetected for a longer period of time. We feel that intensivists and others treating patients with severe head injuries should be aware of this potential problem and that levels of Calcium and Phosphorus should be measured on a routine basis in all patients with severe head injury.

Table 5: Serum Phosphorus (P) derangements following traumatic brain Injury.

Changes in Electrolyte Level	Diagnosis	Number of Subjects (n=315)	Percentage of Subjects
Serum Phosphorus >5 MEq/L	Diffuse TBI	3/14	9.80% (31/315)
	Diffuse Axonal Injury	2/30	
	Intracerebral hemorrhage	7/20	
	Extra dural hematoma	3/52	
	Subdural hematoma	4/70	
	Subarachnoid Hemorrhage	9/112	
	Intra cerebral hematoma	3/17	
Serum Phosphorus <3.MEq/L	Diffuse TBI	1/14	4.80% (15/315)
	Diffuse Axonal Injury	2/30	
	Intracerebral hemorrhage	3/20	
	Extradural hematoma	2/52	
	Subdural hematoma	2/70	
	Subarachnoid Hemorrhage	4/112	
	Intra cerebral hematoma	1/17	
No Change in Serum Phosphorus			84.73% (269/315)

Table 6: Severity of brain injury based on GCS.

Degree of Severity	Number of Patients(n = 315)	Percentage of Subjects
Mild (13-15)	173	55%
Moderate (9-12)	123	39%
Severe (3-8)	18	6%

Conclusion

Electrolyte derangements are very common in patients with head trauma. It is an important and manageable cause of neurological deterioration. Most common derangement is during the first week of injury. But if proper in time detection that is within 24 hours following resuscitation appropriate management of these electrolytes derangements not only improves neurological status but also decrease morbidity and mortality. As Sodium is the most common electrolyte affected. Serum potassium, calcium and Phosphorus level must also be looked for as they plays an important role in prevention of secondary brain insults, preservation of cerebral perfusion pressure (CPP), and optimization of cerebral oxygenation if done within 24 hours following resuscitation.

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