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Evaluation of serum electrolytes imbalance in traumatic brain injury

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Abstract:
Background*: In developing countries like India, Traumatic brain injury (TBI) is one of the predominant causes of morbidity and mortality with great impact on the socioeconomic losses. Every year, 50 - 60% of the road traffic accident patients are being hospitalised for traumatic brain injury. Patients of TBI have high risk of electrolyte derangements and it is likely due to abnormality in serum sodium, potassium, calcium, magnesium. This study was conducted to evaluate serum derangements of different electrolytes in patients with traumatic brain injury.*
Materials and Methods*: This is a prospective study conducted in the department of neurosurgery from October 2019 to June 2020. All adult patients attending emergency department of Gauhati medical college & Hospital(GMCH) with traumatic brain injury with subsequent hospitalisation were included in this study. All those patients who needed ventilator support and those less than 14 years of age were excluded. 314 patients were included. On confirmation of head injury by computed tomography (CT)Head(P), the severity grades of head injury was assessed with glasgow coma scale (GCS) on arrival at the GMCH hospital trauma and emergency department. Serum electrolytes (serum sodium, potassium, calcium and magnesium) were measured at time of arrival in emergency before starting intravenous fluid then once daily for remaining period of hospital stay was carried out.*
Results*: A total of 314 patients were included in this study with mean age of 36.87 ± 16.01years.Hyponatremia (36.3%) is the most common electrolyte imbalance found in this study. Hypernatremia was present in 25.5% patients. Hypokalemia is present in 21.4% patients and hyperkalemia is present in 17.5% patients. Hypocalcemia is present in 14.7% patients and hypercalcemia is present in 8% patients. Hypomagnesemia is present in 6.4% patients and hypermagnesemia is present in 5.4% patients. mortality was seen in 8.6% patients.*
Conclusion: *Electrolyte derangements are most common in patients with traumatic head injury. It is an important and treatable cause of neurologicaldeterioration.*
Key Word*: Traumatic brain injury; Electrolyte imbalance;sodium; potassium; Calcium; Magnesium.*

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I. Introduction

In developing countries like India, Traumatic brain injury (TBI) is one of the predominant causes of morbidity and mortality with great impact on the socioeconomic losses. Every year, 50 - 60% of the road traffic accident patients are being hospitalised for traumatic brain injury[1,2]. Patients of TBI have high risk of electrolyte derangements and it is likely due to abnormality in serum sodium, potassium, calcium, magnesium[3]. It may be due to use of intravenous fluids, syndrome of inappropriate ADH secretion, diuretics and cerebral salt washing. Serum Sodium is the most common and important electrolyte abnormality responsible among all these electrolytes. Potassium changes mainly Hypokalemia and fluid content are noted[4-6]. Most common cause was syndrome of inappropriate anti-diuretic hormone secretion followed by Cerebral salt wasting and use of diuretics[5-7]. Patients may sometimes deteriorates after initial improvement even after a week due to electrolyte disturbances mainly due to sodium[8]. So proper management of dyselectrolytemia is very important[9].

Serum calcium is also one of the important electrolyte abnormality associated with a variety of clinical manifestations in patients with traumatic brain injury[10] mainly development of tetany[11] which may lead to seizures. Abnormal responses of neurons to stimulation secondary to accumulation of intracellular calcium in traumatic brain injury are responsible for these features[12].

Abnormality in serum magnesium following a traumatic brain injury is also been observed. Magnesium has been called "nature's physiological calcium channel blocker" because it appears to regulate the intracellular flow of calcium ions and hypocalcemia is also related with low levels of Mg. There is a strong correlation between Hypomagnesemia and some disorders like ischemic heart disease, hypertension, coronary

vasoconstriction, transient ischemic attacks, cardiac arrhythmias, sudden death, preeclampsia-eclampsia, strokes, seizures, neuromuscular irritability, and diabetes[12-19].

This study was conducted to evaluate serum derangements of different electrolytes in patients with traumatic brain injury.

II. Material And Methods

This is a prospective study conducted in the department of neurosurgery from October 2019 to June 2020. All adult patients attending emergency department of Gauhati medical college & Hospital with traumatic brain injury with subsequent hospitalisation were included in this study. All those patients who needed ventilator support and those less than 14 years of age were excluded. Consent was taken before initiating the study and those patients who are not willing to give consent were excluded from the study. 314 patients were included. On confirmation of head injury by computed tomography (CT)Head(P), the severity grades of head injury was assessed with glasgow coma scale (GCS) on arrival at the GMCH hospital trauma and emergency department. Serum electrolytes (serum sodium, potassium, calcium and magnesium) were measured at time of arrival in emergency before starting intravenous fluid then once daily for remaining period of hospital stay was carried out. All patient received standard treatment as per institutional protocol for TBI. Statistical analysis was performed using SPSS-16.

III. Result

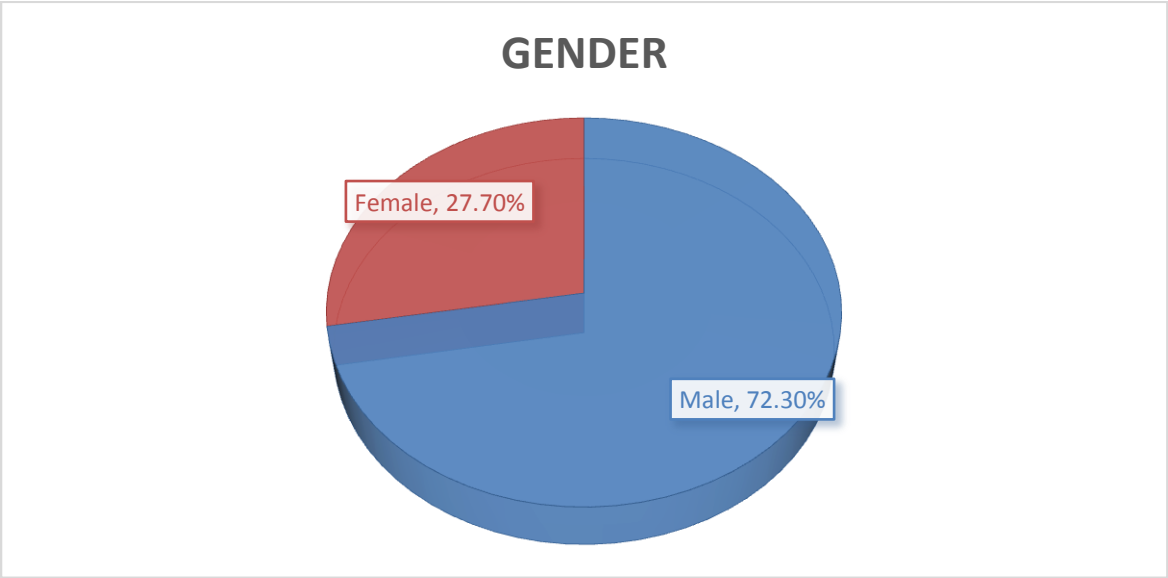

Figure 1: Gender distribution

A total of 314 patients were included in this study with mean age of 36.87 ± 16.01 ranging from 5 years to 85 years. There were 227 males and 87 females. In this study, Most common NCCT findings at the time of admission were Extradural hematoma in 88 (28%) patients followed by Subdural hematoma in 77 (24.5%) patients, Intracerebral hemorrhage in 74 (23.6%) patients, Subarachnoid hemorrhage in 67 (21.3%) patients and Diffuse Axonal Injury in 8 (2.5%) patients. Mean hospital stay in this study was 5.37 ± 1.87 days ranging from 1 day to 8 days.

Out of total 314 patients,92 patients have Mild GCS (13-15) which constitutes 29.3 %, 157 patients have Moderate GCS (9-12) (50%) and 65 (20.7%) patients have severe GCS (</=8).
In this study, mortality was seen in 8.6% patients and 91.4% patients recovered.

Table 1: Incidence of Electrolyte Imbalance

Electrolyte Imbalance		
	Frequency	Percent
Hyponatremia	114	36.3%
Hypernatremia	80	25.5%
No Change	120	38.2%
Hypokalemia	67	21.4%
Hyperkalemia	55	17.5%
No Change	192	61.1%

Hypocalcemia	46	14.7%
Hypercalcemia	25	8.0%
No Change	270	77.3%
Hypomagnesemia	20	6.4%
Hypermagnesemia	17	5.4%
No Change	277	88.2%

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

Change in Electrolye levels	Diagnosis	Frequency	Percent
Hyponatremia	Diffuse Axonal Injury	5/8	36.3% (114/314)
	Extradural hematoma	35/88	
	Intracerebral hemorrhage	30/74	
	Subarachnoid hemorrhage	25/67	
	Subdural hematoma	20/77	
Hypernatremia	Diffuse Axonal Injury	6/8	25.5% 80/314
	Extradural hematoma	20/88	
	Intracerebral hemorrhage	15/74	
	Subarachnoid hemorrhage	17/67	
	Subdural hematoma	22/77	
No Change			38.2% (290/314)

Table 3: Serum potassium (K+) derangements following traumatic brain Injury

Change in Electrolye levels	Diagnosis	Frequency	Percent
Hypokalemia	Diffuse Axonal Injury	2/8	21.4% (67/314)
	Extradural hematoma	16/88	
	Intracerebral hemorrhage	14/74	
	Subarachnoid hemorrhage	16/67	
	Subdural hematoma	19/77	
Hyperkalemia	Diffuse Axonal Injury	1/8	17.5% (55/314)
	Extradural hematoma	20/88	
	Intracerebral hemorrhage	9/74	
	Subarachnoid hemorrhage	10/67	
	Subdural hematoma	15/77	
No Change			61.1% (192/314)

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

Change in Electrolye levels	Diagnosis	Frequency	Percent
Hypocalcemia	Diffuse Axonal Injury	4/8	14.7% (46/314)
	Extradural hematoma	8/88	
	Intracerebral hemorrhage	16/74	
	Subarachnoid hemorrhage	5/67	
	Subdural hematoma	13/77	
Hypercalcemia	Diffuse Axonal Injury	1/8	8.0% (25/314)
	Extradural hematoma	4/88	
	Intracerebral hemorrhage	7/74	
	Subarachnoid hemorrhage	9/67	
	Subdural hematoma	5/77	
No Change			77.3% (270/314)

Table 5: Serum magnesium (Mg2+) derangements following traumatic brain Injury

Change in Electrolye levels	Diagnosis	Frequency	Percent
Hypomagnesemia	Diffuse Axonal Injury	3/8	6.4% (20/314)
	Extradural hematoma	2/88	
	Intracerebral hemorrhage	6/74	
	Subarachnoid hemorrhage	4/67	

	Subdural hematoma	5/77	
Hypermagnesemia	Diffuse Axonal Injury	2/8	5.4% (17/314)
	Extradural hematoma	3/88	
	Intracerebral hemorrhage	4/74	
	Subarachnoid hemorrhage	4/67	
	Subdural hematoma	4/77	
No Change			88.2% (277/314)

Table 6: Association between electrolyte imbalance and GCS score

	GCS </=8	GCS 9-12	GCS 13-15	Total (%)
Hyponatremia	67	30	17	114(36.3%)
Hypernatremia	40	25	15	80(25.4%)
Hypokalemia	27	33	7	67 (21.3%)
Hyperkalemia	25	19	11	55(17.5%)
Hypocalcemia	13	26	7	46(14.6%)
Hypercalcemia	7	9	9	25 (7.9%)
Hypomagnesemia	7	8	5	20(6.3%)
Hypermagnesemia	10	7	0	17 (5.4%)

IV. Discussion

Head injuries are common cause of morbidity and mortality. Electrolyte derangements are common in patients with TBI[20].Patient management of electrolyte balance following head injury is the most important strategy to avoid these.Maintaining of fluidbalance is main stay of treatment. It also greatly affects neurological outcome. Many different type of fluids are used namely hypertonicsaline/dextran solution (HHS). Volume replacement with isotonic fluids not only is therapeutically of limited efficacy but may aggravate posttraumatic brain edema.[21] 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*[22] and the aim was normovolumia. Cintra Ede A *et al*[23] also used these fluids and according to them these are well compensated. However we did not use hypertonic/hyperoncotic saline/dextran solution (HHS) as some of authors did.[21,22]

In this study**,** a total of 314 patients were included in this study with mean age of 36.87 $\pm$ 16.01 ranging from 5 years to 85 years. There were 227 males and 87 females. In this study, Most common NCCT findings at the time of admission were Extradural hematoma in 88 (28%) patients followed by Subdural hematoma in 77 (24.5%) patients, Intracerebral hemorrhage in 74 (23.6%) patients, Subarachnoid hemorrhage in 67 (21.3%) patients and Diffuse Axonal Injury in 8 (2.5%) patients. Mean hospital stay in this study was 5.37 $\pm$ 1.87 days ranging from 1 day to 8 days.

Hyponatraemia is defined as a serum sodium concentration of <135 mmol litre[24] and occurs in up to 15% of the general adult inpatient population. It is more common after brain injury, especially in those patients who are critically ill,[25] usually develops between 2 and 7 days after the injury and is associated with mortality increases of up to 60%.[26,27] In this study, Hyponatremia (36.3%) is the most common electrolye imbalance imbalance found in this study. In a study by Goh KP *et al*.[28] Hyponatremia was a major electrolyte abnormality. Previous studies have suggested that 27 – 41% of TBI patients develop hyponatremia[29,30] and 51% of TBI develop mild hyponatremia whoever 20 % of them develop moderate and severe hyponatremia [31]. Another study proved that TBI patients develop hyponatremia only in 16.8 % [29].In this study, Hypernatremia was present in 25.5% patients.The causefor hypernatremia could be diabetes insipidus, hypothalamicpituitary dysfunction and use of mannitol.

Potassium was the second most common electrolyte which underwent significant derangements followed by serum sodium levels. This is in accordance with the study by Pomeranz S et al.[4]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. In this study, Hypokalemia is present in 21.4% patients and hyperkalemia is present in 17.5% patients. These changes were thought to be due to the large catecholamine discharge that is known to accompany severe head trauma, with resultant beta 2-adrenergic stimulation of the Na+-K+ pump.[4]

Serum calcium changes render a variety of clinical manifestations in patients with traumatic brain injury[10] In this study, Hypocalcemia is present in 14.7% patients and hypercalcemia is present in 8% patients.

In this study, Hypomagnesemia is present in 6.4% patients and hypermagnesemia is present in 5.4% patients. In this study, mortality was seen in 8.6% patients and 91.4% patients recovered. In a study by Mokhtari et al[32], mortality rates were significantly higher in patients with hyponatremia and hypernatremia than in other patients.

V. Conclusion

Electrolyte derangements are most common in patients with traumatic head injury. It is an important and treatable cause of neurologicaldeterioration. Most commonly, electrolyte derangements occur during the first week of injury. Proper and timely management not only improves neurological status but alsodecreases morbidity andmortality. As Sodium is the most common electrolyte affected. Serum potassium, calcium and magnesium levelsmust also be looked for as they play an important role in prevention of secondary brain insults, preservation ofcerebral perfusionpressure, and optimization of cerebral oxygenation if done within 24 hours following resuscitation.

References

[1]. Dandona R, Kumar GA, Ameer MA, Ahmed GM, Dandona L. Incidence and burden of road traffic injuries in urban India. Inj Prev.2008; 14(6): 354-359.
[2]. Burden of disease in India. National Commission on Macroeconomics and Health background papers, Ministry of Health & Family Welfare. New Delhi, India:2005.
[3]. Lath R. Hyponatremia in neurological diseases in ICU. Indian J, Crit. Care Med.2005; 9:47-51
[4]. Pomeranz S, Constantini S, Rappaport ZH. Hypokalaemia in severe head trauma. Acta Neurochir.1989; 97(1-2): 62-66.
[5]. Gaab M, Knoblich OE, Schupp J, Herrmann F, Fuhrmeister U. Effect of furosemide on acute severe experimental cerebral edema. J Neurol.1979; 220(3): 185-197.
[6]. Askar A, Tarif N. Cerebral salt wasting in a patient with head trauma: management with saline hydration and 6ludrocortisone. Saudi J Kidney Dis Transpl.2007; 18(1): 95-99.
[7]. Gribkov AV, Fraerman AP, Salalykin VI, Salmin AA, Sidorkin VG; Regulation of the water-electrolyte balance during neurosurgical operations with balanced anesthesia using sodium oxybutyrate. Anesteziol Reanimatol.1992; (1): 28-31.
[8]. Unterberg A, Kiening K, Schmiedek P, Lanksch W. Long-term observations of intracranial pressure after severe head injury. The phenomenon of secondary rise of intracranial pressure. Neurosurgery.1993; 32(1): 17-23.
[9]. Ishizaki T, Momota H, Kuwahara K, Tanooka A, Morimoto S. A case of symptomatic traumatic cerebral vasospasm associated with hyponatremia. No Shinkei Geka.1999; 27(11): 1031-1036.
[10]. Myshkin KI, Chuenkov VF. Changes in blood serum calcium levels in acute cranio cerebral injury. Vopr Neirokhir.1963; 27: 26-28.
[11]. Kogan OG, Kaishibaev SK. A case of tetany developing after brain injury. Zdravookhranenie Kazakhstana.1961; 21(10): 68-70.
[12]. Gurkoff GG, Shahlaie K, Lyeth BG. In vitro mechanical strain trauma alters neuronal calcium responses: Implications for posttraumatic epilepsy. Epilepsia.2012; 53(Suppl 1): 53-60.
[13]. Gupte M D, Ramachandran V, Utatkar RK. Epidemiological profile of India: Historical and contemporary perspectives. J. Biosci.2011: 26(4); 437–464.
[14]. Alves OL, Bullock R. "Excitotoxic damage in traumatic brain injury". In Clark RSB, Kochanek P. Brain injury. Boston: Kluwer Academic Publishers.2001; 1. ISBN 0-7923-7532-7. Retrieved 2008-11-28.
[15]. Hannay HJ, Howieson DB, Loring DW, Fischer JS, Lezak MD. "Neuropathology for neuropsychologists". In Lezak MD, Howieson DB, Loring DW. Neuropsychological Assessment. Oxford [Oxfordshire]: Oxford University Press.2004; 158–62. ISBN 0-19-511121-4.
[16]. Nadler JL, Rude RK: Disorders of magnesium metabolism. Endocrinol Metab Clin North Am 1995; 24:623– 641
[17]. Altura BM, Altura BT: Magnesium, electrolyte transport and coronary vascular tone. Drugs 1984; 28(Suppl 1):120–142
[18]. Whang R, Hampton EM, Whang DD: Magnesium homeostasis and clinical disorders of magnesium deficiency. Ann Pharmacother 1994; 28:220–226.
[19]. Weisinger JR, Bellorın-Font E: Magnesium and phosphorus. Lancet 1998; 352:9125, 391–396.
[20]. Marik PE, Bedigian MK. Refeeding hypophosphatemia in critically ill patients in an intensive care unit: A prospective study. Arch Surg.1996; 131(10):1043-1047.
[21]. Berger S, Schürer L, Härtl R, Deisböck T, Dautermann C, Murr R, et al. 7.2% NaCl/10% dextran-60 versus 20% mannitol for treatment of intracranial hypertension. Acta Neurochir Suppl (Wien) 1994;60:494–8
[22]. James HE, Schneider S. Effects of acute isotonic saline administration on serum osmolality, serum electrolytes, brain water content and intracranial pressure. Acta Neurochir Suppl (Wien) 1993;57:89–93
[23]. Cintra Ede A, Araújo S, Quagliato EM, Castro M, Falcão AL, Dragosavac D, et al. Vasopressin serum levels and disorders of sodium and water balance in patients with severe brain injury. Arq Neuropsiquiatr 2007;65(4B):1158–65
[24]. Levin ER, Gardner DG, Samson WK. Natriuretic peptides. N Engl J Med 1998; 339: 321–8
[25]. Rabinstein AA, Wijdicks EF. Hyponatremia in critically ill neurological patients. Neurologist 2003; 9: 290–300
[26]. Tisdall M, Crocker M, Watkiss J, Smith M. Disturbances of sodium in critically ill neurologic patients. J Neurosurg Anesthesiol 2006; 18: 57–63
[27]. Diringer MN, Zazulia AR. Hyponatremia in neurologic patients: consequences and approaches to treatment. Neurologist 2006; 12: 117–26.
[28]. Goh KP. Management of hyponatremia. Am Fam Physician 2004;69:2387–94
[29]. 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; 68: 387-93.
[30]. Chitsazian Z, Zamani B and Mohagheghfar M. Prevalence of hyponatremia in intensive care unit patients with brain injury in kashan shahid-beheshti hospital in 2012. Arch Trauma Res.2013; 2: 91-94.
[31]. Yomoto T, Sato K, Ugawa T, Ishiba S and Ujike U. Prevalence, Risk Factors, and Short-term Consequences of Traumatic Brain Injury-associated Hyponatremia. Acta med okayama. 2015; 69 (4): 213-18.
[32]. Mokhtari M, Goharani R, Miri M, Araghi PE. Frequency of Hyperand Hypo-natremia in Patients admitted in the ICU and comparison of their association with mortality. Res Med. 2010; 33(3):183-8