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Evaluation of sodium and potassium levels in early head trauma at Tertiary Care Teaching Centre

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Abstract

Introduction: Head trauma is a major public health problem accounting for thousands of admissions each year and costing the healthcare system billions of dollars. The majority of patients with head trauma are seen in the emergency department; the head injury is often associated with other organ injuries as well. Of all serum electrolytes, the most common electrolyte subject to imbalance in TBI patients is serum sodium. The most common electrolyte imbalance condition in TBI was hypernatremia followed by hyponatremia and hypokalemia. Hyponatremia as being a common electrolyte disturbance in cases of TBI, which was most often caused by a disorder known as the syndrome inappropriate antidiuretic hormone (SIADH).

Material and Methods: This is a prospective and observational study was conducted in the Department of Biochemistry, Shadan Institute of Medical Sciences, Teaching Hospital and Research center among 45 patients admitted to our ICU were enrolled in the study. Either gender between 18-65 years, Injury withing first 12-24 hours including mild, moderate and severe head injury and Resuscitation according to ACLS protocol included. Patients in shock with a low Glasgow Coma Scale (GCS) score or any polytrauma patient where initial aggressive resuscitation was done outside ICU were excluded from the study.

Results: In our study, Males are Affected More than female. In our study, most the (14) patients were age group 20-30 years followed by 13 patients were 50-60 years, 11 patients were 30-40 years and 7 patients were 40-50 years. In our study, Majority Patients are Grades of Injury are Mild. Out of 45 patients, 12 patients had disturbance of K^+ Which is quite less common that hyponatremia but it was more noticeable. In our study, out 45 patients, 6 patients had hyponatremia. Na^+ 130 for contusion and SAH with GCS of 8/15 (Severe TBI). Na^+ 131 for diffusion axonal injury with GCS of 6/15 (Severe TBI). Na^+ 128 for diffusion axonal injury with GCS of 3/15, 8/15 (Severe TBI) in 2 patients. Na^+ 135 in 2 cases with contusions and EDH, SDH.

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Conclusion: Our study demonstrated that patients with TBI have a very strong propensity to develop electrolyte imbalance, particularly hyponatraemia and hypokalaemia. Hyponatremia may be the result of SIADH or cerebral salt wasting syndrome that is common in TBI. Hyponatremia in SAH results from the release of ANP (atrial natriuretic peptide) that can cause fluid and sodium loss in urine. Hypokalaemia may also be the result of urinary loss of potassium.

Keywords: Sodium, Potassium, Head trauma

Introduction

Head trauma is a major public health problem accounting for thousands of admissions each year and costing the healthcare system billions of dollars. [1] The majority of patients with head trauma are seen in the emergency department; the head injury is often associated with other organ injuries as well. [2] The care of a patient with head trauma is by an interprofessional team that is dedicated to managing head trauma patients. [3] Traumatic brain injury (TBI) is a common presentation in emergency departments, which accounts for more than one million visits annually. [4] It is a common cause of death and disability among children and adults. [5] The leading causes of head trauma are (1) motor vehicle-related injuries, (2) falls, and (3) assaults. Based on the mechanism, head trauma is classified as (1) blunt (the most common mechanism), (2) penetrating (most fatal injuries), (3) blast. [6]

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. [7] Head trauma is more common in children, adults up to 24 years, and those older than 75 years. TBI is 3 times more common in males than in females. [8] Although only 10% of TBI occurs in the elderly population, it accounts for up to 50% of TBI-related deaths. [9]

Electrolyte imbalance in cases of TBI is associated with the pathology of the brain itself or is iatrogenic in causation. [10] Unknown pre-existing conditions such as renal failure, cirrhosis, or congestive heart failure share the cause. It is common and considered as one of several preventable secondary injuries. [11] The risk to the development of electrolyte disturbance in TBI patients depends on the severity of head injury, underlying disease, age, and primary therapeutic strategy such as the choice of resuscitation fluid, administration of mannitol or diuretics, and hyperventilation. [12]

Of all serum electrolytes, the most common electrolyte subject to imbalance in TBI patients is serum sodium. [13] The most common electrolyte imbalance condition in TBI was hypernatremia followed by hyponatremia and hypokalemia. Hyponatremia as being a common electrolyte disturbance in cases of TBI, which was most often caused by a disorder known as the syndrome inappropriate antidiuretic hormone (SIADH). [14] Transient hypothalamic-pituitary adrenal (HPA) dysfunction and secondary adrenal insufficiency (AI) were reported in moderate to severe TBI patients and these conditions may cause sodium disturbance. [15] Many previous studies have reported variations in the incidence and type of electrolyte imbalance in TBI patients but not in association with the morbidity and mortality.

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Material and Methods:

This is a prospective and observational study was conducted in the Department of Biochemistry, Shadan Institute of Medical Sciences, Teaching Hospital and Research center among 45 patients admitted to our ICU were enrolled in the study.

Inclusion criteria:

Either gender between 18-65 years.
Injury withing first 12-24 hours including mild, moderate and severe head injury.
Resuscitation according to ACLS protocol

Exclusion criteria:

Duration more than 24 hours after injury.
Patients at extremes of age (<18 years and >65 years) were excluded. Also, patients with chronic kidney disease, diabetic patients on insulin or oral hypoglycaemic agents (OHA), and patients of heart failure on diuretics were excluded from the study.

Patients in shock with a low Glasgow Coma Scale (GCS) score or any polytrauma patient where initial aggressive resuscitation was done outside CICU were excluded from the study. Blood investigations viz. complete blood count (CBC), random blood sugar (RBS), liver function test (LFT), kidney function test (KFT), serum sodium, potassium, calcium, magnesium, and phosphorus along with other investigations as necessary for the individual cases were sent routinely as par CICU protocol. Daily and precise monitoring of input-output was done including all losses via urine, stool, insensible water loss, drain/s, and suctioning. Patients were followed till discharge or demise from CICU and or till baseline values returned to normal. Results obtained were charted and analysed.

Daily measurement of serum sodium, potassium, calcium, magnesium, phosphate, chloride, and alternate day measurement of blood urea, serum creatinine, and albumin was done. The mean age, sex distribution, mean GCS at the time of initial evaluation, mean duration of their ICU stay, and variation in the mean electrolyte profile (of sodium, potassium, calcium, magnesium, phosphate, and chloride) during the course of their stay in the ICU.

Results:

In our study, Males are Affected More than female in Table 1.

Table 1: Distribution of Gender

Gender	Frequency	Percentage
Male	37	82.2
Female	8	17.8
Total	45	100

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Table 2: Distribution of Age group

Age Group (Years)	Frequency	Percentage
20-30 years	14	31.2
31-40 years	11	24.4
41-50 years	7	15.5
51-60 years	13	28.9
Total	45	100

In our study, most the (14) patients were age group 20-30 years followed by 13 patients were 50-60 years, 11 patients were 30-40 years and 7 patients were 40-50 years in Table 2.

Table 3: Distribution of Grades of injury

Grades of injury	Frequency	Percentage
Mild TBI	33	73.4
Moderate TBI	6	13.3
Severe TBI	6	13.3
Total	45	100

In our study, Majority Patients are Grades of Injury are Mild in table 3.

Table 4: Distribution of Hypokalaemia

Hypokalaemia	Frequency	Percentage
Concussion	4	73.4
SDH	3	13.3
Contusions	4	13.3
DAI	1	100
Total	12	100

Out of 45 patients, 12 patients had disturbance of K^+ Which is quite less common that hyponatremia but it was more noticeable in table 4.

Hyponatremia

In our study, out 45 patients, 6 patients had hyponatremia.

Na^+ 130 for contusion and SAH with GCS of 8/15 (Severe TBI).

Na^+ 131 for diffusion axonal injury with GCS of 6/15 (Severe TBI).

Na^+ 128 for diffusion axonal injury with GCS of 3/15, 8/15 (Severe TBI) in 2 patients.

Na^+ 135 in 2 cases with contusions and EDH, SDH.

Discussion

In our study, we found that patients with traumatic brain injury are more likely to develop electrolyte imbalance than patients without head injury. This was consistent with the findings by Viera AJ et al. [16] Most of the patients were male in both groups. The most common

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electrolyte imbalance in patients with traumatic brain injury was hyponatremia followed by hypokalaemia. This was consistent with the finding by Zeiler FA et al. [17] Other important electrolyte deficiencies noted were hypocalcaemia, hypophosphatemia, and hypomagnesemia. This was consistent with the study by Manuel VR et al. [18]

Hyponatremia following TBI can be due to syndrome of inappropriate ADH secretion (SIADH) or cerebral salt wasting syndrome, but it can also be due to decreased serum albumin following acute phase response. [19, 20] Hypomagnesemia is commonly found in patients with hypoalbuminemia. Hypomagnesemia is in turn commonly associated with hypokalaemia (refractory to potassium supplement), hypocalcaemia, and hypophosphatemia. [21] While calcium has an integral role in excitation-contraction coupling of the airway smooth muscles, hypophosphatemia is associated with respiratory muscle weakness and difficulty in weaning from mechanical ventilation. Thus long-term deficiency of either of these electrolytes can increase the duration as well as the cost of ICU stay and also associated with patient morbidity as well as mortality.

Hyponatremia may develop as a result of syndrome of inappropriate secretion of ant diuretic hormone characterized by dilution hyponatremia or cerebral salt-wasting syndrome featured by natriuresis. Similarly a study conducted by Tehse J et al. [22] concluded that ANP (Atrial naturetic 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 Kuhna V et al. [23]

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 Vinas-Rios J et al. [24] 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. [25]

Limitations of the study

The sample size was small. Plasma and urine osmolarity could not be done that could differentiate between SIADH and cerebral salt wasting. Total calcium was used instead of corrected calcium or ionised calcium levels. As our ICU is six bedded, only the severe TBI patients were admitted and included in the study. So our study could very well miss the profile of mild to moderate TBI.

Conclusion

Our study demonstrated that patients with TBI have a very strong propensity to develop electrolyte imbalance, particularly hyponatraemia and hypokalaemia. Hyponatremia may be

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the result of SIADH or cerebral salt wasting syndrome that is common in TBI. Hyponatremia in SAH results from the release of ANP (atrial natriuretic peptide) that can cause fluid and sodium loss in urine. Hypokalaemia may also be the result of urinary loss of potassium.

References

1. Farrell D, Bendo AA. Perioperative Management of Severe Traumatic Brain Injury: What Is New? Curr Anesthesiol Rep. 2018;8(3):279–89.
2. Galgano M, Toshkezi G, Qiu X, Russell T, Chin L, Zhao L-R. Traumatic Brain Injury: Current Treatment Strategies and Future Endeavors. Cell Transplant. 2017 Jul 1;26(7):1118–30.
3. Dang B, Chen W, He W, Chen G. Rehabilitation Treatment and Progress of Traumatic Brain Injury Dysfunction. Neural Plast. 2017;2017(2017):1582182.
4. Vella MA, Crandall M, Patel MB. Acute Management of Traumatic Brain Injury. Surg Clin North Am. 2017 Oct;97(5):1015–30.
5. Kinyanjui B. Traumatic Brain Injury in Kenya: A Preliminary Review of the Literature. SAGE Open. 2016 Jan 1;6(1):2158244016638392.
6. Wong JC, Linn KA, Shinohara RT, Mateen FJ. Traumatic brain injury in Africa in 2050: a modeling study. Eur J Neurol. 2016;23(2):382–6.
7. Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GWJ, Bell MJ, et al. Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery. 2017 Jan 1;80(1):6– 15.
8. Pin-On P, Saringkarinkul A, Punjasawadwong Y, Kacha S, Wilairat D. Serum electrolyte imbalance and prognostic factors of postoperative death in adult traumatic brain injury patients: A prospective cohort study. Medicine (Baltimore). 2018 Nov;97(45):e13081.
9. Suman S, Kumar N, Singh Y, Kumar K, Yadav G, Gupta B. Evaluation of Serum Electrolytes in Traumatic Brain Injury Patients: Prospective Randomized Observational Study. J Anesth Crit Care Open Access. 2016 Jul 28;5(3):1–0.
10. Hoffman H, Jalal MS, Chin LS. Effect of Hypernatremia on Outcomes After severe Traumatic Brain Injury: A Nationwide Inpatient Sample analysis. World Neurosurg. 2018 Oct;118:e880–6.
11. Tan SKR, Kolmodin L, Sekhon MS, Qiao L, Zou J, Henderson WR, et al. The effect of continuous hypertonic saline infusion and hypernatremia on mortality in patients with severe traumatic brain injury: a retrospective cohort study. Can J Anaesth J Can Anesth. 2016 Jun;63(6):664–73.
12. Vedantam A, Robertson CS, Gopinath SP. Morbidity and mortality associated with hypernatremia in patients with severe traumatic brain injury. Neurosurg Focus. 2017 Nov;43(5):E2.
13. Shen B, Li L, Li T. Concurrence of Inappropriate Antidiuretic Hormone Secretion and Cerebral Salt Wasting Syndromes after Traumatic Brain Injury. Front Neurosci. 2017;11:499.

ISSN:0975-3583,0976-2833 VOL13, ISSUE 08, 2022

14. Tudor RM, Thompson CJ. Posterior pituitary dysfunction following traumatic brain injury: review. Pituitary. 2018 Oct 17;
15. Ookuma T, Miyasho K, Kashitani N, Beika N, Ishibashi N, Yamashita T, et al. The clinical relevance of plasma potassium abnormalities on admission in trauma patients: a retrospective observational study. J Intensive Care. 2015;3(1):37.
16. Viera AJ, Wouk N. Potassium Disorders: Hypokalemia and Hyperkalemia. Am Fam Physician. 2015 Sep 15;92(6):487–95.
17. Zeiler FA, Thelin EP, Helmy A, Czosnyka M, Hutchinson PJA, Menon DK. A systematic review of cerebral microdialysis and outcomes in TBI: relationships to patient functional outcome, neurophysiologic measures, and tissue outcome. Acta Neurochir (Wien). 2017;159(12):2245–73.
18. Manuel V-R, Martin S-A, Juan S-R, Fernando M-A, Frerk M, Thomas K, et al. Hypocalcemia as a prognostic factor in mortality and morbidity in moderate and severe traumatic brain injury. Asian J Neurosurg. 2015;10(3):190.
19. Porter C, Sousse LE, Irick R, Schryver E, Klein GL. Interactions of Phosphate Metabolism With Serious Injury, Including Burns. JBMR Plus. 2017 Oct;1(2):59–65. 52.
20. Nayak R, Attry S, Ghosh SN. Serum magnesium as a marker of neurological outcome in severe traumatic brain injury patients. Asian J Neurosurg. 2018 Jul 1;13(3):685.
21. Taha M, Ammar M. Electrolyte And Acid Base Disturbances In Patients With Severe Closed Traumatic Brain Injury. Internet J Neurosurg. 2015;11(1):1–7.
22. Tehse J, Taghibiglou C. The overlooked aspect of excitotoxicity: Glutamate-independent excitotoxicity in traumatic brain injuries. Eur J Neurosci. 2019 May;49(9):1157–70.
23. Kuhna V, Vinas-Rios J, Kretschmer T, Heinen C. Lactate and Hypocalcaemia as Possible Prognostic Factors of Mortality and Morbidity in Early Phases of Moderate and Severe Traumatic Brain Injury. Clin Surg. 2019;4:4.
24. Vinas-Rios J, Kretschmer T, Medina-Govea F, Heinen C. Hypocalcaemia and Its Role in Traumatic Brain Injury. J Neurol Disord. 2017;05(03):1000342.
25. Eisner David A., Caldwell Jessica L., Kistamás Kornél, Trafford Andrew W. Calcium and Excitation-Contraction Coupling in the Heart. Circ Res. 2017 Jul 7;121(2):181–95.