

Correlation Between the Severity of Head Injury And Electrolytes in Patients with Traumatic Brain Injury

Dr Anshul Jain¹, Dr Binod Gouda¹, Dr Ritesh Gajjar¹, Dr P.B. Gupta²

¹Resident Doctor, Department of Emergency Medicine, Government Medical College Surat.

²Professor & Head, Department of Emergency Medicine, Government Medical College Surat.

Corresponding Author: Dr Anshul Jain

Abstract

Introduction: Traumatic brain injury (TBI) occurs whenever mechanical force injures the brain. Electrolyte derangements are common after neurologic injury.

The role of electrolyte imbalance is being delineated in severe cranial trauma and is an essential investigation for its therapeutic managements.

Objectives: This study is designed to ascertain Correlation between the severity of head injury and electrolytes in patients with traumatic Brain injury.

Material and Methods: This observational study was carried out in New Civil Hospital Surat, Gujarat. The total number of patients who fulfilled the inclusion and exclusion criteria were 84. Data was collected in the preformed proforma and analysed for the result. We measured serum levels of sodium and potassium in all these patients.

Results: Out of these 84 Patients, 46(54.7%) had mild injury, 20(23.8%) had moderate injury and 18(21.4%) had severe injury. Out of these 84 patients, 69(82.1%) had normal sodium levels, 13(15.4%) had low sodium levels and 2(2.3%) had high sodium levels whereas 73(86.9%) had normal potassium levels, 10(11.9%) had low potassium levels and 1(1.1%) had high potassium levels. Out of 18 severe injuries, 10(55.5%) had abnormal sodium levels and 3(16.6%) had abnormal potassium levels which were found to be significant ($p < 0.0001$).

Conclusion: we conclude that patients with severe brain injury have significantly deranged sodium and potassium levels as compared to mild and moderate brain injuries.

Keywords: Traumatic brain injury, sodium levels, potassium levels

Date of Submission: 26 -08-2017

Date of acceptance: 09-09-2017

I. Introduction

TBI is a leading cause of death and disability worldwide. It is the leading cause of brain damage in children and young adults and plays a significant role in half of trauma deaths¹. Because a head injury occurs every 15 seconds and a patient dies from head injury every 12 minutes, a day does not pass that an emergency physician is not confronted with a head injured patient². The consequences of TBI results in disability with lifelong financial, medical, emotional, family trauma¹. In this study we establish a correlation between the severity of head injury and serum electrolytes. Hyponatremia is a common electrolyte disturbance following intracranial disorders^{3,4}. Hyponatremia is of clinical significance as a rapidly decreasing serum sodium concentration as well as rapid correction of chronic hyponatremia may lead to neurological symptoms⁵. K is found in high concentration in cell with comparatively low extracellular concentration levels. Small Changes in K ions can severely affect nerve conduction, heart rhythm and muscle contraction⁶. Cerebral injury can lead to polyuresis through a variety of mechanisms. In addition, the role of electrolyte abnormalities in the secondary neurologic injury cascade is being delineated and may offer a potential future therapeutic intervention⁶. Aim of our study is find the correlation of severity of head injury with serum electrolytes.

II. Materials And Methods

During the period of four months i.e., from August 2016 to November 2016, the cross-sectional clinical observation study on “Correlation between the severity of head injury and electrolytes in patients with traumatic Brain injury” has been done at the New Civil Hospital, Surat. The required permission from ethics committee was obtained. The total number of patients who fulfilled the inclusion and exclusion criteria were 84. Data was collected in the preformed proforma and analysed for the result.

III. Results

1. Age Group Distribution :

- In our Cross-sectional clinical observational study, 84 cases were selected. Outof these, the maximum number of cases was within the age group of 21-30 years (44 %). The minimum age of the patient in our series was 1year and the maximum age was 86 years. The mean age was 34years.

Table No.1. Age wise distribution of patients

| Age in years | Total No. of Patients | Percentage |
|--------------|-----------------------|-------------|
| 01-10 | 7 | 8.33 % |
| 11-20 | 12 | 14.2 % |
| 21-30 | 21 | 44.04 % |
| 31-40 | 15 | 17.8 % |
| 41-50 | 16 | 19.04 % |
| 51-60 | 12 | 14.2 % |
| 61-70 | 0 | 0 % |
| 71-80 | 0 | 0% |
| 81-90 | 1 | 1.1% |
| TOTAL | 84 | 100% |

2. Gender Distribution of the study population(n=84)

There were 70 males and 14 females, with male to female ratio 5:1. There was a significant preponderance of male patients.

Table No.2. Gender wise Distribution

| Sex | Total Number of Patients | Percentage |
|--------------|--------------------------|------------|
| Male | 70 | 83.33 % |
| Female | 14 | 16.66 % |
| Total | 84 | 100 |

3. Mode of injury (n=84)

Road traffic accident was the most common mechanism of injury (70%), followed by fall from height (27%) and Assault (2%). Blast injury or firearm related traumatic brain injury was not reported in our study.

Table No.3. Distribution according to mode of injury (n=84)

| Mode of injury | No of Patients | Percentage |
|------------------|----------------|------------|
| Fall from Height | 23 | 27.3 % |
| RTA | 59 | 70.2 % |
| Assault | 2 | 2.3 % |
| Total | 84 | 100 |

4. Risk factors related to trauma (n=84)

Most common risk factor related to trauma found in our study was vomiting (52%) followed by unconsciousness (45%), Ear-nose bleeding (17%), convulsion (11%) and amnesia (3.5%).

| Risk Factors Related to Trauma | No. of Patients | Percentage |
|--------------------------------|-----------------|------------|
| Vomiting | 44 | 52.3% |
| Convulsion | 9 | 10.71% |
| Unconsciousness | 38 | 45.2% |
| Ear / Nose Bleeding | 14 | 16.66% |
| Amnesia | 3 | 3.5% |

5. Severity of Traumatic Brain Injury (n=84)

55% of total patients in our study had mild head injury, 24% of patients had moderate head injury and 21% of patients had severe head injury.

| Severity of head injury | No of Patients | Percentage |
|-------------------------|----------------|-------------|
| Mild(13-15 GCS) | 46 | 54.76% |
| Moderate (9-12 GCS) | 20 | 23.8% |
| Severe (<9 GCS) | 18 | 21.42% |
| Total | 84 | 100% |

6. Electrolyte findings in 84 Patients

| | Hyper-natremia | Normo-natremia | Hypo-natremia | Hyper-kalemia | Normo-kalemia | Hypo-kalemia |
|------------|----------------|----------------|---------------|---------------|---------------|--------------|
| Mild TBI | 0 | 44 | 2 | 0 | 44 | 2 |
| Mod. TBI | 0 | 17 | 3 | 0 | 14 | 6 |
| Severe TBI | 2 | 8 | 8 | 1 | 15 | 2 |
| Total | 2 | 69 | 13 | 1 | 73 | 10 |

TBI= Traumatic brain injury

Sodium and potassium levels of these 84 patients were (mean \pm 2 SD) 137.15 \pm 5.25 and 4.06 \pm 0.53 respectively. Out of these 84 patients, 69(82.1%) had normal sodium levels(135-145 mmol/l), 13(15.4%) had low sodium levels(<135 mmol/l) and 2(2.3%) had high sodium levels(>145 mmol/l) whereas 73(86.9%) had normal potassium levels(3.5-5.5 mmol/l), 10(11.9%) had low potassium levels(<3.5 mmol/l) and 1(1.1%) had high potassium levels(> 5.5 mmol/l). Out of 18 severe injuries, 10(55.5%) had abnormal sodium levels($p < 0.00011$) and 3(16.6%) had abnormal potassium levels which were found to be significant($p < 0.0001$).

IV. Discussion

Our results clearly demonstrate that the patients with severe head injury are at a greater risk of developing electrolyte imbalance mostly hyponatremia and hypokalemia. Hyponatremia may develop as a result of syndrome of inappropriate secretion of antidiuretic hormone characterized by dilutional hyponatremia or cerebral saltwasting syndrome featured by natriuresis in head injury patients. Brain natriuretic peptide (BNP) activities may also responsible for hyponatremia⁷. BNP is a potent diuretic, natriuretic, vasodilating agent, and an inhibitor of the secretion of aldosterone, renin, and vasopressin^{11,12,13}. Increased BNP is most commonly found in patients with subarachnoid hemorrhage or hemorrhage at the base of the brain or in the third ventricle^{14,15}. Atrial natriuretic peptide (ANP) is the potential hormonal mediator of hyponatremia in intracranial disorders¹⁶. Damage to ANP and BNP containing cells in intra-cranial disorders and passage of these peptides across the blood brain barrier might cause 'inappropriate' release of natriuretic peptides¹⁷.

Brain injury is a stress, responding to which the sympathetic nervous system hormones are stimulated which in turn cause both arterial and venous contraction, leading to increased preload, inotropy, and systemic blood pressure. The kidneys could respond to these cardiovascular changes with a pressure-induced natriuresis¹⁸. ANP is produced in the atria of the heart and activated when the atrial stretch receptors become stimulated in response to hypervolemia, increased sodium, and/or an expanded preload¹⁹.

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. Patients with severe head injury are at risk for developing polyuresis through a variety of mechanisms working in polyuresis like the syndrome of inappropriate antidiuretic hormone secretion, cerebral salt loss. The incidence of sodium disorders was high in our study(55.5%) which is in accordance with the other studies^{8,9}.

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 insipidus¹⁰. The positive association of serum sodium and mentation is a known fact. Abnormal mental status including confusion, decreased consciousness, hallucinations and coma are well established facts in hyponatremia.

V. Conclusion

Severe traumatic brain injuries are often accompanied by electrolyte abnormalities especially hyponatremia and hypokalemia. A falling serum sodium level can lead to central nervous system changes, including confusion, seizures, and even coma. Early diagnosis and appropriate treatment of hyponatremia are essential for the recovery of the patients.

References

- [1]. Alves OL, Bullock R (2001). "Excitotoxic damage in traumatic brain injury". In Clark RSB, Kochanek P. Brain injury. Boston: Kluwer Academic Publishers. p. 1. ISBN 0-7923-7532-7. Retrieved 2008-11-28.
- [2]. Schwartz GR: Trauma to the head. Principles and Practice of Emergency Medicine Fourth edition. 1998:232-34
- [3]. Lath R (2005). Hyponatremia in neurological diseases in ICU. Indian J, Crit. Care Med. 9:47-51
- [4]. Isotani E, Suzuki R, Tomita K (1994). Alterations in plasma concentrations of natriuretic peptides and antidiuretic hormone after subarachnoid hemorrhage. Stroke. 25:2198-203
- [5]. Lohani S, Devkota UP (2011). Hyponatremia in patients with traumatic brain injury: etiology, incidence, and severity correlation. World Neurosurg. 76(3-4):355-60.
- [6]. Goldman, M.J. (1973). Principles of Clinical Electrocardiography 8th ed. Los Altos, California: LANGE medical Publications. p. 293

- [7]. Mukoyama M, Nakao K, Hosoda K (1991). Brain natriuretic peptide as a novel cardiac hormone in humans. *J. Clin. Invest.* 87:1402–1412
- [8]. Cole CD, Gottfried ON, Liu JK, Couldwell WT (2004). Hyponatremia in the neurosurgical patient: diagnosis and management. *Neurosurg Focus.* 15; 16(4):E9
- [9]. Donati-Genet PC, Dubuis JM, Girardin E (2001). Acute symptomatic hyponatremia and cerebral salt wasting after head injury: an important clinical entity. *J. Pediatr. Surg.* 36:1094–1097.
- [10]. Audibert G, Hoche J, Baumann A, Mertes PM (2012). Water and electrolytes disorders after brain injury: mechanism and treatment. *Ann FrAnesthReanim.* 31(6):109-15.
- [11]. Goldsmith MF (1987). Atrial peptide study proceeds apace. *JAMA.* 257:287.]
- [12]. Richards AM, Nicholls MG, Espiner EA (1985). Effects of alfa-human atrial natriuretic peptide in essential hypertension. *Hypertension.*7:812–817
- [13]. Taikkanen I, Fyhrequist F, Metsamine K (1986). Plasma atrial natriuretic peptide in normal man. *J. Clin. Invest.*77:734–742
- [14]. Wijdicks EFM, Ropper AH, Hunnicutt EJ (1991). Atrial natriuretic factor and salt wasting after aneurysmal subarachnoid hemorrhage. *Stroke.* 22:1519–1524.
- [15]. Diringner MN, Lim JS, Kirch JR (1991). Suprasellar and intraventricular blood predict elevated plasma atrial natriuretic factor in subarachnoid hemorrhage. *Stroke.* 22:577–581
- [16]. Weinand M, O'Boynick P, Goetz K (1989). A study of serum antidiuretic hormone and atrial natriuretic peptide levels in a series of patients with intracranial disease and hyponatremia. *Neurosurgery* 25:781–5
- [17]. Walter M, Berendes E, Claviez A, Suttorp M (1999). Inappropriate secretion of natriuretic peptides in a patient with a cerebral tumor. *J Am Med Assoc.* 282(1):27–8
- [18]. Singh S, Bohn D, Carlotti APCP, Cusimano M, Rutka JT, Halperin, M.L. (2002). Cerebral salt wasting: Truths, fallacies, theories, and challenges. *Critical Care Medicine,* 30(11): 2575-2579
- [19]. Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL (Eds.) (2001). *Harrison's Principles of Internal Medicine* (15th ed.). New York: McGraw Hill

*DrAnshul Jain. "Correlation Between the Severity of Head Injury And Electrolytes in Patients with Traumaticbrain Injury." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)* 16.9 (2017): 55-58