Original Research Article

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ELECTROLYTES IMBALANCE IN TRAUMATIC BRAIN INJURY PATIENTS

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Abstract:

Objectives: The role of electrolyte imbalance is being delineated in severe cranial trauma and is an essential investigation for its therapeutic managements. This study is designed to uncover the prevalence of electrolyte imbalance in traumatic brain injury (TBI) patients. **Material and Methods:** 50 consecutive patients with head injury and 50 trauma patients without clinical and radiological evidence of head injury were admitted to the emergency service of Geetanjali Medical College, Udaipur during 2 month period. We measured serum level of Magnesium, phosphorus, calcium, potassium and sodium and calculate APACHE score for prognosis at admission. We compared all electrolyte values in two groups taking head injury patient as case and trauma patient without head injury as control. **Results:** Different Electrolyte levels at admission in group 1 vs. group 2 were as follows (mean ±SD): Na levels were 138.85±5.68 vs. 140.62±5.89 in groups 1 and 2, respectively. K levels were not very significant between both groups group 1 vs group 2 (4.23±0.62 mmol/L vs. 4.384±0.54mmol/L; (*p,* .20). Phosphorus 2.971 ±0.91 vs. 3.48±0.91 (*p*, .01). Mg, 2.1086±0.44 vs. 2.96±0.68 (p, .01). Ca levels were 8.17±0.74 vs. 8.68±1.12mg/dl for groups 1 and group 2, respectively (*p=0.008*). **Conclusion:** We conclude that patients with brain injury are at a high risk for the development of electrolyte imbalance including hyponatremia, hypocalemia, hypophosphatemia as well as hypokalemia and (to a lesser degree) Hypomagnesemia.

Keywords: Traumatic Brain Injury, hyponatremia, hypocalcaemia, hypophosphatemia, Hypomagnesemia.

INTRODUCTION:

India is passing through the triple epidemic of communicable, non communicable and injuries, due to epidemiological and demographic transition. **(1)** Among injuries,

traumatic brain injury (TBI) is among the most significant one manifesting high morbidity and mortality. The consequences of TBI results in disability with lifelong financial, medical, emotional, family trauma.TBI is a foremost important cause of death and disability entire the world **(2)** and is the leading cause of brain damage in children and young adults.**(3)**

Patient with TBI have a high risk of developing different type of electrolyte imbalance, at the time of admission and duration of their ICU stay. It will affect treatment and outcome of patient.

Magnesium (Mg) is engaged in so many biomedical important enzymatic reactions as a cofactor and it is also well correlated with control of the sodium/potassium (Na/K) transport across membranes by activating the Na-K ATPase pump. **(4,5)** 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. Previous studies showed 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 **(1–7)**.

Phosphate (P) is a major intracellular anion and play important role in maintaining muscle tone **(7, 8)**. Hypophosphatemia has been shown to be associated with muscle weakness, including weakness of respiratory muscles. (**9, 10)**

Hyponatremia and correction of hyponatremia are clinically significant in neurology as a fast declining serum sodium concentration as well as rapid correction of chronic hyponatremia may lead to neurological symptoms .**(11, 12)**

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 contraction. **(13)**

Calcium is involved in nerve conduction, skeletal and cardiac muscle contractions therefore hypocalcemia may be involved in pathology of some clinical disorders like neuromuscular irritability, muscle spasms, seizures, delayed ventricular repolarization, and cardiac failure. **(14)**

Cerebral injury can lead to electrolyte imbalance which may prove critical for survival of patients. There are different mechanisms to explain electrolyte imbalance in TBI patients. Cerebral injury can cause polyuresis through the syndrome of inappropriate antidiuretic hormone secretion and cerebral salt loss.

Patients with cerebral trauma are commonly managed with mannitol, which can promote polyuresis. Thus, polyuresis is a possible source of loss of different electrolytes in severe head injury patients.

The role of electrolyte imbalance is being delineated in severe cranial trauma and may be essential investigations for its therapeutic managements. This study is designed to uncover the prevalence of electrolyte imbalance in traumatic brain injury (TBI) patients.

MATERIAL AND METHOD

50 consecutive patients with head injury and 50 trauma patients without head injury were admitted to the emergency service of Geetanjali Medical College, Udaipur during 2 month period. We measured serum level of Magnesium, phosphorus, calcium, potassium and sodium and calculate APACHE score for prognosis at admission. We compared all electrolyte values in two groups we took head injury patient as case (**GROUP 1)** and orthopaedic trauma patient without head injury as control (**GROUP 2)**.

RESULTS

Mean age in group 1 was 37.78 (range, 15–73) year. There were 2 females and 48 males in the study. Road traffic accident was mode of injury in 34 and fall from height in 16.According to type injury there were 15 patients had Subdural haemorrhage (SDH), 8 patients with Intracranial haemorrhage (ICH) , 27 patients with contusion. According to site of lesion of 16 patients had lesion frontal temporal region, 14 frontal, 2 temporal, 1crebellum, and 1cerbral injury and 8 patients with no any abnormality in brain.

The average Glass comma scale (GCS) in group 1 was 6.44 and the average apache score was 13.07 at admission to our hospital. Five patients in group 1 used medication that can be associated with loss of Mg and/or P (diuretics). No pre-existing risk factors for electrolyte loss were present in the other patients in group 1. The average age in group 2 was 33.30 yrs (range, 15–65).

The average GCS in group 2 was 13.0 and the average apache score is 4.82 at admission to our hospital. None of the patients in group 2 used medication associated with electrolyte disorders. There were 7 females and 43 males in the study. Road traffic accident was mode of injury in 37, slip in bathroom in 5 and fighting in 8 patents.

Different Electrolyte levels at admission in group 1 vs. group 2 were as follows (mean ±SD): Sodium (Na), Potassium (K), Calcium (Cl), Calcium (Ca) and Phosphorus (P) level.

Na levels were 138.85±5.68 vs. 140.62±5.89 in groups 1 and 2, respectively. Seventeen of 50 patients in Group 1 had Na levels of 135mmol/L or lower vs. 10/50 in group 2 (*p= 0.177*) and hypernatremia (Na level more than 145 mmol/L) 7/50 in group 1 vs. 11/50 in group 2 (*p=0.435*).

K levels were not very significant between both groups group 1 vs group 2 (4.23±0.62 mmol/L vs. 4.384±0.54 mmol/L; *p,* 0.20). Moderate hypokalemia (K levels below 3.6 mmol/L) was present in 10/50 patients in group 1 vs. 2/50 patients in group 2 (*p,* 0.031). Severe hypokalemia (K levels equal

or lower than 3.0) was present in 1/50 patients in group 1 vs. 0/50 patients in group 2 (*p,* 1.00). Hyperkalemia K level (greater than 5.1 mmol/L) 6 patients in group 1 vs. 9/50 patients in group 2 (*p 0*, 575) .

Phosphorus level was 2.971 ±0.91 vs. 3.48±0.91 (*p*, .01). In group 1, 28/50 patients had P levels, less than 2.7 mg/dl vs. 7/50 patients in group 2(p=0.0001) and p level greater than 4.5 (hyperphosphatemia) in group 1, 3/50 patients (*p,* .01) vs. 10/50 patients in group 2(p=0.074).

Mg level, 2.1086±0.44 vs. 2.96±0.68 (p, .01). None of the patients had low Mg level in both groups, in group 1, 3/50 patients had Mg levels, more than 2.6 mg/dl (hypermagnesemia) vs. 32/50 patients in group 2 (*p=0.0001*).

Ca levels were 8.17±0.74 vs. 8.68±1.12mg/dl for groups 1 and group 2, respectively (*p=0.008*). **Hypocalcaemiaca** level (less than 8.5 mg/dl) was present 32 out of 50 patients in group 1 vs. 17 out of 50 in group 2 (p=0.005)and hypercalcaemia (Ca level more than 10.5mg/dl) 0/50 and 4/50 in group 1 vs. group 2 respectively(p=0.126).

Saline infusion (NaCl, 0.9%) was given 15 patients and of Na 0.45%/glucose 2.5% in five patients in group 1. Average volume infused was 899 ml in group 1 before ICU admission. Three patients had also received blood transfusions of the patients in group 1.Fluid resuscitation in group 2 consisted of infusion of saline (NaCl, 0.9%) in 18 patients and of Na 0.45%/glucose 2.5%in seven patients. Average volume infused before ICU admission was 976 ml. Five patients had also received blood transfusions of the patients in group 2, The difference in volume infused between groups 1 and 2 was not significant. No hypertonic saline was used in our head injury patients.

Urine production in both groups before admission was measured using a Foley catheter. The average residual urine volume was 902 ml in group 1 vs. 767 ml in group2 (*p,* = 0.0152) upon insertion of the catheter.

APACHE II scores were significantly higher in group 1 than in group 2 (9.28±5.07 vs. 5.12±2.42), reflecting differences in GCS as well as other factors, such as tachycardia and tachycardic arrhythmias, episodes of low or high blood pressure, and electrolyte disorders (high Na levels and low K) present and blood counts in group 1. There were no differences in the presence of chronic diseases between groups 1 and 2.

DISCUSSION

Our results clearly demonstrate that patients with severe head injury are at a high risk for the development of hyponatremia, hypophosphatemia, hypokalemia, hypocalcemia and hypormagnesemia, when cerebral injury is present in compared to other group while in other orthopaedic injury patients (group 2) developed hyponatremia, hperphosphatemia, hypermagnesemia, hyperkalemia and some extend to hypocalcemia.

Hyponatremia may develop as a result of syndrome of inappropriate secretion of antidiuretic hormone characterized by dilutional hyponatremia or cerebral salt-

wasting syndrome featured by natriuresis in head injury patients. Brain natriuretic peptide (BNP) activities may also responsible for hyponatremia. (**15)** Brain natriuretic peptide is an effective diuretic, natriuretic, vasodilating agent, and an inhibitor of the secretion of aldosterone, renin, and vasopressin. Patients with subarachnoid hemorrhage or hemorrhage at the base of the brain or in the third ventricle are most commonly show enhanced BNP level. **(16-17)**

Diabetes insipidus, have hypothalamic-pituitary dysfunction, particularly growth hormone deficiency, ACTH, TSH and gonadotrophin deficiency and diabetes insipidus that commonly could be caused of hypernatremia. **(18)**

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 has worked in polyuresis like the syndrome of inappropriate antidiuretic hormone secretion, cerebral salt loss.

Hypomagnesemia was associated with hypokalemia in most patients. As outlined in our introduction, hypomagnesemia and, to a lesser degree, hypophosphatemia are associated with various forms of cardiac arrhythmia. **(19)** Causes of hypomagnesemia include protein-calorie malnutrition, intravenous administration of Mg-free fluids and total parenteral nutrition, as well as diarrhoea and steatorrhea, short bowel syndrome, and continuous nasogastric suctioning. Many of these factors may be present simultaneously in brain injury patients. Trauma patients are frequently treated with antibiotics, often including aminoglycosides. Thus, as with hypomagnesaemia, a combination of many factors may put brain injury patients at risk for hypophosphatemia. Polyuresis induced by cerebral injury increases this risk even further, as demonstrated by the results of our study. The process through which patients with severe head injury could be put endangered for the development of electrolyte disturbance is uncertain.**(20-21)**

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. Residual urine volume was higher in group 1 than in group 2; however, the time period in which urine volumes were formed in group 1 is unknown, because we were unable to determine the last time that the patients had urinated before the occurrence of head injury.

In addition, spontaneous urine loss could have occurred in group 1 patients at the scene of their accident; this would lead to an underestimation of residual urine levels. Although this does not establish that polyuresis was the cause of electrolyte deficiencies in group 1, it seems likely that high urine production and renal excretion of electrolytes contributed to the occurrence of electrolyte disorders. It is difficult to

determine to what extent outcome in our patients was affected by the presence of electrolyte disorders. **(22-23)**

Na and K are measured routinely at admission in all patients, including those with cerebral injury. However, Ca, Mg and P 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 intensivist and others physician who are treating patients with severe head injuries should be aware of this potential problem and that levels of Ca, Mg and P should be measured on a routine basis in all patients with severe head injury.

CONCLUSION

We conclude that patients with brain injury are at a high risk for the development of hyponatremia, hypocalcemia, hypocalcemia and hypophosphatemia as well as hypokalemia and (to a lesser degree) Hypomagnesemia.

Increased urinary loss appears to be one of the factors contributing to electrolyte depletion; other, as yet unknown factors, induced by neurologic trauma may also play a role. Levels of Mg and P, as well as K, Na and Ca, should be determined frequently in these patients, and if necessary, adequate supplementation should be initiated promptly.

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Table 1: Various group parameters & their association

PARAMETER	Group 1 (N=50)	Group 2 (N=50)	t- value	p-value
Glass Comma Score	9.46±3.77	12.92±1.70	5.916	< 0.0001
APACHE SCORE II	9.28±5.07	5.12±2.42	5.236	< 0.0001
Age	37.78±15.11	33±13.72		
MAP	98.39±16.9	92.13±9.8	3.6932	0.0004
Heart Rate	84.26±22.60	79.94±15.95	1.1043	0.2722
Respiratory Rate	20.44±3.79	19.44±2.71	1.5177	0.1323
Oxygenation	97.31±2.17	97.28±1.34	0.0832	0.9339
Arterial PH	7.42±0.09	7.45±0.09	1.6667	0.0988
Serum Na	138.85±5.68	140.62±5.89	1.5296	0.1293
Serum K	4.23±0.62	4.384±0.54	1.29	0.2001
Serum Cl	105.88±6.87	106.96±7.41	0.7558	0.4516
Serum Ca	8.17±0.74	8.68±1.12	2.6864	0.0085
Serum P	2.971 ±0.91	3.48±0.91	2.7967	0.0062
Serum Mg	2.1086±0.44	2.96±0.68	7.4330	< 0.0001
Random Blood Sugar	134.58±27.40	131.3±26.01	0.6139	0.5407
Serum Creatinin	0.73±0.17	0.69±0.16	1.2116	0.2286
Hemoglobin	12.76±2.03	12.352±2.06	0.9975	0.3210
Pack Cell Volume (PCV)	36.28±5.63	35.2±5.54	0.9668	0.3360
Total Leucocytes Count (TLC)	13433±4419	12036±3769	1.7008	0.0922
Platelet Count	2.27±0.78	2.26±0.61	0.0714	0.9432
Urine Volume	902±277.85	767.2±267.57	2.4711	0.0152
Fluid Volume	899±280.11	976±272.62	1.3930	0.1668