PROSPECTIVE STUDY OF SERUM ELETROLYTE (NA+, K+, CA++, PO4−) IMBALANCE IN SEVERE AND MODERATE TRAUMATIC HEAD INJURY

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ABSTRACT: SUMMARY: Electrolyte abnormalities are common in patients with traumatic brain injury. Disturbances of serum sodium levels are among the most common and frequently occur in neurologically morbid patients and exacerbate their severity. Hypernatremia usually results from diabetes insipidus syndrome, whereas hyponatremia develops due to syndrome of inappropriate secretion of antidiuretic hormone (SIADH) or cerebral salt-wasting syndrome (CSWS) and contribute to the high morbidity and mortality rates observed in these patients. The aim of this study is to measure the serum levels of Na+, K+, Ca++, PO4− in head injury patients and find the range & compare the levels in severe and moderate head injury patients with mild head injury patients. METHOD: Na+, K+, Ca++, PO4− levels of patients in age group 13-60 years were estimated by ion selective electrode method. RESULTS: it was found that most common electrolyte imbalance found is hyperphosphotemia (87%) followed by hyponatremia (62.90%). In case of hyperelectrotyremia, hyperkalemia (46.6%) to be the most common electrolyte imbalance in mild type of head injury whereas in severe and moderate cases hyperphosphotemia (28.40%) is most common dyselectrolytemia. CONCLUSION: hyponatremia is the most common electrolyte disbalance among Na+, K+, Ca++, PO4− in patients of traumatic head injury which is also very dangerous and need to be correct promptly.

KEYWORDS: Hyponatremia, Hypernatremia. Hypokalemia, hyperkalemia, Hypocalcemia, Hypercalcemia, Hypophosphotemia, Hyperphosphotemia electrolyte imbalance, head injury, glasgow coma score.

INTRODUCTION: Electrolyte abnormalities are common in patients with head injuries, occurring at least once during the hospital course of 59% of the traumatic coma data bank (TCDB) patients,[1] and disturbances in serum sodium level, both hyponatremia and hypernatremia, are common. Serum sodium disturbances frequently occur in neurosurgical and neurologic patients and exacerbate their neurologic and general conditions. The disturbance may manifest as hypernatremia or hyponatremia. Hypernatremia invariably results from the diabetes insipidus syndrome,[2] whereas hyponatremia is caused by syndrome of inappropriate secretion of antidiuretic hormone (SIADH) or cerebral salt-wasting syndrome (CSWS).[3][4][5] and contribute to the high morbidity and mortality rates observed in these patients.[6] K is the major intracellular cation, with relatively low extracellular levels. Changes in K levels can affect muscle contraction and nerve conduction; hypokalemia is associated with cardiac arrhythmias (Especially in patients with ischemic heart disease and left ventricular hypertrophy), muscle weakness (Including weakness of respiratory muscles), rhabdomyolysis, renal failure, and hyperglycemia. Calcium is an important membrane stabilizing ion. Its ionized form is the function
component and deviations from normal prove catastrophic. Hypocalcemia is associated with neuromuscular irritability and muscle spasms, seizures, delayed ventricular repolarization, and cardiac failure.

Phosphate (P) is a major intracellular anion and plays an important role in many biochemical pathways relating to normal physiologic functions, especially in maintaining muscle tone.[7] Hypophosphatemia has been shown to be associated with muscle weakness, including weakness of respiratory muscles,[8][9] and with respiratory infection.[10] Aubier et al.[11] found that hypophosphatemia impairs the contractile properties of the diaphragm in critically ill patients, leading to difficulties in weaning from the ventilator. Low P may also be associated with decreased cardiac output and with ventricular tachycardia after myocardial infarction. Patients with hypophosphatemia may be asymptomatic or may experience weakness, malaise, anorexia, bone pain, and respiratory arrest.[7]

The calcium ion (Ca2+), and maintenance of cellular Ca2+ homeostasis, is essential for the proper function of the nervous system. For example, Ca2+ is critical for growth and development (Ghosh and Greenberg, 1995; Spitzer and Ribera, 1998; Takei et al., 1998), is a necessary component for neurotransmission (Berridge, 1998; Iwasaki et al., 2000), and contributes to distinct patterns of differential gene expression in neurons (Bading et al., 1993; Lerea and McNamara, 1993; Finkbeiner and Greenberg, 1998). Calcium also appears to be required for most forms of activity-dependent synaptic plasticity, generally believed to be the cellular correlate for learning and memory (Bliss and Collingridge, 1993; Malenka and Nicoll, 1999; Lamont and Weber, 2012; Kawamoto et al., 2012).

**METHODOLOGY:** This prospective study was carried out in Department of Surgery, Gandhi Medical College and associated Hamidia Hospital, Bhopal. Institutional Ethical committee approval was sought and written informed consent was obtained from the patient or attendees. TBI Patients of age 13-60 Years who were admitted in Hamidia Hospital from October 2012 - November 2013 were included in the Study. Patients who had dyelectrolytemia on admission, as well as those who developed electrolyte disorders during hospitalization, were included in the study. They did not receive any diuretics and were free from any renal or endocrinal diseases. Patients who presented with other potential causes of electrolyte disorders, systemic trauma, massive transfusion, spinal cord injury, brain insult due to other causes like hypertension, infection, etc. were excluded from the study. The characteristics analysed were as follows: Incidence of electrolyte disorders, Presence of injuries in the first CT scan of the skull after TBI (All scans were evaluated by two team members trained in CT evaluation), and Glasgow coma scale (GCS).[7] Serum Electrolytes sodium, potassium, calcium, and phosphate were estimated by an Electrolyte Analyser (Enlite Instrument), that works on principle of ion selective electrodes (durst 1999). Electrolyte were estimated at the time of admission in emergency room while resuscitation before giving any IV fluid and then electrolytes were estimated every day for five consecutive days, thereafter alternate days for minimum period of fifteen days.

Normal levels of electrolytes are:

- Sodium. 135-145 mEq/L (serum).
- Potassium. 3.5-5.5 mEq/L (serum).
- Calcium. 8.8-10.4 mg/dL (total Ca; serum).
- Phosphate. 2.5-4.5 mg/dL (plasma; adults).
The outcome will be estimated on the basis of Glasgow coma outcome score shown in table below:

| Score | Description                                                                 |
|-------|-----------------------------------------------------------------------------|
| 5     | Good outcome: Resumption of normal life; there may be minor neurological/psychological deficits |
| 4     | Moderately disabled: Able to work in sheltered environment and travel by public transportation |
| 3     | Severely disabled: Dependent for daily support due to mental or physical Disabilities |
| 2     | Vegetative state: Persistently unresponsive and speechless                   |
| 1     | Death                                                                       |

Table 1: GLASGOW OUTCOME SCORE

Consciousness level of patient will be determined on the basis of Glasgow coma scale

| Features          | Score responses            | Score notation |
|-------------------|----------------------------|----------------|
| Eye opening       | Spontaneous                | 4              |
|                   | To speech                  | 3              |
|                   | To pain                    | 2              |
|                   | None                       | 1              |
|                   | Oriented                   | 5              |
|                   | Confused conversation      | 4              |
|                   | Inappropriate words        | 3              |
|                   | Incomprehensible sound     | 2              |
|                   | None                       | 1              |
| Verbal response   | Obeys command              | 6              |
|                   | Localises pain             | 5              |
|                   | Flexion- Normal            | 4              |
|                   | -Abnormal                  | 3              |
|                   | Extends                    | 2              |
|                   | None                       | 1              |
| Motor response    |                            |                |
|                   |                            |                |
| Total             |                            | 15             |

Table 2: Glasgow coma scale

RESULTS AND DISCUSSION: The incidence of head injury is higher in male patients.

Majority patients are in the age group of 20-50 yrs (80.1%) who sustain head injury among the patients included in our study of 13-60yrs and 7.69% cases are >50yrs.

Mode of injury in maximum number of cases is Road Traffic Accident (72.85%) followed by Assault (19.21%) then fall from height (7.29%).

In all head injury case the incidence of SAH(38%) was the most common CT finding of our study other CT finding is EDH(20.53%)>SDH (19.21%)>DAI(12.58%).
Dyselectrolytemia was more amongst those who received mannitol (84.37%). There is progressive increase in severity of dyselectrolytemia, and mannitol may cause more dyselectrolytemia because it causes diuresis. Hence mannitol acts as additive factor for dyselectrolytemia.

Hyponatremia (64.2%) and hypophosphotemia (59.25%) is more in patients to whom mannitol is given as compared to patients to whom mannitol is not given 45.26% and 17.14% respectively.

| ELECTROLYTE STATUS | GCS[138] | TOTAL |
|--------------------|----------|-------|
|                    | MILD(58) | MODERATE+SEVERE(80) | |
| Hypo Na⁺           | 23-0=23  | 38-1=37          | 60   |
|                    | (39.65%) | (46.25%)         |      |
| Hypo K⁺            | 6-0=6    | 21-4=17          | 23   |
|                    | (10.34%) | (22.5%)          |      |
| Hypo Ca₂⁺          | 24-0=24  | 39-8=31          | 55   |
|                    | (41.38%) | (38.75%)         |      |
| Hypo PhO₄⁻          | 0        | 4-3=1            | 1    |
|                    | (0.00%)  | (1.25%)          |      |

TABLE 1: DISTRIBUTION OF DYSELECTROLEMIA IN MILD AND SEVERE HEAD INJURY

- Levels of serum electrolytes was decreased more in moderate and severe head injury except for serum levels of calcium where serum levels of calcium was decreased more in mild head injury.

In our study the incidence of hypernatremia (7.28%) is more in patients to whom mannitol is given as compared to patients to whom mannitol is not given (3.57%). Whereas it is nearly equal in hypercalcemia (1.23) & (1.42) and hyper phosphotemia (87%) & (87%) and nearly equal in hyperkalemia (17.28%) & (14.29%) respectively.

| ELECTROLYTE STATUS | GCS[138] | TOTAL |
|--------------------|----------|-------|
|                    | MILD(58) | MODERATE+ SEVERE(80) | |
| Hyper Na⁺          | 1 [2%]   | 10[11.36%]        | 11 |
| Hyper- K⁺          | 7 [14%]  | 8[9.09%]          | 15 |
| Hyper –Ca₂⁺        | 0 [0.00%]| 1 [1.1%]          | 1  |
| Hyper –PO₄⁻        | 33 [66%]| 19 [21.5%]        | 52 |

TABLE 2: HYPER ELECTROLEMIA IN MILD AND MODERATE+SEVERE HEAD INJURY

- Serum levels of sodium and calcium were increases in moderate and severe head injury whereas serum levels of potassium and phosphate were increases in mild head injury.

In Our study in cases of hyperelectrolytemia, hyper kalemia (46.6%) to be most common electrolyte imbalance in mild type of head injury whereas in severe cases hyper phosphotemia is most common (28.40%) hyperelectrolytemia.
The incidence of death is more common in patients of electrolyte imbalance as compared to patients of normoelectrolemia. Also, incidence of hypernatremia and hyperphosphotemia is more in patients of death due to head injury.

Hypernatremia (76.9%) was the commonest finding in early death within 2 days.

**CONCLUSION:** In our study the incidence of head injury is more common in male patients (6.5:1). Maximum number of cases are in age group of 20-50 yrs. (80%) in which the most common mode of injury is road traffic accident (72.85%) followed by assault (19.21%) then fall from height (7.29%). In all cases of head injury SAH comes to the most common CT finding followed by EDH (20.53%), SDH (19.21%), and DAI (12.58%).

In our study the incidence of dyselectrolytemia occurs more in severe and moderate head injury cases as compared to mild head injury. The incidence in dyselectrolytemia is more among the patients who received mannitol (84.37%).

In all cases of head injury most common electrolyte imbalance found is hyperphosphotemia (87%) followed by hyponatremia (62.90%). In cases of hyperelectrolytemia, hyperkalemia (46.6%) was found to be the most common electrolyte imbalance in mild type of head injury whereas in severe and moderate cases hyperphosphotemia (28.40%) is most common dyselectrolytemia. In cases of early death within 2 days most common electrolyte imbalance is hypernatremia (76.9%). In cases of head injury patients having severe hypocalcemia (66.66% mortality) and severe hyponatremia (44.44% mortality) had poor prognosis but the outcome is worst in patients having hyponatremia (62% good outcome), hyperkalemia (80% good outcome), and hypophosphotemia (81%).

Patients who died of head injury had both severe hyponatremia and severe hypernatremia i.e. 42% and 66.66% respectively. Hypernatremia was the commonest cause in early death i.e. within 2 days. Outcome of mild hyponatremia and mild hypernatremia is not so bad whereas outcome of severe hyponatremia and severe hypernatremia is poor. Patients with severe hypernatremia have poor out come as compared to patients of mild or moderate hypernatremia.

**REFERENCES:**
1. Krug E (ed). Injury: A leading cause of the global burden of disease. Geneva: World Health Organization; 1999.
2. Maggiore U, Picetti E, Antonucci E, et al. The relation between the incidence of hypernatremia and mortality in patients with severe traumatic brain injury. Crit Care. 2009; 13:R110. [PMC free article][PubMed]
3. Wellingson Silva Paiva, Douglas Alexandre França Bezerra, Robson Luis Oliveira Amorim, Eberval Gadelha Figueiredo, Wagner Malago Tavares, Almir Ferreira De Andrade, and Manoel Jacobsen Teixeira. August 2011 volume 2011:7 page 345-349.
4. Maggiore U, Picetti E, Antonucci E, et al. The relation between the incidence of hypernatremia and mortality in patients with severe traumatic brain injury. Crit Care. 2009; 13:R110. [PMC free article][PubMed].
5. Lath R: Hyponatremia in neurological diseases in ICU. Indian J, Crit. Care Med. (2005).9:47-51.
6. Cintra EA, Araujo S, Quagliato EM, de Castro M, Falcao 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): 1158-1165.
7. Lloyd CW, Johnson CE: Management of hypophosphatemia. Clin Pharm 1988; 7:123-128.
8. Polderman KH, Peerdeman SM, Girbes ARJ (2001) Hypophosphatemia and hypomagnesemia induced by cooling in patients with severe head injury. J Neurosurg 94:697–705.
9. Gravelyn TR, Brophy N, Siegert C, et al: Hypophosphatemia-associated respiratory muscle weakness in a general inpatient population. Am J Med 1988; 84:870-876.
10. Fisher J, Magid N, Kallman C, et al: Respiratory illness and hypophosphatemia. Chest 1983; 83:504-508.
11. Aubier M, Murciano D, Lecocguic Y, et al: Effect of hypophosphatemia on diaphragmatic contractility in patients with acute respiratory failure. N Engl J Med 1985; 313:420-424.

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