ORIGINAL ARTICLE

SERUM ELECTROLYTE DERANGEMENTS IN PATIENTS WITH TRAUMATIC BRAIN INJURY

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Background: Electrolyte derangements are common sequel of traumatic brain injury. Use of intravenous fluids, diuretics, syndrome of inappropriate ADH secretion and cerebral salt washing are some of the factors responsible for this. Proper in time detection followed by appropriate treatment not only improves neurological status but also decrease morbidity and mortality. This study was conducted to know serum derangements of different electrolytes in patients with traumatic brain injury. **Methodology:** This cross-sectional study was conducted in Pakistan Institute of Medical Sciences, Islamabad, Pakistan from Feb 2009 to Feb 2010. All adult patients with traumatic brain injury who presented to Neurosurgical department with severe head injury (GCS <8) and who need monitoring in high dependency unit, were included in this study. Initially twice daily serum electrolyte monitoring for one week then once daily for remaining period of hospital stay was carried out. All samples were sent to Pathology department of Pakistan Institute of Medical Sciences, Islamabad. Patients who need corrective measures for imbalance had repetition of sampling after giving appropriate therapy. Statistical analysis was performed on SPSS-16. **Results**: Total 215 patients presented with severe head injury that were managed in high dependency unit. Out of which 127 (59.1%) were male and 88 (40.9%) were females. Most of them were adults between 21–40 years of age (21.4%; 24.7%). Sodium was the main electrolyte that underwent change & out of which hyper-natremia was major abnormality that occurred in 140 (65.1%) of patients. This is followed by hypo-kalemia that occurred in 79 (36.7%) of patients. Serum calcium & magnesium levels show little derangements. **Conclusion**: Electrolyte imbalance following traumatic head injury is an important cause to look for in patient monitoring. Sodium is the chief electrolytes of concern. Serum potassium and calcium levels also under goes notable changes.

Keywords: Traumatic brain injury, serum electrolyte derangements

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INTRODUCTION

Motor vehicle accidents are common squeal of today's modern era. They results in severe intracranial and extra-cranial injuries.[1] Both hydro-electrolytic and haemodynamic factors contribute in patients with head injury and their effect is related to blood-brain barrier integrity and characteristics of cerebral perfusion pressure (CPP). Any disturbance in these results in development of interstitial oedema and worsening of cerebral ischemia. Early interstitial oedema is mainly a consequence of low plasma osmolality, whereas low oncotic pressure plays a minor role.[2] Various mediators are released which enhance vasogenic and/or cytotoxic brain oedema. These include glutamate, lactate, H^+, K^+, Ca^{++}, nitric oxide, arachidonic acid and its metabolites, free oxygen radicals, histamine, and kinins.[3]

Head injury also leads to various electrolyte derangements each of which have different neurologic manifestations.[4] Sodium is the most common and important electrolyte responsible among these. Both hyponatremia and hyernatremia can result. More so changes in potassium chiefly hypokalemia[5] and fluid content [6] are also encountered in clinical practice[7]. Many different causes are responsible for these. The most common being syndrome of inappropriate anti-diuretic hormone secretion,[8] Cerebral salt wasting (CSW),[7] reception of furosemide and mannitol[4]. Age is another important factor that also greatly affects morbidity and mortality. Advancing age has poor outcome.[9] Appropriate fluid management of patients with traumatic brain injury (TBI) presents a challenge in most part of the world.[10] Isotonic fluid can be given without significant fluid disturbances in body.[11] However patients may deteriorate after initial improvement even after 10 days due to electrolyte disturbances chiefly sodium.[12] It can result in depress level of consciousness.

Patient management of electrolyte balance following head injury is the most important strategy to avoid these.[10] The use of hyper osmolar fluids, such as hypertonic saline, has gained significant interest because they are devoid of dehydrating properties and may have other beneficial properties for patients with traumatic brain injury (TBI).[1] Other pharmacological agents such as phenibut (50 mg/Kg) and sodium hydroxybutyrate (0.2 g/Kg)[13] may also be used. They prevent electrolyte changes in blood also. They not only restore cardiovascular function

but also decrease intracranial pressure.[14] However hyperosmolar fluid also results in electrolyte disturbances especially of sodium which in result can alter fluid balance.[4] More so patients management of electrolyte balance is also important in prevention of traumatic cerebral vasospasm.[10] Many different options to lower increased ICP such as hypothermia, barbiturates, and osmotherapy also have some unwanted effects that depends upon underlying pathological lesion with severe head injury.[15] Rehabilitation among these patients is very important and it may take some time.[16]

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

MATERIAL AND METHODS

All adult patients with traumatic brain injury who presented to Neurosurgical Department with severe head injury (GCS <8) and who needed monitoring in high dependency unit, were included in this study. All those patients who needed ventilator support and those less than 14 years of age were excluded.

Initially twice daily electrolyte monitoring for one week then once daily for remaining period of hospital stay was carried out. Patients who needed corrective measures for imbalance had repetition of sampling after appropriate therapy. Statistical analysis was performed on SPSS-16.

RESULTS

Total 215 patients were presented from February 2009 to February 2010 with severe head injury. Out of which 127 (59.1%) were male and 88 (40.9%) were female. Sodium was the major electrolyte that underwent significant change followed by Potassium. Out of 215 patients 31 patients develop Hyonatremia (14.4%); 140 (65.1%) develop Hypernatremia; 44 (20.5%) patients had no change in serum sodium levels (Table-1).

Sodium is followed by Potassium. Serum Potassium level changed as; Hypokalemia in 79 (n = 215) (36.7%); Hyperkalemia in 48 (22.3%) and 88 (40.9%) had static potassium levels (Table-2). Other electrolytes which we look for were serum Calcium and Magnesium. Calcium levels were changed as; Hypocalcaemia in 71 (33%), Hypercalcaemmia in 10 (4.7%) and 134 (62.3%) had static serum calcium levels (Table-3). Changes in Magnesium levels remained stable in majority. There was hypo-magnesemia in 12 (5.6%), and hyper-magnesemia in 6 (2.8%) patients (Table 4).

Table-1: Changes in serum sodium levels (n=215)

Parameter	Frequency	Percentage
Hyponatremia	31	14.4
Hypernatremia	140	65.1
No change	44	20.5

Table-2: Changes in serum potassium levels (n=215)

Parameter	Frequency	Percent
Hypokalemia	79	36.7
Hyperkalemia	48	22.3
No change	88	40.9

Table-3: Changes in serum calcium levels

Parameter	Frequency	Percent
Hypocalcemia	71	33.0
Hypercalcemia	10	4.7
No change	134	62.3

Table-4: Changes in serum magnesium levels

Parameter	Frequency	Percent
Hypomagnasemia	12	5.6
Hypermagnesemia	6	2.8
No change	197	91.6

DISCUSSION

Electrolyte derangements are common in patients with head injury.[4] Most common and notable changes occur in serum Sodium and Potassium levels.[6,17] Changes in fluid level secondary to resuscitative measures and pharmacological therapy (use of Furosemide and Mannitol)[2] are mainly responsible for these. However other causes can also contribute. Out of which cerebral salt wasting, syndromes of inappropriate ADH secretion (SIADH) secretion are important.[18] Maintaining of fluid balance is main stay of treatment. It also greatly affects neurological outcome. Many different type of fluids are used namely hypertonic/hyperoncotic saline/dextran solution (HHS). Volume replacement with isotonic fluids not only is therapeutically of limited efficacy but may aggravate posttraumatic brain edema.[14] 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*[11] and the aim was normovolumia. Cintra Ede A *et al*[17] 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.[14,11] The use of these solutions was in specific conditions in our study like in SIADH and Diabetes Inspidis.

Controlling intra cranial pressure (ICP) is pivotal in traumatic brain injury patients. Unterberg A *et al*[12] used different pharmacological agents like mannitol and furosemide are used for this purpose.[2] We also used the same to control ICP. Dexamethasone is also a valueable adjunct.[18] Gaab M[6] routinely used Dexamethasone for this purpose and they found it more useful than mannitol and furosemide.[6] These results are in accordance with our study. However we did not use Furosemide routinely as we did Dexamethasone. Furosemide and Mannitol were used in specific situations in our study.

Electrolyte derangements in our study did not correlated with Goh KP *et al*.[18] They had Hyponatremia as major electrolyte abnormality. Whereas we had (14.4%) of patients with Hyponatremia as compared to 140 (65.1%) patients who develop Hypernatremia; 44 (20.5%) patients had no change in serum sodium levels.

So hypernatremia was more common in our study. The logical reason for this contrast may be the relative dehydrating policy as we used up to two litres of fluids per 24 hours routinely as in accordance with Steinbok P *et al*[19] along with the insensible losses. More so most of the patients were with poly trauma, which need tracheostomy and some develop fever also. So perhaps insensible losses were the cause of it. Monitoring of electrolytes was twice daily in first week of trauma as in accordance with Steinbok P *et al*.[19]

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*.[5] In our study low serum potassium was in 36.7% of patients as compared to 22.3% 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 beta 2-adrenergic stimulation of the Na^+-K^+ pump.[5]

Serum calcium changes render a variety of clinical manifestations in patients with traumatic brain injury.[20] Right from development of tetany[21] to seizures all can happen. Abnormal responses of neurons to stimulation secondary to accumulation of intracellular calcium in traumatic brain injury are responsible for these.[22] Both serum hypocalcaemia and hypercalcaemia can occur.[20] Hyperphenylalaninemia,[23] decrease in serum magnesium concentration and hyperventilation to control raised intracranial pressure[22] are factors responsible for these. We had more patients with hypocalcaemia 33% than hypercalcaemia 4.7%. Above mentioned factors were responsible for these. However we did not check phenylalanine levels as Plöchl E *et al*[23] did. Most of patients had stable serum magnesium levels. Only 12 (5.6%) had hypo and 6 (2.8%) had hypermagnesemia.

CONCLUSION

Electrolyte derangements are very common in patients with head trauma. It is an important and manageable cause of neurological deterioration. Most common derangement is during the first week of injury. Sodium is the most common electrolyte affected. Serum potassium and calcium levels must also be looked for, especially in first week of trauma.

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