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Short Communication

Electrolyte derangements in traumatic brain injury

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Introduction: Traumatic brain injury (TBI) occurs whenever mechanical force injures the brain. Electrolyte derangements are common after neurologic injury. Many of these patients may receive osmotic diuretics for the treatment of increased intracranial pressure or develop sodium disturbances, which act to alter fluid balance. Aim of our study is to find the prevalence of electrolyte disturbances in traumatic brain injury (TBI) patients. Methods: Sodium and potassium levels of twenty five TBI patients in the age group of 18-45 years were estimated by ion selective electrodes. Results: It was found that 4% of our patients had hypernatremia, 64% of the study subjects suffered from hyponatremia, 4% had hyperkalemia and 4% of the patients had hypokalaemia. Sodium levels bear a positive correlation with Glasgow coma scale (r = 0.634). Conclusion: Seventy five percent of the patients suffered from electrolyte disturbances of which hyponatremia was the commonest and the most dangerous which needs to be diagnosed and corrected at the earliest.

Keywords: Trauma, brain injury, electrolytes

INTRODUCTION

Electrolyte derangements are common after neurologic injury, with many having neurologic manifestations. It could be partially iatrogenic by the administration of mannitol, corticosteroids or diuretics or merely due to the intracranial disorder itself. Hyponatremia is a common electrolyte disturbance following intracranial disorders (Lath 2005; Isotani et al., 1994; Sane et al., 1994; Narotam et al., 1994). Hyponatremia is of clinical significance as a rapidly decreasing serum sodium concentration as well as rapid correction of chronic hyponatremia may lead to neurological symptoms (Lohani and Devkota 2011; Yee et al., 2010; Sterns 1987).

Potassium is the major intracellular cation, with relatively low extracellular levels. 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 intervention. We hypothesize that patients with cranial trauma might be at risk for electrolyte loss resulting in electrolyte disturbances.

Aim of our study is to find the prevalence of electrolyte disturbances in traumatic brain injury (TBI) patients.

METHODOLOGY

The study was carried out in Pondicherry institute of medical sciences in collaboration with the department of neurosurgery. Institutional ethical committee approval was sought and written informed consent was obtained from the patients/by-standers. TBI patients who were admitted in PIMS were included in the study. It included 25 male patients in the age group of 18-45 yrs. They did not receive diuretics and were free from renal or other endocrine diseases. Patients who presented with other potential causes of sodium disorders and systemic trauma, brain insult due to other causes like hypertension, infections were excluded from the study.

The severity of trauma was assessed by cerebral computed tomography and level of consciousness was assessed with Glasgow coma scale (GCS) (shown in Table 1).

Serum electrolytes were estimated by an electrolyte

Table 1. Severity of TBI based on GCS

GCS	Degree of severity	Number of patients(n=25)	% of subjects
13-15	Mild	10	40
9-12	Moderate	10	40
3-8	Severe	1	4

Table 2. Electrolyte derangements in traumatic brain injury

Electrolyte level	Diagnosis	Number of subjects (out of 25)	Percentage of subjects (%)
Sodium > 145 MEq/L	Diffuse TBI	1	4
Sodium < 130 MEq/L	Subdural hematoma	4	16
	Intracerebral hematoma	5	20
	Diffuse axonal injury	1	4
	Diffuse TBI	6	24
Potassium > 5 MEq/L	Subarachnoid haemorrhage	1	4
Potassium < 3.5 MEq/L	Intracerebral haemorrhage	1	4

analyzer (Ilyte instrument) which works on the principle of ion selective electrodes (Durst 1999). Sodium disorders were considered if serum sodium detection <130 mEq/L or >145 mEq/L. Normal range for potassium was considered to be 3.5-5mEq/L. Electrolytes were estimated within 24 hours of sustaining the trauma.

Descriptive statistics was applied to express the results. The changes in the electrolyte levels in different types of brain injuries were expressed in percentage. The association between the electrolyte levels and level of consciousness was assessed by using Pearson's correlation coefficient.

RESULTS

Hypernatremia was seen only in 4% of our patients. A greater incidence of hyponatremia was found in patients with subdural hematoma (16%), intracerebral hematoma (20%) and with diffuse axonal injury (4%). The incidence of hyponatremia was higher in patients with diffuse traumatic brain injuries(24%).Hyperkalaemia was detected only in 4% of subarachnoid haemorrhage whereas hypokalaemia was noted in 4% of intracerebral haemorrhage patients. In total, 75% of our TBI patients were found to have electrolyte disturbances.

We found a statistically significant ($p<0.05$) association between sodium levels and Glasgow coma scale($r = 0.634$).The positive correlation implies that lower the sodium level, lesser is the GCS.

DISCUSSION

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, including the syndrome of inappropriate antidiuretic hormone secretion, cerebral salt loss.

The incidence of sodium disorders was high (68%) in this study which is in accordance with the other studies (Donati-Genet et al., 2001; Cole et al., 2004).

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 insipidus. 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. Audibert et al., 2012; Harrigan 1996

Hyponatremia may also be caused by the activity of the brain natriuretic peptide (BNP) (Sudoh et al., 1988; Mukoyama et al., 1991). BNP is a potent diuretic, natriuretic, vasodilating agent, and an inhibitor of the secretion of aldosterone, renin, and vasopressin (Goldsmith 1987; Richards et al., 1985; Taikkanen et al., 1996). Increased BNP is most commonly found in patients with subarachnoid hemorrhage or hemorrhage at the base of the brain or in the third ventricle (Wijdicks

et al., 1991; Diringer et al., 1991).

Atrial natriuretic peptide (ANP) is the potential hormonal mediator of hyponatremia in intracranial disorders (Weinand et al., 1989).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 peptides (Walter et al., 1999).

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 natriuresis (Singh et al., 2002). 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 preload (Braunwald et al., 2001; Sviri et al., 2000).

ANP was suggested to 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 (Palmer 2000). At the same time sodium is being blocked from returning to the bloodstream and there is an increased glomerular filtration rate contributing to natriuresis and diuresis. In addition, the secretion of renin and aldosterone diminish as the adrenal gland is directly inhibited, and indirectly inhibition due to the suppression of renin release from the juxtaglomerular kidney cells (Palmer 2000; Tomida et al., 1998). The decrease in circulating aldosterone levels that is thought to prevent potassium wasting from occurring in conjunction with the sodium loss (Palmer 2000).

Brain natriuretic peptide (BNP), action is similar to ANP; it is a potent vasodilator, causes sodium and fluid excretion, and leads to reduced circulating levels of renin and aldosterone (Braunwald et al., 2001). Palmer et al suggested that it may be released as a protective measure for increased intracranial pressure, while Tomida et al theorized that it may be activated as a stress response to surgery or the intensive care setting or as a result of damage in the hypothalamic region (Palmer 2000; Tomida et al., 1998).

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. This explains the positive association of GCS and serum sodium levels.

CONCLUSION

Hyponatremia is the commonest electrolyte disturbance occurring in TBI.

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.

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