Electrolyte And Acid Base Disturbances In Patients With Severe Closed Traumatic Brain Injury

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Citation

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Abstract

Aim: The aim of this study was to evaluate the alteration of serum electrolytes and arterial blood gases as a secondary insult in patients with severe closed traumatic brain injury and to estimate its impact on patients' outcome.

Materials and Methods: 80 patients with severe closed traumatic brain injury were admitted to neurosurgical intensive care unit between November 2013 and November 2014. All patients were treated conservatively without surgical intervention. Data collected include demographic characteristics, daily ABG, and serum electrolytes for the first 5 days, as well as patients, outcome.

Results: Of the 80 patients, 71 (88.8%) were male and 9 (11.2%) were female with mean age of 27.45±16.46 year. Road traffic accident was the commonest cause of head injury in 65% of patients, and 18.8% of patients had traumatic brain injury only while the remaining 81.2% of the patients had associated systemic or spine injury. On day one, 71. 3% of the patients had abnormal pH with acidosis more commonly encountered in 37.5% of patients. 72.5% had abnormal serum Na+, with hyponatremia in 41.3% of the patients. Abnormal serum K+ was reported in 55% of patients with hypokalemia in 37.5%. Bad outcome was reported in 73.75%. Abnormal pH, serum electrolytes were significantly associated with bad outcome in all 5 readings reported for pH, serum Na+ and K+.

Conclusion: The results of this study showed that in patients with severe closed traumatic brain injury abnormal pH and serum Na+ and K+ are common events and are significantly associated with bad outcome

INTRODUCTION

Head injury is the leading cause of death in adult and pediatric trauma patients and is responsible for 80% of all trauma mortality (14). In head trauma, brain injury occurs due damage of the primary insult followed by potential secondary insult which may be the result of both ischemic and non-ischemic mechanisms. This may lead to further loss of potentially viable cerebral tissue and decreasing the chance of good functional recovery (18,19).The goal of treatment in patients with severe closed head injury without neurosurgical intervention is minimizing secondary injury (1). Secondary injury is influenced by different factors such as hypoperfusion, hyperperfusion of cerebral blood flow, impairment of cerebrovacular autoregulation, cerebral vasospasm, cerebral metabolic dysfunction, electrolyte derangements (2). The aim of this study is to evaluate the changes of arterial blood gas and serum Na+ and K+ in patients with severe closed head injury and their impact on functional recovery of the patients.

METHODS

In this study, the medical data of all patients with severe closed traumatic brain injury admitted to the emergency unit of Zagazig University Hospital, a referral trauma center, were collected between November 2013 and November 2014. All patients with severe traumatic brain injury, defined by an initial Glasgow Coma Scale ≤ 8 with closed head injury without neurosurgical intervention were included in this study. Patients with surgical intervention after initial resuscitation and patients with moderate and mild head injury were excluded from this study. All patients treated at the intensive care unit. Computed tomography was performed on arrival and 24 hours later or less if the patients showed more deterioration. Clinical and demographic characteristic including age, gender, associated injury, GCS

(20), type of trauma, daily arterial blood gases, and serum Na+, K+ for the first 5 days were considered. Na disorders were considered if serum Na+ detection <135 mEq/L or >145 mEq/L. normal range for serum K+ was considered to be 3.5-5.5mEq/L and the normal range for pH was considered to be 7.35-7.45. Any deviation from normal ranges was reported daily for the first 5 days. All patients received cerebral protective management in the form of endotracheal intubation, hyperventilation in an attempt to maintain arterial PCo2 at 25-30 mmHg. Patients received dehydrating measures including corticosteroids, mannitol, and diuretics. The acid base and electrolyte disturbance was corrected properly once reported. Outcome was reported on discharge from the intensive care unit to the ward or home with a minimum follow up of 3 months for survivors. Outcome was according to Glasgow Outcome Scale. Favorable outcome included both good recovery and moderate disability, while bad outcome included the remaining categories of sever disability, persistent vegetative state and death (20).

STATISTICAL ANALYSIS

Statistical analysis based on SPSS file version 10 software package for social science quantitative data expressed is frequency, percentage and the results carried out for analysis using descriptive t-test and chi-square test to compare continuous variables and categorical data respectively considering the significance of difference at P-value less than 0.05.

RESULTS

A total of 71 male (88.8%), and 9 female (11.2%) with mean age of 27.45± 16.46 year were reported in this study. Road traffic accident was the commonest cause of head injury in 52 (65%) of patients followed by falling from height in 12 (15%) patients. Clinical presentation on admission included vomiting in 42 (52.5%) of patients, convulsion in 29 cases and 32 patients were GCS 8, 13 patients GCS 7, while 4 patients were in deep coma. Tense brain edema was the commonest finding in 62 (77.5%) patients followed by traumatic brain contusions in 48 (60%) of the patients and traumatic subarachnoid hemorrhage in 24 (30%) of the patients. Associated injuries were encountered in 65 (81.8%) of patients. Orthopedic injuries were the commonest injury in 15 (18.8%) of patients while multiple system injuries occurred in 32 (40%) of patients. Abnormal acid base balance was encountered in 57 (71.3%) patients on the first day, acidosis was the commonest finding in 30 (37.5%) patients while alkalosis was reported in 27 (33, 8) patients. In the 30 patients with acidosis, metabolic acidosis was the most common finding in 23 patients (76.7%). Respiratory alkalosis was the commonest finding in 22/27 patient as shown in table (1). Serial measurement of pH in the next 4 days showed decreased incidence of abnormal acid base balance to be 53.2% of the living patient 41/77 and still associated with bad outcome as shown in table (2).

Abnormal serum sodium occurred in 58 (72.5%) of patients with hyponatremia occurred in 33 (41.3%) of patients as shown in table (1). Serum potassium abnormalities occurred in 44 (55%) with hypokalemia encountered in 30 (37.5%) of patients. The incidence of electrolyte disturbance varied at the next 4 days and still associated with bad outcome as shown in tables (3,4). Abnormal acid-base homeostasis and electrolyte derangements are more commonly encountered in patients with associated injury as shown in table (5). The changes were significantly associated with extreme of age than the middle age as shown in table (6). Good outcome was reported in 21/80 patients (26.25%) of the patients. The incidence of mortality was 43. 8% of the patients (35/80), 10 patients had persistent vegetative state, and 14 patients had sever disability as shown in table (7).

DISCUSSION

Severe traumatic brain injury is one of the main causes of morbidity and mortality in the world especially among younger population. Annually in the United States 1.5 million American citizens have a mild, moderate, or severe head injury. 50,000 people die and 80,000 to 90,000 people experienced long term disability (21). Despite the advances in trauma research, the exact mechanisms and biochemical cascade that lead to secondary brain insult is still poor (5). Traumatic brain injury results in a range of neurological and cognitive impairment. Posttraumatic endocrine complications also yield a great challenge of management of traumatic brain injury (4). Electrolyte derangements are common after neurologic injury, it could be partially iatrogenic by the administration of brain dehydrating measures (1).

The role of electrolyte abnormalities in the secondary traumatic brain injury cascade is being delineated and may offer a potential future therapeutic intervention (1). Potassium is the major intracellular cation, with relatively low extracellular levels. Cerebral injury can lead to polyuresis through a variety of mechanisms (1). Potassium derangements after severe traumatic brain injury implies that the normal buffering mechanisms that maintain the ionic homeostasis needed for establishment of the resting

membrane potential have been overcome (16). The incidence of serum K+ is higher than reported in previous studies. Adiga and others in their study reported 4% as the incidence of hypokalemia and hyperkalemia (1). Reinert and others reported increased dialysate potassium in 20% of measurements after traumatic brain injury. Their results provide strong evidence that prolonged ionic dysfunction, leading to cytotoxic swelling, which is a major cause of raised intracranial pressure and poor outcome after severe traumatic brain injury (16).

The incidence of sodium disorders was high 72.5% on the first day and varied between 55.8% to 66.2% on the following days of measurements. This coincides with other studies. Adiga and others reported 68% of their patients had sodium disorders, Born et al (4) reported that in their series of 109 patients with severe traumatic brain injur, 33% presented hyponatremia during the time course. About 15-30% of patients with traumatic brain injury have hypothalamic-pituitary dysfunction, particularly growth hormone deficiency, ACTH, TSH, and gonadotrophin deficiency. Hyponatremia may develop as a result of inappropriate secretion of antidiuretic hormone secretion or cerebral salt- wasting syndrome characterized by natriuresis (audibert. Harrigan). Hyponatremia may also cause by the activity of brain natriuretic peptide which is a potent diuretic, natriuretic, and vasodilator agent (7). Hyponatremia is associated with abnormal mental status including confusion, decreased consciousness, hallucination and coma (6). This could explain the significant association of hyponatremia with bad outcome in our study.

Moro and his colleagues evaluated the incidence of hyponatremia in 298 patients with head injury .50 (16.8%) patients reported hyponatremia, however, the authors included patients with mild and moderate head trauma, with and without surgical intervention. In their study, The incidence of decreased sodium level was more in patients with Severe head injury, 46/50 of their patients with hyponatremia were GCS<8. Born et al, reported that in their series of 109 patients with severe traumatic brain injury, 36 patient (33%) presented hyponatremia during the time course (13).

Patients with severe traumatic brain injury have a high risk of developing hypernatremia over the course of their intensive care unit stay due to coexistence of predisposing factors such as impaired sensorium, altered thirst, and central diabetes insipidus with polyurea and increased insensible losses (20).Maggiore and his colleagues in their study of 130 patients with severe traumatic brain injury reported 51.5% incidence of hypernatremia, the occurrence of hypernatremia was highest in patients with suspected diabetes insipidus and was associated with increased risk of death (12). The authors reported 26.9% of mortality within 14 days of ICU admission

Previous studies claimed brain tissue acidosis to mediate brain damage after traumatic and ischemic brain insults (10,18). A low pH is believed to increase edema formation due to activation of an Na+/ H+ antiporter system coupled with a passive Cl -/HCO3- antiporter which increased inflow of Na+, Cl- and osmotically obligated water into the cell during acidosis. Acidosis may interfere with mitochondrial ATP generation both directly and indirectly which can impair mitochondrial metabolism (11). Furthermore, the formation of free radicals, one of the principal causes of delayed cell death after traumatic brain injury is significantly increased at low pH values (15)

Traumatic brain injury profoundly altered the cerebral acid-base homeostasis and brain tissue acidosis can cause neuronal damage. Brain edema is one of major causes of morbidity and mortality following traumatic brain injury. Secondary injury as acidosis exacerbates cerebral edema by altering the expression of water channel in the brain (23)

In the current study, the most common arterial blood gas imbalance upon admission was metabolic acidosis in 23/80 patients and respiratory alkalosis in 22/80 patients. These changes were significantly associated with bad outcome. There are not many studies evaluating the effect of arterial acid-base balances on mortality. In a study of Zupping and others, they reported CSF metabolic acidosis with respiratory alkalosis and hypoxemia in the arterial blood was the commonest characteristic finding after head injury and are associated with severe brain damage and bad outcome (24). Eberhard and others reported the association of degree of metabolic acidosis at the time of admission with the probability of acute lung injury (8). Rahmimi and his colleagues in their study of pediatric patients with severe head injury concluded that initial mixed metabolic acidosis plus respiratory acidosis and GCS are significant predictors of mortality with 31.5% mortality rate (14).

Our results showed significant correlation between extreme of age and abnormal pH, Serum Na+ and serum K+ and had bad outcome compared to middle aged patients. There are some differences in severe head injuries between adults and children. Our results are different from that previously

published which showed better outcome in children. Alberico et al., in their study on severe head injured patients had a significantly higher incidence of good outcome in pediatric (43%) than adult patients (28%) and had a significantly lower mortality rate (24%) than the adult patients (45%) (2).

In summary, to our knowledge, this is the first Egyptian study of acid-base and electrolyte disturbances in patients with severe traumatic brain injury and highlights the importance of such derangements in patients with severe closed head injury being a major secondary insult with high morbidity and mortality.

Figure 1

The incidence of acid-base and electrolyte disturbance in 5 days

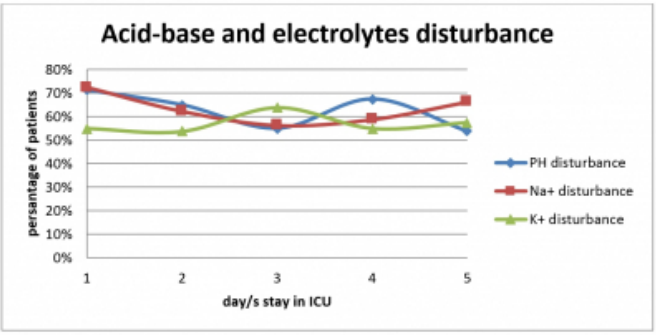

Table 1

The incidence of serum acid-base and serum electrolytes (sodium and potassium) disturbance in severe head injury within 24 hours

Occurrence of disturbance	Number of patients No (%)	Percentages (%)
Acid-base PH (normal range)	23	28.7 %
Acid-base PH (**abnormal** range)	**57**	**71.3 %**
- Alkalosis:	*27*	*33.8 %*
• Metabolic	5	6.3 %
• Respiratory	22	27.5%
- Acidosis:	*30*	*37.5 %*
• Metabolic	23	28.75%
• Respiratory	7	8.75 %
Serum sodium (normal range)	22	27.5 %
Serum sodium (**abnormal** range)	**58**	**72.5 %**
- Hypernatremia	25	31.2 %
- Hyponatremia	33	41.3 %
Serum potassium (normal range)	36	45.0 %
Serum potassium (**abnormal** range)	**44**	**55.0 %**
- Hyperkalemia	14	17.5 %
- Hypokalemia	30	37.5 %

Table 2

Serial measurements of serum pH level and outcome

Serial acid-base level	Glasgow Outcome Scale: Bad Outcome	Glasgow Outcome Scale: Good Outcome	Chi-Squire	P value
PH within 24 hours n(80):				
Normal range 23(28.7%)	59(73.7%)	21(26.3%)	15.28	0.000
Abnormal 57(71.3%)	10 (43.5%)	13 (56.5%)		
	49 (86.0%)	8 (14.0%)		
PH within 2nd day n(79):				
Normal range 28(35.4%)	58(73.4%)	21(26.6%)	12.19	0.000
Abnormal 51(64.6%)	14 (50.0%)	14 (50.0%)		
	44 (86.3%)	7 (13.7%)		
PH within 3rd day n(77):				
Normal range 35(45.4%)	56(72.7%)	21(27.3%)	14.68	0.000
Abnormal 42(54.6%)	18 (51.4%)	17 (48.6%)		
	38 (90.5%)	4 (9.5%)		
PH within 4th day n(77):				
Normal range 25(32.5%)	56(72.7%)	21(27.3%)	8.01	0.004
Abnormal 52(67.5%)	13 (52.0%)	12 (48.0%)		
	43 (82.7%)	9 (17.3%)		
PH within 5th day n(77):				
Normal range 36(46.8%)	56(72.7%)	21(27.3%)	13.57	0.000
Abnormal 41(53.2%)	19 (52.8%)	17 (47.2%)		
	37 (90.2%)	4 (9.8%)		

Table 3

Serial measurements of serum sodium Na+ level and outcome

Serial Na^+ level	Glasgow Outcome Scale		Chi–Squire	P value
	Bad Outcome	Good Outcome		
Na^+ within 24 hours n(80):				
Normal range 22(27.5%)	59(73.7%)	21(26.3%)	8.84	0.002
Abnormal 58(72.5%)	11 (50.0%)	11 (50.0%)		
	48 (82.8%)	10 (17.2%)		
Na^+ within 2nd day n(79):	58(73.4%)	21(26.6%)		
Normal range 30(38.0%)	16 (53.3%)	14 (46.7%)	9.99	0.001
Abnormal 49(62.0%)	42 (85.7%)	7 (14.3%)		
Na^+ within 3rd day n(77):	56(72.7%)	21(27.3%)		
Normal range 34(44.2%)	18 (25.9%)	16 (47.1%)	12.02	0.000
Abnormal 43(55.8%)	38 (88.4%)	5 (11.6%)		
Na^+ within 4th day n(77):	56(72.7%)	21(27.3%)		
Normal range 32(41.6%)	17 (53.1%)	15 (46.9%)	10.61	0.001
Abnormal 45(58.4%)	39 (86.7%)	6 (13.3%)		
Na^+ within 5th day n(77):	56(72.7%)	21(27.3%)		
Normal range 26(33.8%)	14 (53.8%)	12 (46.2%)	7.05	0.003
Abnormal 51(66.2%)	42 (82.4%)	9 (17.6%)		

Table 4

Serial measurements of serum K+ level and outcome

Serial K^+ level	Glasgow Outcome Scale		Chi–Squire	P value
	Bad Outcome	Good Outcome		
K^+ within 24 hours n(80):				
Normal range 36(45.0%)	59(73.7%)	21(26.3%)	23.79	0.000
Abnormal 44(55.0%)	17 (47.2%)	19 (52.8%)		
	42 (95.5%)	2 (4.5%)		
K^+ within 2nd day n(79):				
Normal range 37(46.8%)	58(73.4%)	21(26.6%)	21.88	0.000
Abnormal 42(53.2%)	18 (48.6%)	19 (51.4%)		
	40 (95.2%)	2 (4.8%)		
K^+ within 3rd day n(77):				
Normal range 28(36.4%)	56(72.7%)	21(27.3%)	11.46	0.000
Abnormal 49(63.6%)	14 (50.0%)	14 (50.0%)		
	42 (85.7%)	7 (14.3%)		
K^+ within 4th day n(77):				
Normal range 35(45.5%)	56(72.7%)	21(27.3%)	14.68	0.000
Abnormal 42(54.5%)	18 (51.4%)	17 (48.6%)		
	38 (90.5%)	4 (9.5%)		
K^+ within 5th day n(77):				
Normal range 33(42.9%)	56(72.7%)	21(27.3%)	13.1	0.000
Abnormal 44(57.1%)	17 (51.5%)	16 (48.5%)		
	39 (88.6%)	5 (11.4%)		

Table 5

Correlation of associated injury with pH, Serum Na+, serum K+, and outcome

	Associated injury	Disturbance	GOS		Chi–Squire	P value
			Bad GOS 59(73.7%)	Good GOS 21(26.3%)		
PH	Without 15(18.8%)	No 6(40.0%)	1 (16.7%)	5 (83.3%)	5.4	0.020
		Yes 9 (60.0%)	7 (77.8%)	2 (22.2%)		
	Present 65(81.2%)	No 17 (26.2%)	9 (52.9%)	8 (47.1%)	8.87	0.002
		Yes48 (73.8%)	42 (87.5%)	6 (12.5%)		
Na	Without 15(18.8%)	No 7(46.7%)	1 (14.3%)	6 (85.7%)	5.52	0.018
		Yes 8 (53.3%)	6 (75.0%)	2 (25.0%)		
	Present 65(81.2%)	No 15 (23.1%)	6 (40.0%)	9 (60.0%)	19.5	0.000
		Yes 50(76.9%)	46 (92.0%)	4 (8.0%)		
K	Without 15(18.8%)	No 8 (53.3%)	1 (12.5%)	7 (87.5%)	5.4	0.020
		Yes 7 (46.7%)	5 (41.4%)	2 (28.6%)		
	Present 65(81.2%)	No 28 (43.1%)	18 (64.3%)	10 (35.7%)	9.72	0.001
		Yes 37(56.9%)	35 (94.6%)	2 (5.4%)		

Table 6

Correlation of extreme of age with the pH, Serum Na+, serum K+, and outcome

	Age	Disturbance	GOS		Chi–Squire	P value
			Bad GOS 59(73.7%)	Good GOS 21(26.3%)		
PH	Extreme 25 (31.2%)	No 7 (28.0%)	3 (42.9%)	4 (57.1%)	8.3	0.003
		Yes 18(72.0%)	17 (94.4%)	1 (5.6%)		
	Middle 55 (68.8%)	No 16 (29.1%)	7 (43.8%)	9 (56.2%)	8.06	0.004
		Yes 39(70.9%)	32 (82.1%)	7 (17.9%)		
Na	Extreme 25 (31.2%)	No 8 (32.0%)	4 (50.0%)	4 (50.0%)	6.6	0.010
		Yes 17(68.0%)	16 (94.1%)	1 (5.9%)		
	Middle 55 (68.8%)	No 14 (25.5%)	7 (50.0%)	7 (50.0%)	3.9	0.046
		Yes 41 (74.5%)	32 (78.0%)	9 (22.0%)		
K	Extreme 25 (31.2%)	No 11 (44.0%)	5 (45.5%)	6 (54.5%)	4.5	0.03
		Yes 14(56.0%)	12 (85.7%)	2 (14.3%)		
	Middle 55 (68.8%)	No 25 (45.5%)	17 (68.0%)	8 (32.0%)	1.7	0.1
		Yes 30 (54.5%)	25 (83.3%)	5 (16.7%)		

Table 7

Glasgow Outcome Score of patients

Glasgow Outcome Score GOS	Number of patients	Percentage %
Bad outcome:	59	73.75 %
(1) Death	35	43.8 %
(2) Vegetative	10	12.5 %
(3) Severe disability	14	17.5 %
Good outcome:	21	26.25 %
(4) Moderate disability	10	12.5 %
(5) Good recovery	11	13.8 %

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