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Serum Electrolytes Profile Of Patients With Traumatic Brain Injury; Prospective Observational Study In Sokoto, Nigeria

Ogunleye OO

Ismail NJ

Lasseini A

Shehu BB.

Regional Centre for Neurosurgery (RCN),
Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria

Abstract:

Background: Traumatic brain injury (TBI) constitutes major health problem in both developing and developed nations, with significant morbidity and mortality. Electrolytes derangements are commonly seen and as well may worsen the managements of the patients. The aim of this is to demonstrate the prevalence of electrolytes derangements and the relationship between the electrolytes derangements and severity of traumatic brain injury (TBI) patients.

Method: All patients with TBI who merited inclusion criteria were recruitment using systematic sampling technique into three groups based on severity of injury. Serum electrolytes (Sodium, Potassium and Chloride) was measured within 24hrs after resuscitation. Serum electrolytes concentration was correlated with varying degree of severity of brain injuries.

All patients had standard treatment according to our institutional protocol for TBI patients.

Results: Sixty (60) patients (Mean age 35.1± 11.4 years; 83.3% male, 16.7% female).

This study showed that 18.3% had hyponatremia, 8.3% hypokalaemia and 6.7% hypochloremia. A Tukey Post Hoc Test revealed that the mean sodium concentration in Severe TBI (133.52 ± 7.26) was significantly lower than the mean sodium concentration in Moderate TBI (138.20 ± 4.12) and the mean sodium concentration in Mild TBI (140.76 ± 3.28).

Conclusion: Thirty-three percent of the patients had electrolytes derangements of which sodium is the most affected electrolytes. Monitoring of serum electrolytes during the management of TBI patients is of utmost important and requires earliest possible correction.

Keywords: Traumatic brain injury, Serum Electrolytes.

I. INTRODUCTION

Traumatic Brain Injury (TBI) is the leading cause of death in young adults. TBI will surpass many diseases and will become the third cause of death and disability in the general population by the year 2020 (1, 2). The financial cost implication in the management of patients with TBI is excessively high and burdensome to the relative and the government (3).

Electrolytes derangements are seen following neurologic injury, either from iatrogenic administration of osmotic diuretic and corticosteroid or due to secondary brain injury (4). Other causes of electrolytes derangements in TBI includes syndrome of inappropriate antidiuretic hormone secretion (SiADH) and cerebral salt wasting (5, 6).

Neurological symptoms can result from rapidly decreasing serum sodium or rapidly correcting serum sodium and this makes sodium derangements very important in clinical practice(7). Potassium has high intracellular

concentration with comparatively low extracellular concentration levels. Small changes in serum potassium can severely affect nerve conduction, heart rhythm and muscle contraction (8).

Improving TBI outcomes will greatly reduce the heavy societal and economic burden currently associated with this condition. Electrolytes imbalance appear to be a primary driver of TBI pathophysiology. In addition, the role of electrolyte abnormalities in the secondary neurologic injury cascade is being delineated and may offer therapeutic intervention in future.

In order to prove the likelihood of therapeutic intervention that will be of benefit for TBI patients with electrolytes derangements, there must be demonstration of clinical picture of electrolytes derangements in patients with TBI.

The present study aims to demonstrate prevalence of electrolyte derangements in our environment as well as confirm if TBI in our environment is associated with electrolyte derangements.

II. METHODOLOGY

Sixty patients with TBI were recruited for the study. Systematic sampling technique was used to recruit patients into various categories of TBI (based on post resuscitation Glasgow Coma Score), each has twenty patients. Patients with other systemic injuries and previously known or recently diagnosed systemic illnesses e.g. Diabetic, Hypertension, etc. were excluded.

Clinical evaluation and resuscitation according to our hospital protocol was done. Serum electrolytes (Sodium, Potassium and Chloride) were estimated by an electrolyte analyser. Major components of electrolyte analyser are reagents, electrode module, peristaltic pump and sample probe. The method of analysis used was Ion Selective Electrode (ISE) and the results were expressed as mmol/L.

Descriptive statistics was used to express results. The electrolytes changes in different categories of TBI was expressed in percentages and concentrations (mmol/L). The association between serum electrolytes and various categories of TBI was assessed using one way ANOVA and Turkey Post Hoc Test.

III. RESULTS

The socio-demographic data are represented in Table 1 below. Majority of the patients for the study (81.7%) were below 40 years of age while only 18.35% were between 41-65 years of age {X = 35.50 ± 11.35}. Fifty of the recruited patients (83.3%) were male while females were 17.7% with female-male ratio 1:5.

Figure 1 depicts the serum concentration of electrolyte among TBI patients. The serum electrolytes among varying categories of TBI patients were showed by Figure 2 while Table 3 showed an association between various categories of TBI and serum electrolytes.

VARIABLES	FREQUENCY (%)
Age of Respondents (years)	
18 – 40	**49 (81.7)**
41 – 65	**11 (18.3)**
Mean = 35.50 ± 11.35	
Gender	
Males	**50 (83.3)**
Females	**10 (16.7)**
Tribe	
Hausa	**45 (75)**
Yoruba	**8 (13.3)**
Igbo	**6 (10)**
Igala	**1 (1.7)**
Religion	
Islam	**49 (81.7)**
Christian	**11 (18.3)**

Table 1: Demographic data

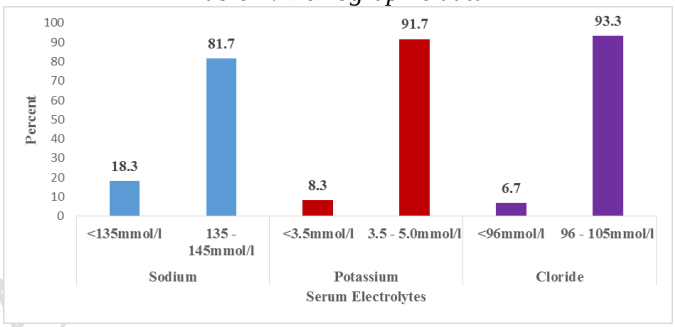

Figure 1: Serum electrolytes concentration among TBI patients

Variables	Traumatic Brain Injury		
	Mild	**Moderate**	**Severe**
	Mean ± SD	**Mean ± SD**	**Mean ± SD**
Sodium	140.76 ± 3.28	138.20 ± 4.12	133.53 ± 7.26
Potassium	4.06 ± 0.25	5.59 ± 7.64	3.73 ± 0.39
Chloride	99.71 ± 3.07	99.75 ± 2.79	99.71 ± 3.07

Table 2: Serum electrolytes concentration among categories of TBI

VARIABLES	Traumatic Brain Injury (GCS)			p value
	Mild (14 – 15) Mean ± SD	**Moderate (13 – 9) Mean ± SD**	**Severe (3 – 8) Mean ± SD**	
Sodium	140.76 ± 3.29 **(A)**	138.20 ± 4.12 **(B)**	133.53 ± 7.26 **(C)**	p = **.003****
Potassium	4.06 ± 0.25 **(D)**	5.59 ± 7.64 **(E)**	3.73 ± 0.39 **(F)**	p = .376
Chloride	99.71 ± 3.07 **(G)**	99.75 ± 2.79 **(H)**	99.55 ± 2.87 **(I)**	p = .777

**p = <.05; **p = <.001; One – way ANOVA test*
A vs B, p = .252 A vs C, p = .001 B vs C, p = .016
D vs E, p = .818 D vs F, p = .971 E vs F, p = .396
G vs H, p = .999 G vs I, p = .819 H vs I, p = .801

Table 3: Association between Traumatic Brain Injury (TBI) and Serum Electrolytes

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A Tukey Post Hoc Test reveals that the mean sodium concentration in Severe TBI (133.52 ± 7.26, p = < .001) was significantly lower than the mean sodium concentration in Moderate TBI (138.20 ± 4.12, p = <.016) and the mean sodium for Mild TBI (140.76 ± 3.28, p = < .001). However the mean Sodium concentration in Moderate TBI was not statistically significantly lower than Mild TBI (140.76 ± 3.28, p = < .252).

IV. DISCUSSION

The high percentage of young adult in this study (81.7%) shows high prevalence of TBI among the youth as reported by Hyder *et al* (1) and this makes TBI a leading cause of morbidity and mortality among the youth population worldwide (9). In addition, male gender has been the most involved in trauma due to his adventurous role and this is confirmed in this study with male constitutes 83.3% of the study group.

The incidence of hyponatremia in this study was small (18.3%) which is in variance with studies by Adigal *et al* (10) where it was 68%, Donati-Genet *et al* (11) and Cole *et al* (12). This disparity may be explained by small number of patients used by other studies mentioned. Hyponatremia observed may have occurred either as a result of syndrome of inappropriate release of antidiuretic hormone (SiADH) which is normally presents as dilutional hyponatremia or from cerebral-salt wasting syndrome featured by Naitriuresis (5,6,13). The incidence of hypokalaemia and hypochloremia was small as well 8.3% and 6.7% respectively, this is in conformity with study by Adigal *et al* (10). The low serum potassium seen in some of the patients may be due to an increase in urinary loss from brain trauma. Potassium is the second most common electrolyte which underwent significant derangements following the serum sodium levels and this is in accordance with the study conducted by Pomeranz *et al* (14). In our study low serum potassium was seen in 8.3% of patients, as no patient 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 beta2-adrenergic stimulation of the Na+-K+ pump.

In this study, there was statistically significant association between severe TBI and hyponatremia (p= <.001). This finding was also seen in study by Adigal *et al* (10). The positive association between severe TBI and Hyponatremia is an established fact. Alteration in conscious level and coma are well established clinical features of hyponatremia and explain the association between Glasgow coma score (GCS) and serum hyponatremia.

V. CONCLUSION

Traumatic brain injured patients are at risk of developing electrolytes derangements. Hyponatremia is the commonest of all electrolytes derangements followed by hypokalaemia. Electrolytes derangements play a major role in secondary brain injury, therefore early detection and correction of the electrolytes derangement are essential to early recovery and will prevent further neurologic injury in TBI patients.

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