

Research Article

Serum sodium: A useful prognostic factor for non-severe traumatic brain injury in children?

Parsa Yousefichaijan¹, Kiandokht Karimian², Abdolghader Pakniyat^{3*}, Mina Sheikhi-dezfoli⁴ and Mohammad Rafiei⁵

¹Associated Professor of Pediatric Nephrology, Arak University of Medical Sciences, Arak/Iran

²Emergency Medicine Specialist, Arak University of Medical Sciences, Arak/Iran

³Emergency Medicine Resident, Student Research Committee, Arak University of Medical Sciences, Arak/Iran

⁴MD, Arak University of Medical Sciences, Arak/Iran

⁵Associated Professor of Biostatistics, Arak University of Medical Sciences, Arak/Iran

Abstract

Background: Traumatic brain injury is one of the most common causes of mortality and morbidity in children and young age people. Following brain trauma and damage to the blood brain barrier, sodium entrance in the interstitial space induces fluid accumulation, causing cerebral edema and worsening of symptoms.

Objectives: In this study for first time we investigate the relationship between serum sodium and prognosis of children whose imaging has no severe brain trauma or acute injury.

Materials and methods: In a prospective-descriptive study patients aged between one month to 18 years old with isolated mild and moderate brain injury were studied. Basic information, primary GCS and CT reports were recorded and preliminary tests such as serum sodium were requested. During hospitalization, daily GCS, daily sodium, result of 6 hour later brain CT scan (if indicated), were recorded. Data was analyzed by SPSS version 18.

Results: (Chi-square) X² showed a significant relation (p value=0.05) between sodium level and gender ($0.25=P$ value) as well as Sodium level and GCS at arrival ($P=0.001$) while no relation was founded between Sodium level and patients' consciousness level in days after arrival.

Conclusions: There is significant difference between the changes in sodium & sex, arrival GCS & seizures during admission, but there was no effect on the final prognosis and no significant difference between the changes in sodium and age & patients' GCS in days after admission.

Introduction

Background: Traumatic brain injury is one of the most common causes of mortality and morbidity in children and young age people [1]. Its mortality is about 30-40 percent that mostly occurs immediately after trauma and is manifested by progressive symptoms and loss of consciousness. In some cases, normal consciousness is maintained followed by gradual diminution of consciousness and sometimes sudden death; this is secondary to electrolyte changes, brain edema, hypoxia & ischemia [2]. In these patients, consciousness per se is not an appropriate criterion to estimate the severity of trauma. Prognosis of these patients has been different in various centers making identification and putting them in high-risk group a critical step [3]. Electrolyte imbalances are seen in acute phase of head trauma with the Sodium change as the most prevalent one [4,5]. Following brain trauma and damage to the blood brain barrier, sodium entrance in the interstitial space induces fluid accumulation, causing cerebral edema and worsening of symptoms [6,7].

Objectives: Importance of sodium changes in the prognosis of patients with brain trauma and subarachnoid hemorrhage has been addressed in some studies [4,8]. In this study for first time we investigate the relationship between serum sodium and prognosis of children whose imaging has no severe brain trauma or acute injury.

Materials and methods

In a prospective-descriptive study from July 2013 to the February 2014, all patients aged between one month to 18 years old (referred to emergency department of Valiasr Hospital) with isolated mild and moderate brain injury were studied. Patients were selected based on inclusion and exclusion criteria using Poisson sampling. Inclusion criteria consisted all children aged from one month to 18 years old with consciousness score between 9 and 15 & an indication for admission due to loss of consciousness, vomiting more than 4 times, severe trauma & etc. Patients with acute hemorrhagic lesions on primary CT (except cerebral edema), lesions requiring surgery (including depressed fractures) & patients with a history of underlying diseases or drug consumption were excluded from the study. Basic information including patient's history and demographic information (age & sex), type of trauma, primary GCS and CT reports were recorded and preliminary tests such as serum sodium were requested. During

Correspondence to: Abdolghader Pakniyat, Emergency Medicine Resident, Student Research Committee, Arak University of Medical Sciences, Arak/Iran, Tel: +98-912-1947601; E-mail: abdolghader.pakniyat@gmail.com

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hospitalization, daily GCS, result of 6 hour later brain CT scan (if indicated) and daily sodium, were recorded. In case of hyperglycemia, Serum sodium level was corrected. During hospitalization, patients with a decrease in consciousness level re-underwent CT brain and necessary treatments such as correction of electrolyte were fulfilled. In case of full recovery, including cessation of nausea and vomiting, oral tolerance and full consciousness, patients were discharged with warning signs.

Data was analyzed by SPSS version 18. Of descriptive statistics, mean and frequency and of statistical analysis chi-square test were used; significance level was assigned less than 0.05.

Before entering the study, all patients and their parents were given proper explanation & informed consent was obtained. The study complied with all the provisions of the Declaration of Helsinki and University of medical ethics statements.

Results

During the study, 110 children with mild to moderate isolated brain injuries (that did not require a surgical procedure on arrival) which their CT scans did not show evidence of acute brain lesions (except cerebral edema) were included; of whom 61 patients (55.5%) were male (Table 1).

At time of arrival, 87 patients (79.1 percent) were at normal levels of GCS (=15), 19 patients (17.3%) had been in the 14-13 level

Table 1. Age distribution in children with non-sever traumatic brain injury.

Age	Number
< 1 year	9 (8.2%)
1-4 years	30 (27.3%)
5-9 years	52 (47.3%)
10-14 years	16 (14.5%)
15-18 years	3 (2.7%)

Table 2. GCS distribution during hospitalization in children with non-sever traumatic brain injury; Day: hospitalization day, GCS: Glasgow coma scale.

GCS \ Day	0	1	2	3	4	5
15	87(79.1 %)	103(93.6)	33(30%)	15(13.6%)	8(7.3%)	6(5.5%)
13-14	19(17.3 %)	5(4.5)	4(3.6%)	0	0	0
13>	4(3.6 %)	2(1.8)	1(0.9%)	1(0.9%)	1(0.9%)	1(0.9%)
Total (n)	110(100)	110(100)	38(34.5%)	16(14.5%)	9(8.1%)	7(6.3%)
Discharge(n)	0	0	72(65.5%)	94(85.5%)	101(91.9%)	103(93.7%)

Table 3. Serum Na distribution during hospitalization in children with non-sever traumatic brain injury; Day: hospitalization day.

GCS \ Day	0	1	2	3	4	5
15	98(89.1)	99(90)	38 (34.5%)	16(14.5%)	9(8.1%)	7(6.3%)
13-14	12(10.9)	11(10)	0	0	0	0
Total (n)	110(100)	110(100)	38 (34.5%)	16(14.5%)	9(8.1%)	7(6.3%)
Discharge(n)	0	0	72(65.5%)	94(85.5%)	101(91.9%)	103(93.7%)

Table 4. Serum Na distribution according to gender in children with non-sever traumatic brain injury.

Na	Sex		total	P value
	male	female		
145-135	58	40	98	0.025
<135	3	9	12	
total	61	49	110	

and 4 patients (3.6%) had GSC below 13. GCS was recorded daily in hospitalized patients. All patients with full consciousness and stable vital signs were discharged after 5 days of hospitalization (Table 2).

Sodium of 99 patients (90%) was in range of 145-135 while 11 patients (10%) had serum levels below 135. daily Sodium check was requested for admitted patients (Table 3).

There was no significant relation between age and serum sodium level (p=0.38).

(Chi-square) X2 showed a significant relation between sodium level and gender (PV=0.025) (Table 4).

By using (Chi-square) X2, a significant correlation between sodium level and patients` GCS on arrival day was found (p=0.001) but there was no similar relation between serum sodium & consciousness of patients in other days of admission. (P>0.05) (Table 5).

Five patients (5.4%) had seizure; there was a significant relation between serum Sodium level and seizure during the hospitalization (p=0.009) (Table 6).

Discussion

In this study, we investigated the role of serum sodium changes in prognosis of brain injury at the age of one month to 18 years old. Several studies have been conducted regarding sodium change in prognosis of head injury, while no study has been done to assess the relation between serum sodium and mild to moderate traumatic brain injury (without acute findings in imaging) so far. Therefore, we did not incur any conflict with other studies.

The study conducted in 2011 by Lohani S entitled as `Hyponatremia in patients with traumatic brain injury` concluded that Hyponatremia due to SIADH was observed frequently in patients with traumatic brain injury but was not as good as CT in assessing the severity of brain lesions [9].

Table 5. Serum Na distribution according to GCS on admission day in children with non-sever traumatic brain injury.

Na	GCS on admission day			total	P value
	15	14-13	<13		
145-135	84	10	4	98	0.001
<135	3	9	0	12	
total	87	19	4	110	

Table 6. Serum Na distribution seizure in children with non-sever traumatic brain injury.

Na	Seizure		total	P value
	No	Yes		
145-135	96	2	98	0.009
<135	9	3	12	
total	105	5	110	

In a study done by Human T in 2012, Conivaptan was useful in patients with acute Hyponatremia after traumatic brain injury [10]. In a study of Katada R on mice Ethanol, after head injury caused an increase in cerebral edema & Hyponatremia [11].

A study by Costa KN showed that the most common cause of Hyponatremia in the brain lesions was Sodium excretion secondary to brain natriuretic hormone and there was no clear relationship between brain natriuretic hormone, aldosterone and vasopressin with serum sodium [12].

Dhai R study showed that Bolus conivaptan was not only effective in rapid improvement of acute Hyponatremia but also associated with a significant decrease in ICP [13].

Prevention from recurrence of Hyponatremia can be very effective in management of patients with traumatic brain injury [14].

Moro N deduced that increased possibility of Hyponatremia after head injury can clearly lead to a decreased favorable prognosis in patients with Hyponatremia [15].

In a study performed by Wright WL in 2012 in the United States as 'sodium and fluid therapy in acute traumatic brain injury', it was concluded that sodium and fluid therapy directly affects the brain edema and intracranial pressure. Sodium was a leading factor in the size of cerebral neurons and its deficiency caused brain edema. On the other hand impaired brain perfusion caused cerebral ischemia and neuronal damage. Asmothrapy is a method for the treatment of cerebral edema with a need for further studies in this area [16].

Paiva WS et al in a study in 2011 showed that the frequency of sodium imbalances in patients with cerebral hemorrhage, SDH, DAI & diffuse cerebral lesions (compared to patients with brain Contusion) were higher and there was no association between sodium disorders & focal lesions [17].

In study by Cintra Ede A, Incidence of sodium imbalances in deceased patients was higher during the first week after the injury [18].

Our study showed a statistically significant difference between the changes in sodium & sex, arrival GCS & seizures during admission, but there was no effect on the final prognosis and no significant difference between the changes in sodium and age & patients' GCS in days after admission.

Conclusion

In our study regarding lack of significant differences in sodium imbalances (hyponatremia or hypernatremia), it cannot predict prognosis in the mild and moderate brain injury (without acute lesions in the imaging). Regarding limited time of our practice, further studies with more patients in multiple centers and for longer time are prudent. In addition better results can be achieved by including the cases with brain hemorrhage and changes in CT scan.

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Conflict of interest

Authors have no conflict of interest to declare.

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