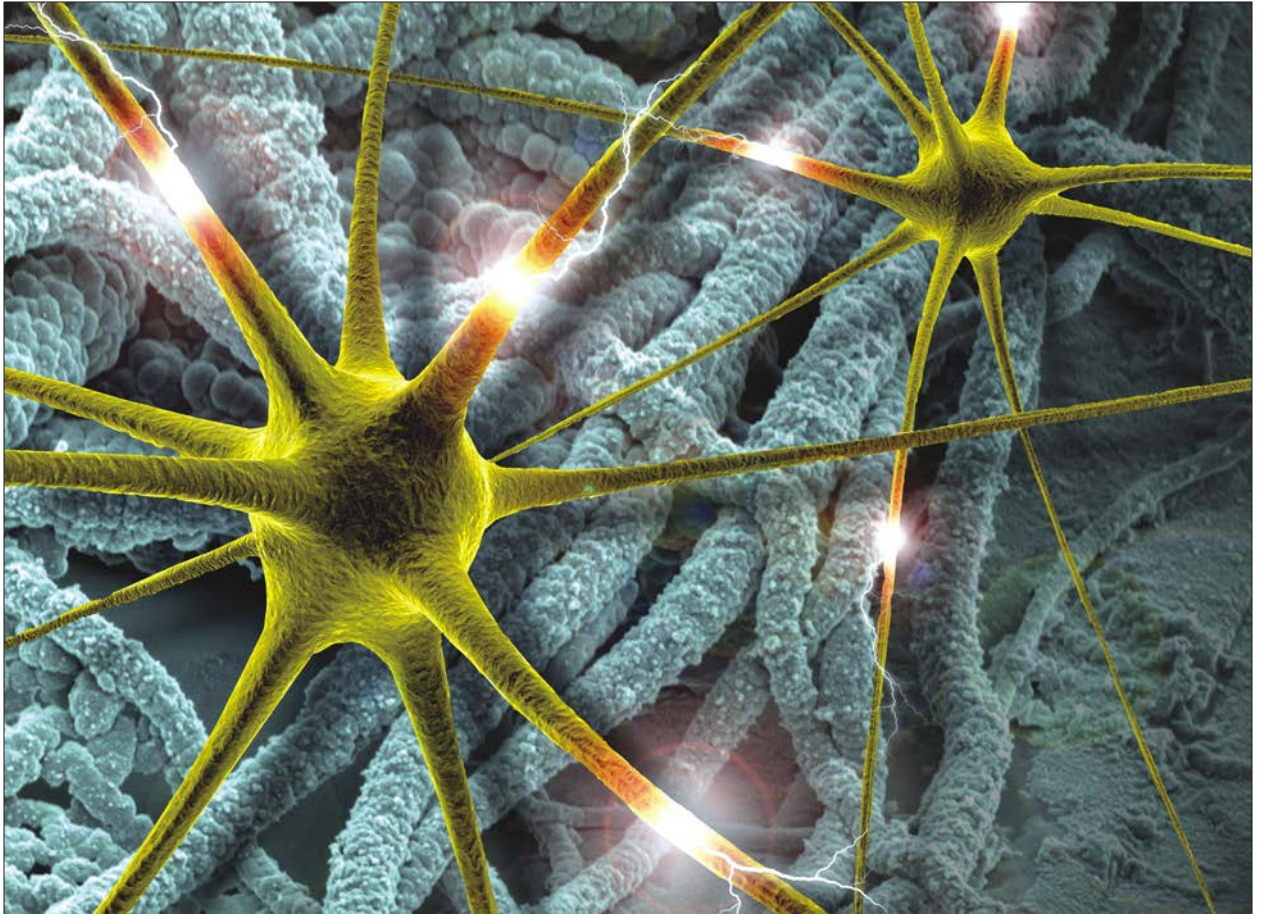




Current Research in **Neuroscience**

ISSN 1996-3408



Academic
Journals Inc.

www.academicjournals.com

Hyponatremia in Acute Head Injury: Correlation with Severity and Mortality

¹Fouad H. El-Dabah and ²Mohammed E. Elgeballi

¹Department of Forensic Medicine and Clinical Toxicology, Al-Azhar University, Cairo, Egypt

²Department of Neurosurgery, Al-Azhar Faculty of Medicine, Al-Azhar University, Cairo, Egypt

Corresponding Author: Fouad H. El-Dabah, Department of Forensic Medicine and Clinical Toxicology, Al-Azhar Faculty of Medicine, Al-Azhar University, Cairo, Egypt

ABSTRACT

Disorders of sodium and fluid regulation are common in patients with central nervous system disorders. To avoid deleterious effects of hypo-osmolality, sodium concentration must be preserved within normal range. It is hypothesized that, hyponatremia measured at admission can be used as an indicator or predictor for fatal outcome associated with head injuries. Thus, the present study designed to test this hypothesis. Thirty patients with acute head injury were included as a study group. In addition, another 30 apparently healthy subjects matched for age and gender were included as a control group. Both serum and urine concentration of sodium were done. Males were predominant; there was significant decrease of blood sodium concentration and increase of urine sodium concentration in study in comparison to control group. In addition, both blood and urine sodium concentrations correlated with GCS. Furthermore, urine sodium had more significant predicting power for mortality in those patients. Its predicating power was better than GCS. Both urine sodium concentration and GCS are good predictors of mortality in cases with acute head injury. Urine sodium concentration had a better predictability than GCS.

Key words: Hyponatremia, disorders of sodium, fluid regulation

INTRODUCTION

In healthy subjects, osmolality of the serum is maintained relatively constant between 285 and 295 mOsm kg⁻¹. Hypoosmolality must be avoided due to its deleterious effects and since sodium is the major determinant of serum osmolality, the management principle is to maintain sodium at least within the normal range of 135 to 145 mmol L⁻¹ (Bhardwaj and Ulatowski, 2004).

Central hyponatremia is that caused by diseases of the central nervous system. It had a death rate 60 times higher than that for patients without it (Roca-Ribas *et al.*, 2002).

When hyponatremia is associated with hypotonicity, it contributes to increased intracellular fluid volume, cause or enhances cerebral edema and intracranial hypertension, with subsequent brain injury (Brimioulle *et al.*, 2008).

Hypoosmolality and hypovolemia had deleterious effects as they worsen cerebral edema and cerebral ischemia (Bhardwaj, 2006).

It must be confirmed that, disorders of sodium and fluid regulation are common in patients with central nervous system disorders; these conditions may be poorly tolerated in brain injured patients due to cerebral edema from the primary insult and impaired adaptive mechanisms (Wright, 2012).

Common pathologies resulting in hyponatremia include Acute Brain Injuries (ABIs), especially Subarachnoid Hemorrhage (SAH) (Sherlock *et al.*, 2006) and Traumatic Brain Injury (TBI) (Rabinstein and Wijdicks, 2003). In addition, hyponatremia is associated with significant morbidity and mortality, especially if corrected too fast or too slowly (Diringer and Zazulia, 2006).

It is hypothesized that, hyponatremia measured at admission can be used as an indicator or predictor for fatal outcome associated with head injuries. Thus, the present study designed to test if hyponatremia can be used as a predictor for fatal outcome in those patients.

MATERIALS AND METHODS

Between January, 1st 2012 and January, 1st 2013, thirty patients who presented with acute head injury were included. They were selected from Al-Azhar University hospitals. They were 22 males and 8 females; their average age was 33.50 years.

Another 30 apparently healthy subjects matched for age and gender were included as a control group (they were 19 males and 11 females and their average age was 33.40 years). They all had normal heart, liver, kidney and thyroid functions and normal blood pressure.

Inclusion criteria included the following: (1) Patients hospitalized within 24 h after head injury without shock, (2) The diagnosis confirmed by skull CT scan, (3) Patients had no history of drinking alcohol or abusing any drugs before the injury, (4) Patients had no hypertension or other heart diseases, (5) Patients had normal function of other organs (e.g., liver, kidneys, thyroid and adrenal gland) and (6) They had not received glucocorticoid hormone after injury.

The case group was subdivided into two categories based on Glasgow Coma Scale (GCS) score (determined at admission): The slight to medium injury group ((GCS 9-15), the severe injury group contained (3-8).

Both serum and urine concentration of sodium were done by emission flame spectrophotometry. It is simple, rapid and accurate. It was done using Beckman flame spectrophotometer according to method described by Gilbert *et al.* (1950). The basic principle of the method can be summarized as the following: A diluted sample of the biological material (e.g., serum, urine) was dispersed in the mixture of gas flame (butane, propane) and air. The energy emitted by excited particles in the flame passes through a filter and transforms to photocurrent in a photoelectric cell. The emitted light intensity is proportional to the strength of photocurrent, i.e., sodium concentration.

Statistical analysis of data: Collected data were organized, tabulated and statistically analyzed using Statistical Package for Social Science (SPSS) version 16 (SPSS Inc, USA), running on IBM compatible computer. Quantitative data were represented as Mean $\pm$ SD (standard deviation) while categorical data were represented as relative frequency and percent. Comparison between both groups was conducted using student (t) test for quantitative data and Chi square test for categorical variables. To determine predictive power, simple linear regression was conducted. p-value<0.05 was considered significant.

RESULTS

Table 1 revealed that, there was no significant difference between study and control groups as regard gender or age; males were more predominant than females. On the other hand, there was significant decrease of sodium concentration in blood in study group in comparison to control group (135.73 $\pm$ 4.18 vs. 140.76 $\pm$ 2.06, respectively) while there was significant increase of sodium concentration in urine in study group in comparison to control group (304.66 $\pm$ 6.78 vs. 285.20 $\pm$ 4.18, respectively).

Table 1: Comparison between cases and controls as regard gender, age and sodium concentration in blood and urine

Parameters	Study	Control	Test	P
Gender (male/Female)	22/8	19/11	0.69	0.41(NS)
Age (years) (mean±SD)	33.50±14.45	33.40±13.58	0.03	0.97(NS)
Sodium concentration in blood (mEq L ⁻¹) (Mean±SD)	135.73±4.18	140.76±2.06	5.90	<0.001*
Sodium concentration in urine (mEq L ⁻¹) (Mean±SD)	304.66±6.78	285.20±4.18	13.36	<0.001*

NS: non-significant, *Significant, SD: Standard deviation, mEq L⁻¹: Milliequivalent /litre

Table 2: Relation between blood and urine concentration of sodium and mortality rate in relation to severity (GCS) in study group

Parameters	GCS		Statistics	
	>8 (17 cases)	= 8 (13 cases)	Test	p
Sodium concentration in blood (mEq L ⁻¹) (Mean±SD)	140.07±2.21	132.41±1.03	12.70	<0.001*
Sodium concentration in urine (mEq L ⁻¹) (Mean±SD)	308.76±25.03	343.23±21.42	4.06	<0.001*
Mortality	1(7.7%)	3(17.6%)	0.63	0.42(NS)

GCS: Glasgow coma scale, NS: Non-significant, *Significant; SD: Standard deviation, mEq L⁻¹: Milliequivalent/litre

Table 3: Relation between blood and urine concentration of sodium and mortality in study group

Parameters	Died (4 cases)	Live (26 cases)	t	p
Sodium concentration in blood (mEq L ⁻¹) (Mean±SD)	132.75±2.21	136.19±4.25	1.56	0.13(NS)
Sodium concentration in urine (mEq L ⁻¹) (Mean±SD)	376.25±4.78	320.92±22.74	4.78	<0.001*
GCS	5.50±2.64	9.34±3.09	2.34	0.026*

GCS: Glasgow coma scale, NS: Non-significant, *Significant, SD: Standard deviation, mEq L⁻¹: Milliequivalent /litre

Table 4: Simple linear regression of blood and urine concentration of sodium and GCS to predict mortality

Parameters	Death			
	r	R square	Adjusted R square	p
Sodium concentration in blood	0.28	0.08	0.048	0.13(NS)
Sodium concentration in urine	0.67	0.45	0.43	<0.001*
GCS	0.41	0.16	0.13	0.026*

GCS: Glasgow coma scale, NS: Non-significant, *Significant

Table 2 showed that, there was significant decrease of sodium concentration in blood in cases with GCS = 8 in comparison to those cases with GCS>8 (132.41±1.03 vs. 140.07±2.21, respectively); while there was significant increase of sodium concentration in urine in cases with GCS = 8 in comparison to those cases with GCS>8 (343.23±21.42 vs 308.76±25.03 respectively). In addition, there was non-significant increase of mortality rate in cases with GCS = 8 in comparison to those cases with GCS>8 (17.6% vs. 7.7%, respectively).

Table 3 revealed that, there was non-significant decrease of sodium concentration in blood in died cases when compared to live cases (132.75±2.21 vs. 136.19±4.25, respectively); while there was significant increase of sodium concentration in urine in died cases in comparison to live cases (376.25±4.78 vs. 320.92±22.74, respectively). In addition, there was significant decrease of GCS in died cases when compared to live cases (5.50±2.64 vs. 9.34±3.09, respectively).

Table 4 showed that, sodium concentrations in blood had mild predictive power for mortality while sodium concentration in urine had moderate, significant predicting power for mortality and similarly, GCS had a moderate significant predicting power for mortality. Interestingly, the sodium concentration in urine had a more powerful predictive power of mortality than GCS.

DISCUSSION

Hyponatremia is considered to be the most important electrolyte disturbance encountered in the neuro-critical care patients (Tisdall *et al.*, 2006), as it is reported to affect up to 38% of intensive care patients (Hoorn *et al.*, 2006) and up to 50 % of patients undergoing neurosurgery (Zada *et al.*, 2007; Upadhyay *et al.*, 2006).

The present study hypothesized that, hyponatremia measured at admission can be used as an indicator or predictor for fatal outcome associated with head injuries. Thus, the present study was designed to test this hypothesis. Results of the present study showed that males were predominant; there was significant decrease of blood sodium concentration and increase of urine sodium concentration in study in comparison to control group. In addition, both blood and urine sodium concentrations correlated with GCS (where cases with lower GCS had lower blood and higher urine sodium concentration when compared to cases with higher GCS). Furthermore, urine sodium had more significant predicting power for mortality in those patients, even better than GCS.

In another study, Zhang *et al.* (2008) included 68 patients (51 male and 17 female), with an age range of 4-60 years and an average age of 27.8 years. Similar findings of both blood and urine osmotic pressures were reported in their work. In addition (Brimioulle *et al.*, 2008) reported that, there was non significant difference between hyponatremic and control subjects as regard demographic characteristics, severity (GCS) or laboratory findings (cortisol, TSH and bilirubin).

An interesting unique finding in the present study is that, urine sodium had more significant predicting power for mortality in hyponatremic patients than GCS. Supporting these findings, it was reported that, Post-traumatic Brain Injury (TBI) hyponatremia is not related to severity of brain insult (i.e., as measured by the Glasgow coma scale) or presence of brain edema (Agha *et al.*, 2004).

Searching for causes of hyponatremia development with acute head injury, it was reported that, the syndrome of inappropriate antidiuretic hormone secretion (SIADH) is the main reasonable cause. In addition to SIADH, other causes of hyponatremia after head injury include Cerebral Salt Wasting (CSW), glucocorticoid deficiency, medications and aggressive use of intravenous fluids (Agha *et al.*, 2005; Palmer, 2003).

In short, the present study confirmed that, hyponatremia and urine hypernatremia are associated with acute head injury. In addition, the present study is unique in revealing that, urine sodium concentration is a good predictor for mortality from such injuries, even better than Glasgow coma scale. Further studies on a large number of patients are needed to confirm this finding.

REFERENCES

- Agha, A., E. Thornton, P. O'Kelly, W. Tormey, J. Phillips and C.J. Thompson, 2004. Posterior pituitary dysfunction after traumatic brain injury. *J. Clin. Endocrinol. Metab.*, 89: 5987-5992.
- Agha, A., M. Sherlock and C.J. Thompson, 2005. Post-traumatic hyponatraemia due to acute hypopituitarism. *QJM*, 98: 463-464.
- Bhardwaj, A. and J.A. Ulatowski, 2004. Hypertonic saline solutions in brain injury. *Curr. Opin. Crit Care*, 10: 126-131.
- Bhardwaj, A., 2006. Neurological impact of vasopressin dysregulation and hyponatremia. *Ann. Neurol.*, 59: 229-236.
- Brimioulle, S., C. Orellana-Jimenez, A. Aminian and J.L. Vincent, 2008. Hyponatremia in neurological patients: Cerebral salt wasting versus inappropriate antidiuretic hormone secretion. *Intensive Care Med.*, 34: 125-131.

- Diringer, M.N. and A.R. Zazulia, 2006. Hyponatremia in neurologic patients: Consequences and approaches to treatment. *Neurologist*, 12: 117-126.
- Gilbert, P.T., R.C. Hawes and A.O. Beckman, 1950. Beckman flame spectrophotometer. *Anal. Chem.*, 22: 772-780.
- Hoorn, E.J., J. Lindemans and R. Zietse, 2006. Development of severe hyponatraemia in hospitalized patients: Treatment-related risk factors and inadequate management. *Nephrol. Dial. Transplantation*, 21: 70-76.
- Palmer, B.F., 2003. Hyponatremia in patients with central nervous system disease: SIADH versus CSW. *Trends Endocrinol. Metab.*, 14: 182-187.
- Rabinstein, A.A. and E.F. Wijdicks, 2003. Hyponatremia in critically ill neurological patients. *Neurologist*, 9: 290-300.
- Roca-Ribas, F., J.E. Ninno, A. Gasperin, M. Lucas and C. Llubia, 2002. Cerebral salt wasting syndrome as a postoperative complication after surgical resection of acoustic neuroma. *Otol. Neurotol.*, 23: 992-995.
- Sherlock, M., E. O'Sullivan, A. Agha, L.A. Behan and D. Rawluk *et al.*, 2006. The incidence and pathophysiology of hyponatraemia after subarachnoid haemorrhage. *Clin. Endocrinol.*, 64: 250-254.
- Tisdall, M., M. Crocker, J. Watkiss and M. Smith, 2006. Disturbances of sodium in critically ill adult neurologic patients: A clinical review. *J. Neurosurg. Anesthesiol.*, 18: 57-63.
- Upadhyay, A., B.L. Jaber and N.E. Madias, 2006. Incidence and prevalence of hyponatremia. *Am. J. Med.*, 119: S30-S35.
- Wright, W.L., 2012. Sodium and fluid management in acute brain injury. *Curr. Neurol. Neurosci. Rep.*, 12: 466-473.
- Zada, G., C.Y. Liu, D. Fishback, P.A. Singer and M.H. Weiss, 2007. Recognition and management of delayed hyponatremia following transsphenoidal pituitary surgery. *J. Neurosurg.*, 106: 66-71.
- Zhang, W., S. Li, M. Visocchi, J. Jiang and X. Wang, 2008. Natriuretic peptides, antidiuretic hormone and hyponatraemia after acute craniocerebral injury. *J. Int. Med. Res.*, 36: 648-655.