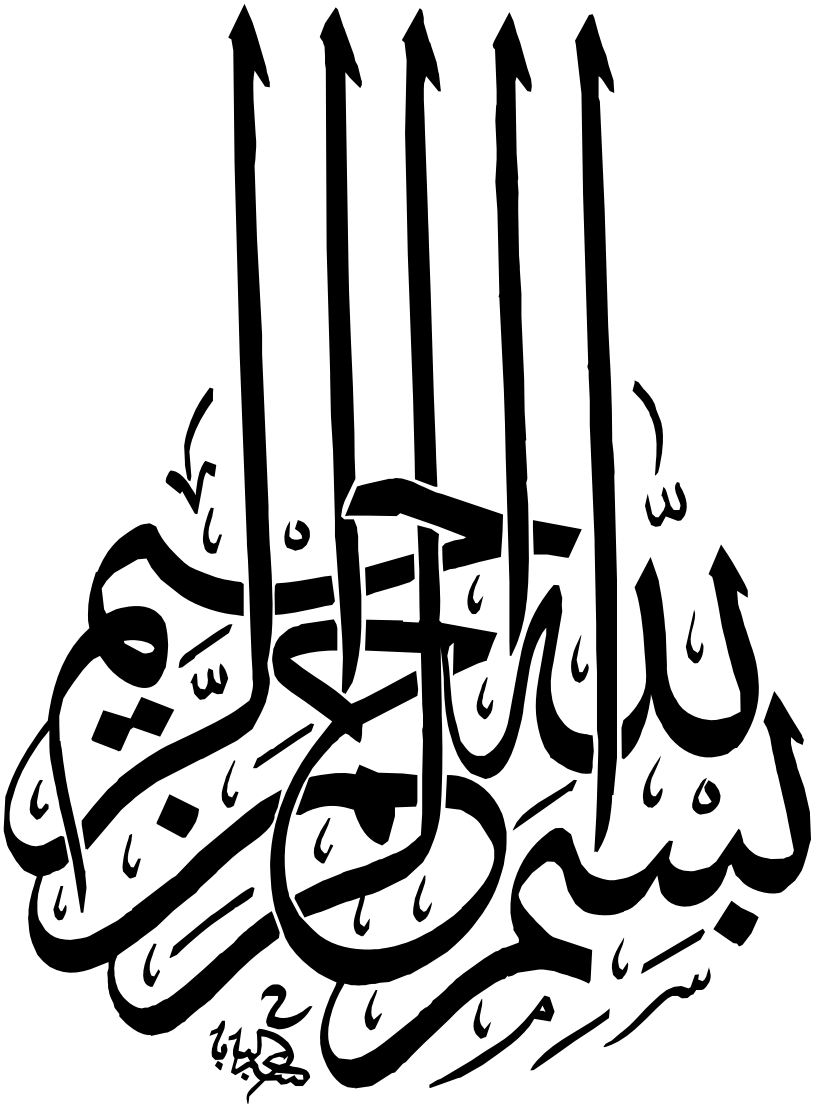
بسم الله الرحمن الرحيم

Prevalence And Prognostic Value Of Sodium Disturbances In Neurocritically-Ill Patients

Thesis Submitted by

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معدل اضطراب الصوديوم بالدم و تأثيره فى مرضى الأعصاب فى الحالات الحرجة

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Abstract

Introduction: **S**odium disturbances and water balance disorders are frequently encountered in critically ill patients and have been associated with an increased risk of death and complications in recent retrospective and prospective studies in neurocritical intensive care units.

Aim of Work: The study was conducted on 50 neuro-critically ill patients who were admitted to Kasr El Eini hospital after traumatic brain injury or subarchnoid hemorrhage or other involvements of neurological events.

Patients & Methods: The studied population was divided into 3 groups according to plasma sodium level: Group A with normal plasma sodium level 135-145 mEq/L. Group B with low plasma sodium level < 135 mEq/L Group C with high plasma sodium level > 145 mEq/L.

Results: There were 35 patients with different traumatic head injury, 7 patients with subarachnoid hemorrhage, 4 with non hemorrhagic cerebrovascular stroke (CVS) and 4 patients with intracerebral hemorrhage. Sixteen (32%) patients were eunatremic, 22 (44%) were hyponatremic and 12 (24%) were hypernatremic. The duration of ICU stay was statistically longer in group C (hypernatremia) than in group A and B, 27.1±7.9 versus 20.8±7.3 and 20.7±7.8 days, respectively and p value was 0.05. There was also a positive significant correlation with duration of ICU stay and sodium level and the correlation factor was 0.4, P value 0.02. Regarding the need for mechanical ventilation, there were higher need for mechanical ventilation in group C (hypernatremic) than group B (hyponatremic) as (11/22, 50%, in group B versus 11/12, 91.6%, in group C were mechanically ventilated) and the p value was 0.02. Also, there were longer duration of mechanical ventilation in the group of hypernatremia than the other 2 groups. There were higher incidence for mortality in group of hypernatremia 8/12 (66.6%) versus 3/22 (13.6%) and the p value was 0.005.

Conclusion: We concluded that Hyponatremia is more frequently occurring in neurocritically ill patients than hypernatremia. Hypernatremia has a more deleterious effects on ICU outcome than hyponatremia in neurocritically ill patients. Hypernatremia is an independent factor that prolongs duration of mechanical ventilation and ICU stay in neurocritically ill patients. Hypernatremia was found to increase risk for mortality independently in neurocritically ill patients and Further prospective, large scale randomized studies are needed for more accurate evaluation of hypernatremia effects on outcome.

Acknowledgement

For **ALLAH** the merciful, the compassionate, I kneel to express my gratitude for all the countless gifts I have been offered, including those who gave their hands to enable me to fulfill this work.

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Dr. Salah M. Dosoki

Contents

List of Abbreviations

ANP	:	Atrial Natriuretic peptides.
BNP	:	Brain Natriuretic peptides.
ADH	:	Ant. Diuretic hormone
SIADH	:	Syndrome of inappropriate ADH secretes
CSWS	:	Cerebral salt wasting syndrome
DI	:	Diabetes insipidus
ECG	:	Electrocardiogram
CBC	:	Complete blood count
PTT	:	Partial thromboplastine time
PC	:	Prothrombin concentration
SD	:	Standard deviation
SPSS	:	Statistical package for the special science
K	:	Potassium
TLC	:	Total leacocytic count
Bun	:	Blood urea nitrogen
NICU	:	Neuro intensive care unit
TBI	:	Traumatic brain injury
CVS	:	Cerebrovascular stroke
SAH	:	Subarchnoid haemorrhage.
GCS	:	Glusgow coma score

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Introduction

Disorers of sodium and water balances are common in neurocritically ill patients ***(Levin Er, 1998)***.

Regulation of salt and water balance:

Sodium is the major extracellular cation and one of the most important osmotically active solutes.

The extracellular to intracellular fluid sodium concentration gradient is maintained by the sodium-potassium ATP ase pump and total body sodium is controlled by renal excretion. Sodium is freely filtered at the glomerulus and the majority is reabsorbed at the proximal tubule under the control of sympathetic nerves and atrial (ANP) and brain (BNP) natriuretic peptides.

The latter causes natriuresis by inhibition of sodium transport in the meduallary collecting duct, by increasing glomerular filtration rate, and by inhibiting rennin and aldosterone release ***(Levin Er, 1998)***.

Because sodium concentration is a major determinant of serum osmolality, it is largely for the normal regulation and distribution of total body water.

In essence, total body water is controlled by renal manipulation of sodium with resulting water adjustment to maintain tonicity.

Sodium and water balances are regulated primarily by serum osmolality but also by intravascular volume and pressure.

An increase in extracellular fluid osmolality is detected by osmoreceptors in the hypothalamus that stimulates synthesis of Arginine vasopressin or antidiuretic hormone (ADH).

ADH is then transported to, and released from the posterior lobe of the pituitary gland. This results in reabsorption of water in the distal tubule and collection duct the kidney.

ADH is also released in reasponse to decrease in arterial pressre and intravascular volume that are detected by low pressure baroreceptors in the right atrium and great veins and high pressure baroreceptors in the carotid sinus.

Hypovolaemia and hypotension also result in increased sympathetic activation of the rennin-angiotensin-aldosterone system ***(Rabinstein AA, 2003)*.**

Hyponatomia: is defined as a serum sodium concentration of < 135 mmol liter and occurs in up to 15% of the general adult hospitalized population. It is more common after brain injury, especially in those patients

who are critically ill and is associated with mortality increase of up 60% ***(Tisdall M, 2006).***

* Causes:

Introgenic hyponatraemia is not uncommon and usually results from the administration of inappropriately hypotonic fluids.

However, after brain injury, hyponatraemia occurs most frequently because of the syndrome of inappropriate ADH seceretion **(SIADH)** or the cerebral salt wasting syndrome **(CSWS)**, ***(Tisdall M, 2006).***

* Complication of hyponatraemia in brain:

The most dangerous complication of hyponatraemia is life threatening metabolic encephalopathy that is often associated with cerebal edema, increased intracranial pressure and seizures.

Correction of the hyponatraemia can also be associated with an encephalopathy that is characterized by demyelinating lesions, pituitary damage and oculomotor nerves paralysis. This is usually seen when sodium concentration is corrected too rapidly. Specific demyelinating disorder know as central pontine myelinolysis that can be irreversible and even fatal ***(Diringer MN, 2006).***

Hypernatraemia:

Hypernatraemia defined as a serum sodium of > 145 mmol/litre, occurs less commonly than hyponatraemia and its incidence is 1% in the general inpatient hospital population and 9% in the intensive care setting. It is more common in brain-injured patients and is often an indicator of the severity of the underlying disease ***(Tisdall M, 2006).***

* Causes:

Hypernatraemia is usually related to inadequate free water intake or excess water loss.

After brain injury, hypernatraemia, is commonly related to the development of central diabetes insipidus **(DI)** or the overzealous use of osmotic diuretics such as mannitol.

Iatrogenic causes are relatively easy to recognize and manage and usually respond to normalization of sodium intake ***(Powner DJ, 2006).***

Complication of hypernatraemia in the brain:

Hypovolemic hypernatraemia leads to hypertoncity. Hypertoncity of extra cellular fluids predisposes to cellular dehydration which leads to metabolic encephalopathy ***(Powner DJ, 2006).***

Clinical finding include depressed consciousness that can progress to frank coma, generalized seizures, and focal neurogical deficits,

hypernatremic, encephalopathy has been associated with mortality up to 50% ***(Powner DJ, 2006).***