

Serum and urinary electrolyte levels in Cerebro-Vascular Accident patients: A cross sectional study

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Abstract: This cross sectional study was designed to document sequential changes in serum as well as urinary sodium and potassium levels in cerebrovascular accident (CVA) patients, in relation to a control group, in an attempt to precise the role of these electrolytes in occurrence of CVA.

Keywords: Cerebrovascular Accident, Serum Electrolyte, Urinary Electrolyte

1. Introduction

Stroke or cerebrovascular accident is a state of non-convulsive, focal neurological deficit of abrupt onset usually caused by ischemia infarction or hemorrhage in the brain¹. It is the third leading cause of death and is the most disabling of all neurological diseases. Nearly one third of stroke patient die within 3 weeks and 48% die within one year¹. High mortality in stroke is due to some complications like cerebral edema and brainstem herniation; infection, electrolyte imbalance and associated heart disease and metabolic disorder. Hypo and hypernatremia are reported to be the most common electrolyte abnormality in hospitalized sick adults. The pathophysiological implications and outcome of these are uncertain. Some authors consider sodium imbalance to be of little significance²⁻⁵—whereas some others are of the opinion that high morbidity and mortality of patients are related to these electrolyte imbalance⁶⁻⁸.

Patients with intracerebral disorders are prone to develop a state of hypernatremia and are unable to prevent loss of

sodium in urine⁹⁻¹¹. The hypernatremia with excretion of hyperosmolar urine occurs. Excessive salt excretion in urine occurs as a result of insult to CNS¹². But the interpretation and significance of the condition is beset with controversy and confusion^{2, 3, 13, 14}.

Mild hypo or hypernatremia may be auto reversible—but when it becomes severe and develops all on a sudden—it itself can cause death of a patient. Further- convulsion due to hyponatremia may aggravate the intracerebral edema in a stroke patient to worsen the situation. In a state of hyponatremia- hypoxia causes further damage to the brain in a stroke patient.

This study is therefore designed to document the sequential changes in serum sodium and potassium levels in stroke patients as well as the urinary excretion of these electrolytes.

2. Methods

After approval from institutional ethical and written informed consent of the patients, this study was conducted

in a tertiary care medical college hospital in eastern India. 50 cases of cerebrovascular accident (C.V.A.) diagnosed clinically and subsequently confirmed by C.T. Scan of brain, were registered for study. However, patient presenting with preexisting edema, on diuretic therapy, on intra venous fluid therapy, renal failure- are excluded from study. Subsequently patients with hyperglycemia and hyperlipidemia are also excluded to rule out possibilities of misinterpretation of serum sodium level and to exclude pseudo hyponatremia. Patients with T.I.A. and S.A.H. were also excluded from study. 50 age and sex matched healthy controls were subjected to detailed serum and urine electrolyte estimation –who were admitted with some other ailment other than C.V.A. and not suffering from conditions known to produce hyponatremia or hypernatremia. Each patient was subjected to thorough history and detailed examinations with special emphasis on central nervous system. In drowsy or disoriented patients the history was obtained from accompanying relatives. Features of hyponatremia i.e. anorexia, vomiting in mild hyponatremia and restlessness, irritability, confusion, convulsion and coma (in profound hyponatremia) were carefully noted keeping the fact in mind that all these signs and symptoms may occur with stroke per se.

Apart from routine investigations, Blood and Urine samples for electrolytes (Na^+/K^+) were examined on immediate post admission day.

Urinary electrolytes were examined from a sample of 24 hours collected specimen. Other parameters of significant values were repeated as and when necessary.

Serum and Urine- sodium and potassium were measured by Flame Photometry

3. Statistical Analysis

Data was expressed as mean and standard deviation. The homogeneity in case and control groups of mean and SD was analyzed using SPSS version 16.0 software. Comparisons of serum sodium and potassium concentrations among three patient groups and the control group were analyzed by one-way analysis of variance (ANOVA). The differences between continuous variables among case and control groups were analyzed by independent t test. Categorical data was compared using Chi-square test. A p value of less than or equal to 0.05 was considered as statistically 'significant'.

4. Results

Present study was conducted on 50 stroke patients of which 10 had cerebral hemorrhage; 38 had cerebral infarction and 2 had combined lesion of thalamic hemorrhage with same sided lacunar infarction.

Table 1. Demographic profile of the patients

Variables	Patients with CVA				Control (n = 50)	P value
	Total (n = 50)	Hemorrhagic (n = 10)	Ischemic (n = 38)	Both (n = 2)		
Sex	Male, n (%)	32 (64%)	8 (16%)	22 (44%)	2 (4%)	30 (60%)
	Female, n (%)	18 (36%)	2 (4%)	16 (32%)	0 (0%)	20 (40%)
Age in years, mean±SD	57.4±10.11	63±7.15	55.35±9.31	58.5±2.12	60.15±8.99	0.1538
Weight in Kg, mean±SD	70.3±13.61	73±6.33	69.05±11.23	71.5±3.54	72.1±14.89	0.5295

CVA: Cerebro Vascular Accident. Data derived from independent t test.

As evidenced from table 1, there was no significant difference ($p>0.05$) between CVA group and control group in respect demographic profile. From post-hoc analysis, it is seen that patients with hemorrhagic CVA were

significantly older than patients with ischemic CVA and control group ($P < 0.001$).

Table 2. Serum electrolytes of the patients

Variables	Patients with CVA			Control (n = 50)	P value
	Hemorrhagic (n = 10)	Ischemic (n = 38)	Both (n = 2)		
Na^+ (meq/l), mean±SD	146±2.61	142.3±3.01	144±1.41	138.1±2.53	<0.001
K^+ (meq/l), mean±SD	3.61±0.21	4.03±0.16	3.75±0.07	4.63±0.19	<0.001

CVA: Cerebro Vascular Accident. Data derived from one-way ANOVA test.

Table 2 comprises of serum electrolyte levels of patients of CVA and control groups. Post hoc analysis revealed that serum sodium level of control group was significantly lower ($p<0.001$) than CVA group whereas serum potassium

level was significantly higher ($p<0.001$) in control group. Patients with hemorrhagic CVA were found having significant higher sodium and lower potassium levels ($p<0.001$).

Table 3. 24 hours urinary electrolytes of the patients

Variables	Patients with CVA			Control (n = 50)	P value
	Hemorrhagic (n = 10)	Ischemic (n = 38)	Both (n = 2)		
Na ⁺ (meq/24 hrs), mean±SD	255.6±7.83	232.1±13.31	233.9±9.4	188.8±10.78	<0.001
K ⁺ (meq/24 hrs), mean±SD	53.2±0.89	61.9±2.76	55.1±3.11	69.7±1.49	<0.001

CVA: Cerebro Vascular Accident. Data derived from one-way ANOVA test.

Table 3 comprises of 24 hours urinary electrolyte levels of patients of CVA and control groups. Here also the post hoc analysis was similar to table 2 with control group having significantly lower sodium and higher potassium and hemorrhagic CVA group having significant higher sodium and lower potassium ($p < 0.001$ in each case).

5. Discussion

There are studies indicating that exchangeable sodium is increased in hypertensive patients which is well corroborated with arterial pressure¹⁵. Again, it has been previously shown that for an actual increase in brain volume to occur, additional fluid must be added to the brain's extracellular space. During permanent ischemia, blood sodium rapidly enters the extracellular fluid of the brain, leading to consequences of brain edema^{16, 17}, therefore we hypothesized that higher serum sodium levels are associated with higher risk and exacerbation of events following brain ischemia. In line with our findings, in a large cohort study, O'Donnell *et al.*, have reported higher urinary sodium excretion, which is a valid reflection of dietary sodium intake, to be associated with a higher risk of different types of CVA. However, this study has also shown that less than 3 g per day sodium excretion is related with higher mortality following cerebrovascular events and longer hospitalization because of congestive heart failure¹⁸. Here, it is to be noted that, normal 24 hours urinary sodium varies with sodium intake and it could be upto 100-260 meq/24-hours²⁶. In the same way, normal 24 hours urinary potassium excretion can vary upto 25-125 meq/24-hours²⁶.

Previous studies also indicate that systolic but not diastolic blood pressure changes in concordance with the 24 hour urinary sodium excretion. However, only baseline 24-hour urinary sodium excretion is inversely associated with cardiovascular mortality¹⁹. Evidence derived from studies such as Trials of Hypertension Prevention Collaborative Research Group indicate the association of a reduction in sodium intake with long-term reduction in CVA, suggesting a protective role of limiting dietary sodium intake in primary prevention of vascular events²⁰. Experimental studies on hypertensive animal models are suggestive of a protective role of potassium intake on vascular events^{21, 22}. Furthermore, Khaw and Barrett-Connor first reported the protective effect of dietary potassium intake on risk of stroke²³. Similarly, O'Donnell

et al., discovered that higher potassium excretion is related with lower risk of CVA¹⁸. It is previously established that high potassium intake causes a mild reduction in blood pressure of hypertensive patients^{24, 25}. There are some limitations that should be considered in generalizing the findings of this study. First, this study has a comparative cross-sectional nature, therefore, no causal relationship can be drawn. Second, in this study we did not assess the role of some variables which may influence serum electrolyte levels such as participants' medical comorbidities, medications, and risk factors. Therefore higher serum sodium or lower serum potassium levels might not independently be associated with CVA.

6. Conclusion

This study shows that higher serum sodium levels and lower serum potassium levels may be associated with higher incidence of CVA. Although, further studies with larger control are needed to establish this.

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