

Study of Serum Sodium and Potassium Levels in Patients with Acute Subarachnoid Haemorrhage

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ABSTRACT

Aim: The aim the study was to evaluate the serum sodium and potassium levels in patients presenting with acute subarachnoid haemorrhage (SAH) as compared to controls.

Materials & Methods: It is a single institutional cross-sectional study which includes 30 patients and 50 healthy controls. Serum Na⁺ and K⁺ levels of patients presenting with acute sub arachnoid haemorrhage (< 2 wks duration) were compared with Na⁺ and K⁺ levels of apparently healthy controls. Data collected were analysed by Microsoft excel software using unpaired t test.

Results: The serum sodium and potassium values in the SAH group were found to be significantly lower than the controls with P values of 0.0034 and < 0.0001 respectively. 40% of SAH patients developed hypokalemia and 26.7% of SAH patients developed hyponatremia. Hypernatremia was seen in 6.6% of patients. None of the patients developed hyperkalemia.

Conclusion: SAH patients are susceptible to develop hyponatremia and hypokalemia which can lead to serious medical complications. Continuous monitoring, early detection of serum electrolytes and prompt correction is essential to decrease the morbidity and duration of hospital stay of these patients.

Keywords: subarachnoid haemorrhage; aneurysm; hyponatremia; hypokalemia

INTRODUCTION

The brain suffers from both primary and secondary insults as a result of acute SAH. The most common cause of spontaneous subarachnoid haemorrhage is rupture of intracranial aneurysm. Other causes of atraumatic SAH are ruptured arteriovenous malformations, dural/pial arteriovenous fistulae, primary intraparenchymal haemorrhage extending into the subarachnoid space. [1]

The incidence of subarachnoid haemorrhage increases with advanced age and is more common in women of > 50 years of age as compared to men. The mortality rate of aneurysmal subarachnoid

haemorrhage who arrive alive at hospital is 45% over the next 1 month. [1,2]

More than 50% of patients who survive after SAH are left with serious neurological deficit. [1] However, the non-neurological complications that occur after SAH have significant effect on the morbidity and prognosis of these patients. The common non-neurological complications associated with SAH patients are pulmonary oedema, cardiac, renal, hepatic dysfunctions, electrolyte disturbances and haematological derangements. [3] These factors have major impact on the outcome and duration of hospital stay of SAH patients. Several

previous studies on electrolyte imbalance of SAH patients have given emphasis on the occurrence of hyponatremia in SAH patients. [4-6,7] However, there are very few studies on the pattern of changes of serum potassium level in SAH patients. In the present retrospective study, we tried to evaluate the pattern of serum sodium and potassium abnormalities in SAH patients in the acute and subacute stage.

MATERIALS AND METHODS

It was a single institutional retrospective study conducted in our tertiary care hospital.

In this present randomised case control study, 30 randomly selected SAH patients attending our hospital from February 2016 to June 2017 were included and the results were compared with 50 apparently healthy controls.

Controls were selected randomly from the apparently healthy individuals who attended the outpatient department for post-employment or pre-employment general health check-up without any specific health issues.

Inclusion criteria:

1. Nontraumatic subarachnoid haemorrhage
2. Traumatic subarachnoid haemorrhage

Exclusion criteria:

1. SAH patients admitted after 2 weeks of bleed were not included in the study.

Diagnosis of SAH was made by CT scanning and the source of bleeding was evaluated by digital subtraction angiography (DSA):

4 Patients presented with post traumatic isolated SAH. In view of definite history of trauma and CT evidence of calvarial fracture, DSA was not done in these patients. DSA was performed in all cases with spontaneous SAH (n=26). Ruptured intracranial aneurysm was found in 18 patients, arteriovenous malformation

in 3 patients, dural arteriovenous fistulae in one patient and 4 cases were angio negative.

Patients' data including past history, clinical details were collected from the hospital medical record department (MRD) retrospectively. Data of serum sodium and potassium values were collected from the departmental log book of Clinical biochemistry department. Investigations done after 14 days from the onset of ictus were not considered.

Estimation of serum sodium and potassium were carried out by ion selective methods using Radiometer ABL 80 Flex by standardised method.

Data collected were analysed by Microsoft excel software using unpaired t test. P value of < 0.05 was considered as statistically significant.

RESULTS

The serum sodium and potassium values in the SAH group (table 1) were found to be significantly lower than the controls with P values of 0.0034 and < 0.0001 respectively.

Table1: comparison of Na⁺ and K⁺ in SAH patients and controls

GROUP	Na ⁺ (mmol/L) Mean ± SD	K ⁺ (mmol/L) Mean ± SD
SAH patients	137.2 ± 4.1	3.7 ± 0.8
Controls	139.3 ± 2.2	4.3 ± 0.3
P value	0.0034	<0.0001

Table2: percentage of cases with deranged sodium and potassium level among the study group

	Total No of cases	Total %
Hyponatremia (Na ⁺ <135 mmol/l)	8	26.7%
Hypokalaemia (K ⁺ <3.5 mmol/l)	12	40%
Hypernatremia (Na ⁺ >145 mmol/l)	2	6.6%
Hyperkalemia (K ⁺ > 5.5 mmol/l)	0	0%

It was observed (Table 2) that SAH patients were prone to develop both hypokalaemia and hyponatremia with occurrence of hypokalaemia (40%) being more as compared to hyponatremia (26.7%). Only 6.6% of patients were hypernatremic with serum Na⁺ value >145mmol/l and none of the patients found to be hyperkalemic among the study group.

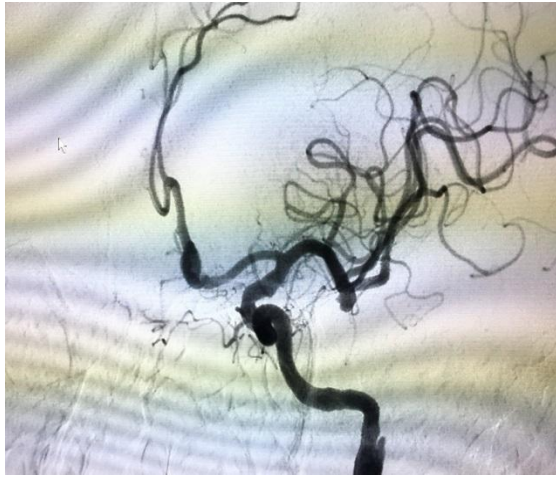


Fig 1: Digital subtraction angiography showing left middle cerebral artery bifurcation saccular aneurysm. Another fusiform aneurysm involving left anterior cerebral artery.



Fig 2: NCCT brain showing diffuse sub arachnoid hemorrhage involving sylvian fissure cisterns, basal cisterns, anterior interhemispheric fissure with dilated temporal horns of lateral ventricles.

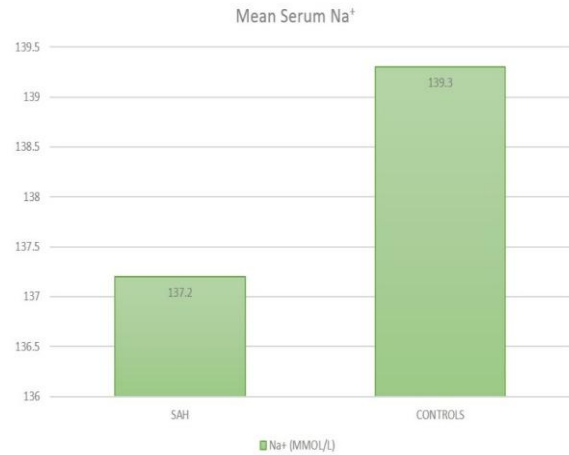


Fig 4: Bar diagram showing serum potassium value in SAH patients and controls in mmol/l

DISCUSSION

In this study it was observed that patients with SAH were prone to develop both hyponatremia and hypokalaemia. The mean serum sodium in SAH patients was lower (137.2 ± 4.1) as compared to the controls (139.3 ± 2.2) with p value 0.002 which is highly significant. The mean serum potassium in SAH patients and controls were 3.7 ± 0.8 and 4.3 ± 0.3 respectively with p value <0.0001 which is statistically highly significant. Although the mean sodium and mean potassium among the study group was within normal limit, the incidence of hyponatremia (<135 mmol/l) and hypokalaemia (<3.5 mmol/l) among the study group were very high, 26.7% and 40% respectively.

Inga Chen et al [8] in their study on 114 SAH patients observed that SAH patients are likely to develop both hyponatremia and hypokalaemia.

Fukui S et al [9] in their study on 133 SAH patients observed low serum potassium level in female SAH patients (3.29 ± 0.47 mEq/L) as compared to male (3.68 ± 0.38 mEq/L).

Alimohamadi M et al [2] in a prospective study on “impact of electrolyte imbalances on the outcome of aneurysmal subarachnoid haemorrhage” observed that hyponatremia was the most common electrolyte disturbances in SAH patients.

Alejandro A. Rabinstein et al [10] in their study of “Management of

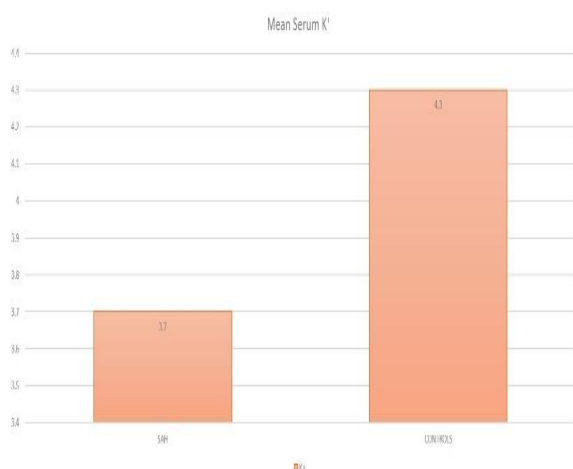


Fig 3: Bar diagram showing serum sodium value in SAH patients and controls in mmol/l

Hyponatremia and Volume Contraction” mentioned that hyponatremia is the most common electrolyte imbalance in 30% - 50% of aneurysmal subarachnoid haemorrhage cases.

Qureshi et al [11] observed in their study that hyponatremia (30%) is more common than hypernatremia (19%) in patients with aneurysmal subarachnoid haemorrhage.

Hyponatremia in SAH:

Hyponatremia is associated with increased morbidity and increases the duration of hospital stay. [4] The possible causes of hyponatremia following SAH are

1. Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
2. Cortisol insufficiency
3. Cerebral salt wasting syndrome
4. Excessive fluid therapy and/or diuretic therapy

SIADH in SAH: The association of SIADH in SAH patients can be due to intracranial haemorrhage and surgical repair of aneurysm which interrupt neuronal communication and hormonal feedback mechanisms. [7] The release of Antidiuretic Hormone (ADH) can also be due to pain, stress, raised intracranial pressure and hypovolemia. [7]

Stimulation of the hypothalamus as a result of traumatic or ischemic brain injuries leads to secretion of excess ADH which enhances the reabsorption of water in the distal convoluted tubule of the kidney causing water retention and dilutional hyponatremia. [4] However, Controversies exist as to which of these mechanisms most commonly cause hyponatremia in SAH patients. [6] Blood volume can help distinguishing SIADH from CSWS, it will be either normal or increased in SIADH whereas, it will be low in CSWS. The distinction between the two is very crucial as the management of the patient in these two conditions is completely opposite. Fluid and sodium restriction is required in SIADH while intake of large quantity of salt is recommended in CSWS. [12]

M. J. Hannon et al in their study mentioned that syndrome of inappropriate antidiuresis (SIAD) is the predominant cause of hyponatremia after SAH. [6] Some of the previous studies suggested that cerebral salt wasting syndrome (CSWS) is the most common cause of hyponatremia after SAH. [6]

Mechanism of hyponatremia in CSWS after SAH: [7] CSWS is a transient phenomenon leading to hyponatremia as a result of inability of kidneys to conserve sodium. Sympathetic nervous system stimulation as a result of brain injury due to SAH causes release of hormones like epinephrine and norepinephrine. These hormones stimulates vasoconstriction which leads to increased systemic blood pressure, inotropy and pressure load. Kidneys respond to these changes by natriuresis causing hyponatremia. Additional mechanism of CSWS causing hyponatremia are release of digoxin-like peptide and atrial and brain natriuretic peptides after SAH.

However, according to the recent studies, elevated plasma BNP are not a reliable predictor of blood volume status and hyponatremia. [6,13]

Hypokalemia in SAH: After SAH, there is release of catecholamines. The circulating catecholamines stimulates Na⁺ - K⁺ ATPase by activation of beta2 adrenergic receptors. As a result, the extracellular K⁺ shifts intracellularly causing hypokalemia. [3] Hypokalemia can lead to ECG changes in the form of QT prolongation and ventricular. [14] Women are at higher risk of developing hypokalemia after SAH. Serum potassium level should be repeated daily and as and when required and corrective measures should be taken promptly to maintain serum potassium level ≥ 3 mmol/l. [14]

All our patients with hyponatremia were detected early and were treated with oral intake of extra salt. Hypertonic saline was used in three patients. Likely causes of hyponatremia were cerebral salt wasting / use of diuretic due to prevent raised intracranial pressure/ cortisol insufficiency.

SIADH was ruled out in all patients. Patients with hypokalemia were treated with oral supplementation of potassium chloride or intra venous infusion of 20 mEq potassium slowly over 30 min with daily monitoring of electrolytes.

The limitation of the study is small number of patients. Hence significance of Na⁺, K⁺ levels in patients presenting with aneurysmal and non-aneurysmal SAH could not be assessed. Study with larger number of patients is necessary to evaluate the difference in serum Na⁺, K⁺ levels in different aetiology of SAH.

CONCLUSION

SAH patients are susceptible to develop electrolyte disturbances both in the form of hyponatremia and hypokalemia that lead to serious medical complications and exert major impact on the prognosis of the patient. Continuous monitoring of serum electrolytes and timely correction to minimise the non-neurological complications would decrease the morbidity and duration of hospital stay of these patients.

Compliance with ethical standards:

Funding: no funding was received for this study.

Conflict of interest: authors of the study declare that there is no conflict of interest.

Informed consent: being a retrospective study, informed consent from the study subjects was not taken.

Ethical approval: since it is a retrospective study, formal consent is not required.

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