**ASSOCIATION BETWEEN SERUM POTASSIUM ION ABNORMALITIES AND CLINICO-RADIOLOGIC PARAMETERS IN SEVERE TRAUMATIC BRAIN INJURY**

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**ABSTRACT**

**Background:** Severe traumatic brain injury is a major cause of death and disability worldwide. Inflammatory and biochemical cascades triggered by primary brain injury may lead to electrolyte dysfunction that contributes to secondary brain injury. The objective of this study was to determine the incidence of serum potassium ion abnormalities and their association with specific clinical and radiological parameters. **Methodology:** This was a prospective cross-sectional study of 117 patients with severe head injury admitted between November 2019 and February 2020. Data collected included the demographics of patients, injury mechanisms, pre-hospital interventions, clinical examination findings, CT Scan head findings, serum potassium ion levels (at admission and 48 hours later) and the outcome (30 days). The data collected was entered in the Social Sciences Statistical Package for analysis. **Results:** Hypokalemia was noted in 25(21.4%) and 21(23.9%) of cases at admission and 48hrs post-admission respectively. Hyperkalemia was noted in 8(6.8%) cases at admission and in 6(6.8%) patients 48hrs post-admission. Hypokalemia was associated with the presence of epidural hematoma (p=0.005) while increased pulse rate (p=0.007) and traumatic SAH (p=0.045) were mainly seen in patients with hyperkalemia. Hypokalemia was associated with significantly increased risk of mortality OR 4.12(p=0.031) and OR 5.12 (p=0.039) at admission and 48hrs post-admission respectively. **Conclusion:** Hypokalemia is the commonest potassium ion abnormality seen in severe head injury, and is associated with significant increased risk of mortality.

**Key words:** Traumatic brain injury, hyponatremia, hypernatremia

**INTRODUCTION**

Serum potassium abnormalities are common following trauma 1. Hypokalemia and hyperkalemia can occur and are associated with poor outcomes. Post-traumatic hyperkalemia, defined as potassium level >5 mmol/l, is caused by extensive tissue damage, aggressive transfusion and hemorrhagic shock 1. Hypokalemia which is defined as potassium level <3.5mmol/l is postulated to arise from intracellular shift of potassium ions caused by epinephrine released during stress response to trauma 2–4. Both hypokalemia and hyperkalemia are associated with life-threatening cardiac arrhythmias which may lead to death 1,5. There is paucity of local data on the incidence of potassium ion abnormalities following traumatic brain injury.

Severe Traumatic brain injury (TBI), defined as Glasgow Coma Scale ≤ 8, is a major cause of death and disability worldwide and is associated with huge direct and indirect costs to the public 6–8. Traumatic Brain Injury (TBI) is more in developing nations because of the increasing number of road traffic accidents 9–11. In our setup, most hospital-based studies have revealed that severe head injury is associated with mortality of >50% and poor functional outcomes 12–14. These bad outcomes may be associated with secondary brain insults such as electrolyte abnormalities that arise from inflammatory and biochemical cascades initiated by the primary injury insult to the brain 7,15. This study aimed at determining the incidence of serum potasium ion abnormalities in severe TBI patients, and their association with clinico-radiologic parameters and 30-day outcome.

**MATERIALS AND METHODS**

***Study design and site:*** An analytical cross-sectional study conducted over 4 months (1st November 2019 to 28th February 2020). The study site was the Kenyatta National Hospital Accident and Emergency Unit and Intensive Care Unit. Kenyatta National Hospital is located in Nairobi7, Kenya, and is the largest hospital and the country's leading neurotrauma referral center. Hospital serves patients from various regions and socioeconomic backgrounds.

***Study population:*** One hundred and seventeen patients with severe head injury defined by Glasgow Coma Scale ≤ 8 and whose next of kin had given informed consent were recruited into the study. Study excluded patients with known pre-existing chronic disease.

***Study variables:*** Data collected included demographics of patients, injury mechanisms, pre-hospital interventions, clinical examination findings, CT Scan head findings, serum potassium levels (at admission and 48 hours later) and outcome (30 days). The Injury Severity Score (ISS) was used to measure the severity of the injury to the patient 16. Serum potassium tests were performed using Biolis 50i Superior Chemistry Analyzer (Tokyo Boeki Medisys – Japan). Daily internal quality control checks were carried out to ensure that the results were valid. In addition, external quality control checks were conducted through the Randox International Quality Assessment Scheme (RIQAS). The reference range for serum potassium in our laboratory is 3.5-5.0 mmol/L.

***Statistical analysis:*** The data collected was entered in the Statistical Package for Social Sciences (SPSS) version 20.0 for analysis. Variables were tested for normal distribution using the Kolmogorov-Smirnov test in addition to histograms. If normality assumptions were violated, the Mann-Whitney U and Kruskal-Wallis tests were performed to test for differences between groups, instead of the student's t-test and the ANOVA (Analysis of Variance) tests. Admission and 48hrs post-admission variables were compared using a paired t-test. Categorical data were analyzed by Pearson's Chi-square test. The correlation between serum potassium and study variables (clinical, radiological) was determined using Pearson's correlation coefficient. The odds ratio was calculated for each electrolyte abnormality to determine its associated mortality risk (30-day mortality). A p-value of < 0.05 was considered to be significant.

***Ethical considerations:*** The study was carried out in accordance with the principles of the Helsinki Declaration. The protocol of the study was reviewed and approved by the Kenyatta National Hospital-University of Nairobi Ethics and Research Committee (P723/08/2019). Written informed consent was obtained from the next of kin of the patients as the patients could not consent in view of their low Glasgow Coma Score

**RESULTS**

**General information**

The study recruited 117 patients out of whom 111 (94.9 %) were male. The average age was 32.41 ± 14.59 years. Pre-hospital administration of intravenous normal saline and mannitol solutions was reported in 65 (55.6 %) and 16 (13.7%) of patients, respectively. The mean Glasgow Coma Score and Injury Severity Scores at admission were 6.41±1.69 and 21.06±7.74.

**Incidence of serum potassium ion abnormalities**

The mean serum potassium ion level was 3.936±0.63mmol / l (n=117) and 3.99±0.81mmol / l (n=88) at admission and 48 hours after admission. Paired T-test did not reveal any statistically significant differences between the potassium levels at admission and those at 48 hours post-admission (p=0.461). The predominant abnormality was hypokalemia, which was reported in 25 (21.4%) and 21 (23.9%) of admission and 48 hours post-admission tests, respectively (Figure 1). Hyperkalemia was reported in 8 (6.8%) cases at admission and in 6 (6.8%) patients 48 hours after admission.

**Association between serum potassium and clinical parameters**

The only parameter that displayed a statistically significant difference among the three groups, as well as significant correlation with admission serum potassium levels, was the heart rate (Table 1 and Table 2).

**Association between serum potassium & radiologic parameters**

Epidural hematoma was mostly associated with hypokalemia at admission (p=0.005), while traumatic SAH was primarily seen in patients with hyperkalemia (p=0.045) 48 hours after admission (Table 3). These variables showed statistically significant correlations with 48hr post-admission potassium levels (Table 4). Other radiological parameters did not exhibit association with serum potassium ion levels.

**Association between serum potassium & 30-day mortality**

Hypokalemia was associated with significantly increased risk of mortality OR 4.12(95% CI: 1.14-14.83, p=0.031) and OR 5.12 (95 % CI: 1.08-24.25, p=0.039) at admission and 48hrs post admission respectively. Although hyperkalemia was also associated with an increased risk of mortality, the risks were not statistically significant OR 0.34 (95 % CI: 0.06-1.50, p=0.154) and OR 2.20 (95 % CI: 0.41-11.64, p=0.36) at admission and 48 hours post-admission, respectively.

**DISCUSSION**

The most common serum potassium abnormality reported in the current study was hypokalemia, seen in 21.4% and 23.9% of admission and 48hrs post-admission respectively. Previous studies have also reported that hypokalemia is the most common potassium abnormality in patients with head injury, with an incidence of 21.5-68.3%.17–19. The main mechanism of post-traumatic hypokalemia is postulated to result from potassium shifts into the intracellular compartment due to epinephrine surge caused by injury 1. The post-traumatic surge in catecholamines leads to β-2-adrenergic receptor stimulation and activation of the sodium-potassium pump, causing an intracellular K+ shift 20. Studies have also shown that, following head injury, there is an increase in cardiac output, blood pressure, pulse rate and pulmonary shunt, along with decreased or normal systemic and pulmonary vascular resistance, consistent with increased circulating catecholamines. 21. This may explain the observation in the current study that admission heart rate had a statistically significant correlation with serum potassium levels.

Although not observed in the present study, a previous study demonstrated a strong correlation between [K+] and GCS. In that study of 46 patients, the degree of hypokalaemia was found to be proportional to the fall in GCS 22. Serum catecholamine concentration is related to ICP in the setting of brain injury and the relationship between the two is dynamically dependent on ICP 23. Insulin, known to cause hypokalaemia, is not elevated in patients with a traumatic brain injury 2. Hypokalemia is associated with life-threatening cardiac arrhythmias and is a major risk factor for death in TBI patients 1,5. In the present study, hypokalemia was associated with 4 fold and 5 fold increased risk of mortality at admission and 48hrs post admission respectively.

Hyperkalemia in the present study was reported in 6.8% of the cases at admission and 48hrs post-admission. Previous studies have reported a prevalence rate of 0.9-17.7% 17–19. Post-traumatic hyperkalemia is induced by extensive tissue damage and aggressive transfusion 1. Additionally, prolonged hemorrhagic shock theoretically leads to hyperkalemia due to alterations in the cellular membrane function. Other causes of hyperkalemia in TBI include mannitol and anaesthetic agents such as succinylcholine and barbiturates 20,24–27. Although hyperkalemia is rare in TBI, it should be timely managed as it is also associated with life-threatening cardiac arrhythmias which may lead to death 1,5.

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**Figure 1:** Serum potassium levels at admission and 48hrs post admission

**Table 1: Association between serum potassium and clinical parameters**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Variable** |  | **Hypokalemia** | **Normal** | **Hyperkalemia** | **P value** |
| Age (yrs) | *Admission* | 37.5±10.2 | 31.0±15.4 | 31.3±15.7 | 0.168 |
| *48hrs post admission* | 35.0±16.4 | 33.9±13.5 | 24.8±14.7 | 0.353 |
| Time from injury to presentation (hrs) | *Admission* | 18.1±41.1 | 23.9±46.6 | 34.1±34.8 | 0.664 |
| *48hrs post admission* | 31.2±47.1 | 23.4±38.3 | 9.3±7.1 | 0.469 |
| Pre-hospital use of IV fluids | *Admission* | 42.3% | 60.0% | 25.0% | 0.066 |
| *48hrs post admission* | 61.9% | 57.4% | 66.7% | 0.871 |
| Pre-hospital use of Mannitol | *Admission* | 7.7% | 15.6% |  | 0.305 |
| *48hrs post admission* | 19.0% | 13.1% |  | 0.485 |
| Systolic BP  (mmHg) | *Admission* | 131.4±27.2 | 126.3±25.5 | 126.8±13.1 | 0.657 |
| *48hrs post admission* | 135.5±26.8 | 129.2±22.7 | 116.3±7.3 | 0.194 |
| Diastolic BP (mmHg) | *Admission* | 78.3±19.1 | 75.5±18.0 | 72.4±14.2 | 0.662 |
| *48hrs post admission* | 76.3±14.3 | 76.4±17.2 | 66.0±11.1 | 0.323 |
| Heart rate  (/min) | *Admission* | 80.8±19.9 | 98.4±25.4 | 100.1±30.1 | ***0.007\**** |
| *48hrs post admission* | 91.9±30.5 | 93.4±24.0 | 83.2±23.6 | 0.644 |
| Respiratory rate (/min) | *Admission* | 19.5±3.9 | 21.3±4.9 | 20.5±3.7 | 0.257 |
| *48hrs post admission* | 19.5±2.6 | 20.4±4.1 | 21.5±4.0 | 0.513 |
| Saturation O2 | *Admission* | 92.3±7.8 | 90.6±10.3 | 93.0±6.1 | 0.698 |
| *48hrs post admission* | 90.7±8.8 | 92.8±7.1 | 93.3±6.5 | 0.577 |
| Total GCS Score | *Admission* | 6.0±1.7 | 6.4±1.7 | 7.3±1.0 | 0.197 |
| *48hrs post admission* | 5.9±1.8 | 6.6±1.5 | 6.7±2.0 | 0.180 |
| ISS Score | *Admission* | 21.4±7.9 | 21.2±7.9 | 18.1±5.5 | 0.540 |
| *48hrs post admission* | 23.2±9.2 | 20.0±7.1 | 21.5±8.1 | 0.248 |

**Table 2: Correlations between serum potassium and clinical parameters**

|  |  |  |  |
| --- | --- | --- | --- |
|  |  | **K+ levels at admission (n=124)** | **K+ levels 48hrs post admission (n=88)** |
| Age | Pearson Correlation | -0.144 | -0.097 |
| Sig. (2-tailed) | 0.134 | 0.393 |
| Time from injury to presentation (hrs) | Pearson Correlation | 0.107 | -0.088 |
| Sig. (2-tailed) | 0.243 | 0.420 |
| Pre-hospital use of IV fluids | Pearson Correlation | 0.024 | -0.007 |
| Sig. (2-tailed) | 0.792 | 0.950 |
| Pre-hospital use of Mannitol | Pearson Correlation | -0.005 | 0.156 |
| Sig. (2-tailed) | 0.960 | 0.147 |
| Mechanism of injury | Pearson Correlation | 0.059 | -0.128 |
| Sig. (2-tailed) | 0.516 | 0.234 |
| Systolic BP | Pearson Correlation | -0.140 | -0.185 |
| Sig. (2-tailed) | 0.121 | 0.085 |
| Diastolic BP | Pearson Correlation | -0.106 | -0.023 |
| Sig. (2-tailed) | 0.240 | 0.831 |
| Heart rate | Pearson Correlation | ***0.327\*\**** | -0.058 |
| Sig. (2-tailed) | ***<0.001*** | 0.601 |
| Respiratory rate | Pearson Correlation | 0.105 | 0.096 |
| Sig. (2-tailed) | 0.288 | 0.409 |
| Temperature | Pearson Correlation | 0.006 | -0.076 |
| Sig. (2-tailed) | 0.956 | 0.521 |
| Mannitol use | Pearson Correlation | 0.164 | -0.025 |
| Sig. (2-tailed) | 0.070 | 0.817 |
| Pupil examination | Pearson Correlation | -0.078 | -0.049 |
| Sig. (2-tailed) | 0.388 | 0.648 |
| Total GCS Score | Pearson Correlation | 0.068 | 0.155 |
| Sig. (2-tailed) | 0.453 | 0.149 |
| Injury Severity Score [head] | Pearson Correlation | -0.078 | -0.141 |
| Sig. (2-tailed) | 0.386 | 0.190 |
| ISS Score | Pearson Correlation | -0.101 | -0.045 |
| Sig. (2-tailed) | 0.264 | 0.678 |

**Table 3: Association between serum potassium and radiologic parameters**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
|  |  | **Hypokalemia** | **Normal** | **Hyperkalemia** | **P value** |
| Midline shift (mm) | Admission | 11.4±5.7 | 8.3±6.2 | 7.5±7.6 | 0.223 |
| 48hrs post admission | 12.5±6.3 | 9.5±5.8 | 5.0±5.0 | 0.126 |
| Compressed/absent basal cisterns | Admission | 92% | 71% | 77.5% | 0.552 |
| 48hrs post admission | 90.5% | 91.8% | 66.6% | 0.149 |
| Presence of epidural hematoma | Admission | 44% | 14.3% | 12.5% | ***0.005\**** |
| 48hrs post admission | 23.8% | 23.0% |  | 0.411 |
| Presence of subdural hematoma | Admission | 40% | 34.5% | 37.5% | 0.878 |
| 48hrs post admission | 38.1% | 41.0% | 33.3% | 0.920 |
| Presence of traumatic SAH | Admission | 40% | 38.1% | 12.5% | 0.332 |
| 48hrs post admission | 19.0% | 37.5% | 50.0% | ***0.045\**** |
| Presence of contusion hemorrhages | Admission | 56% | 39.3% | 50% | 0.312 |
| 48hrs post admission | 38.1% | 42.6% | 50% | 0.861 |
| SDH Thickness (mm) | Admission | 9.4±4.2 | 12.2±4.4 | 6.7±2.9 | 0.059 |
| 48hrs post admission | 11.1±3.5 | 11.7±5.1 | 7.5±3.5 | 0.500 |
| Rotterdam CT Score | Admission | 3.9±1.3 | 3.8±1.1 | 3.5±1.2 | 0.726 |
| 48hrs post admission | 3.9±1.0 | 3.9±1.1 | 3.7±1.4 | 0.842 |

**Table 4: Correlations between serum potassium and radiologic parameters**

|  |  |  |  |
| --- | --- | --- | --- |
|  |  | **K+ levels at admission (n=117)** | **K+ levels 48hrs post admission (n=88)** |
| Rotterdam CT head Score | Pearson Correlation | -0.024 | -0.005 |
| Sig. (2-tailed) | 0.797 | 0.967 |
| Midline shift (mm) | Pearson Correlation | -0.009 | -0.093 |
| Sig. (2-tailed) | 0.922 | 0.389 |
| Basal cisterns | Pearson Correlation | -0.065 | -0.115 |
| Sig. (2-tailed) | 0.486 | 0.284 |
| Presence of Epidural Hematoma | Pearson Correlation | ***0.270*** | 0.024 |
| Sig. (2-tailed) | ***0.003\**** | 0.826 |
| Presence of Subdural hematoma | Pearson Correlation | 0.005 | 0.002 |
| Sig. (2-tailed) | 0.956 | 0.989 |
| Presence of Intracerebral hematoma | Pearson Correlation | -0.020 | -0.054 |
| Sig. (2-tailed) | 0.831 | 0.614 |
| Presence of Traumatic Subarachnoid hemorrhage | Pearson Correlation | -0.117 | ***0.213\**** |
| Sig. (2-tailed) | 0.208 | ***0.047*** |
| Presence of Contusion hemorrhages | Pearson Correlation | 0.081 | -0.103 |
| Sig. (2-tailed) | 0.387 | 0.338 |
| Epidural Hematoma volume (ml) | Pearson Correlation | 0.104 | -0.265 |
| Sig. (2-tailed) | 0.723 | 0.431 |
| Subdural hematoma Thickness (mm) | Pearson Correlation | -0.036 | -0.213 |
| Sig. (2-tailed) | 0.835 | 0.258 |
| Intracerebral hematoma Volume (ml) | Pearson Correlation | 0.225 | -0.393 |
| Sig. (2-tailed) | 0.593 | 0.383 |