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# Acquired Hypernatremia in ICU Patients

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**A B S T R A C T**

A common ailment in intensive care units (ICUs), hypernatremia presents serious hazards to the physiological balance of patients and can sharply increase the death rate of critically ill patients. To determine if the cause of this electrolyte imbalance is an excess of sodium consumption or a lack of free water availability, a thorough clinical evaluation and urine electrolyte testing are frequently required. Careful monitoring of salt-to-water ratios becomes crucial in the complicated setting of intensive care units (ICUs), where patients may suffer from cognitive deficits that compromise natural physiological mechanisms such as thirst management. In order to properly treat hypernatremia, healthcare professionals—especially intensivists—play a crucial role in assuring watchful surveillance and prompt action. Treatment strategies typically involve the administration of free water and/or diuretics to enhance salt excretion, with correction rates carefully tailored to the individual patient's condition to mitigate risks such as cerebral edema. This multifaceted approach to managing hypernatremia underscores the importance of a coordinated and interdisciplinary effort in ICUs to optimize patient care and outcomes while minimizing potential complications associated with electrolyte disturbances.

**Key Words**: *Acquired Hypernatremia, Intensive care unit, Critically ill patients, Electrolyte imbalance, SOFA score*

## INTRODUCTION

After hospitalization and treatment, serum sodium concentrations (sNa) in ICU patients might rise above 145 mmol/L, which is known as ICU-acquired hypernatremia (IAH). Previous studies indicate IAH prevalence rates between 3 to 17%, which are associated with increased rates of death, morbidity, and prolonged hospitalizations in the intensive care unit[1-6]. The relationship between IAH and mortality is confirmed by recent studies, such as those conducted by Darmon et al., even when the s.Na threshold is as low as 143 mmol/L[6]. The Edelman equation explains how abnormalities in sodium and water levels lead to hypernatremia. Mostly iatrogenic, IAH can result from consuming too much salt through fluids like regular saline or by having less total body water in the body for a variety of reasons. The results of Kumar et al. highlight the connection between hypernatremia and higher mortality even if it is less common at the time of hospital admission.[7]

Hypernatremia stems from water loss, sodium gain, or a combination, with thirst regulation serving as a primary defense. Critically ill patients, often unconscious or sedated, rely on healthcare providers for water intake, heightening their susceptibility to hypernatremia. This prevalent ICU issue entails neurological, metabolic, and cardiac risks. Despite observing elevated mortality and hypernatremia cases in our ICU, prior investigations have not addressed this concern. Hence, our study endeavors to quantify hypernatremia incidence and pinpoint potential iatrogenic factors to mitigate future instances.

## RESEARCH METHODS AND METHODOLOGY

The study was conducted at the Central Intensive Care Unit (CICU) of Assam Medical College & Hospital, Dibrugarh, during the period from May 2023 to July 2023. It was a hospital-based prospective observational study with a sample size of 140 patients.

The selection criteria included patients with a length of stay in the ICU of 48 hours or more, while those with serum sodium levels ≥ 143mmol/L on admission, undergoing renal replacement therapy, or deceased/discharged before 48 hours were excluded from the study. Ethical clearance was obtained from the Institutional Ethics Committee (H) of Assam Medical College and Hospital, and written informed consent was obtained from all patients.

Upon admission, routine investigations including serum sodium, urea, creatinine, platelet count, serum bilirubin, and spot urine sodium and potassium were conducted. The Sequential Organ Failure Assessment Score (SOFA score)[8] was assessed on admission and at 24-hour and 48-hour intervals. Analysis of serum sodium, serum urea, serum creatinine, and serum bilirubin was performed using the Vitros 5600 integrated system at the Assam Clinical Biochemistry Laboratory (ACBL). The SOFA score, based on six different systems including respiratory, cardiovascular, hepatic, coagulation, renal, and neurological systems, was utilized for patient assessment. Additionally, input/output charts were maintained, and the administration of diuretics was monitored throughout the study period.

Microsoft Excel 2010 and the Statistical Package for Social Sciences (SPSS, version 20.0) were used for data analysis. Student T-test was used to compare continuous variables, which were reported as mean ± standard deviation. Fischer's exact test (where cell counts were 0 or less) and the Chi-square test were used to analyze discrete data, which were expressed as numbers (%). The relationships between continuous variables were evaluated using Pearson's correlation coefficient (r). For statistical significance, a p-value of less than 0.05 was used.

**DISCUSION**

In our thorough investigation of ICU-acquired hypernatremia, we meticulously scrutinized the intricate interplay between sodium overload, disrupted water balance, and their potential combined effects on patient outcomes. Among the cohort of 140 patients subjected to rigorous analysis, a predominant demographic trend emerged within the 18 to 30 age bracket, with a notable mean age of 42.75±17.24 years. Significantly, a substantial subset of 61 patients developed hypernatremia, with a discernible predilection towards older age groups, particularly those aged 50 and above—a trend corroborated by established literature[9]. Gender distribution among hypernatremic patients, while noted, did not yield statistically significant disparities.

To ensure the robustness of our findings, stringent exclusion criteria were applied, including meticulous screening for kidney injury through comprehensive assessments encompassing serum urea, serum creatinine, urine sodium, urine potassium, and SOFA score evaluations. The resultant mean SOFA score of 4.71 ± 1.29 aligned closely with established benchmarks in critical care research10, validating the methodological rigor of our approach.[10]

Detailed analyses conducted at the 24 and 48-hour intervals unearthed intriguing insights regarding the relationship between hypernatremia and key parameters such as fluid or sodium intake, fluid balance, serum urea, and creatinine levels. Contrary to prevailing assumptions, no significant correlations were observed, challenging conventional paradigms and suggesting the involvement of nuanced, multifactorial processes in hypernatremia development.

Notably, even the administration of furosemide—a commonly prescribed diuretic—yielded negligible impact on serum sodium levels, further underscoring the complexity of electrolyte regulation in critically ill patients. However, a stark revelation emerged concerning hypernatremia's association with heightened mortality post-48 hours ($p = 0.005^*$), emphasizing the profound clinical implications of this electrolyte imbalance.

**Table and Figures: Sofa Scoring System**

1. Respiratory System

| $PaO_2/FiO_2$ [mmHg (kPa)] | SOFA score |
|---|---|
| ≥ 400 (53.3) | 0 |
| < 400 (53.3) | +1 |
| < 300 (40) | +2 |
| < 200 (26.7) and mechanically ventilated | +3 |
| < 100 (13.3) and mechanically ventilated | +4 |

2. NERVOUS SYSTEM

| Glasgow Coma Scale | SOFA Score |
|---|---|
| 15 | 0 |
| 13–14 | +1 |
| 10–12 | +2 |
| 6–9 | +3 |
| < 6 | +4 |

3. CARDIOVASCULARSYSTEM

| Mean arterial pressure OR administration of vasopressors required | SOFA score |
|---|---|
| MAP ≥ 70 mmHg | 0 |
| MAP < 70 mmHg | +1 |
| dopamine ≤ 5 µg/kg/min or dobutamine (any dose) | +2 |
| dopamine > 5 µg/kg/min OR epinephrine ≤ 0.1 µg/kg/min OR norepinephrine ≤ 0.1 µg/kg/min | +3 |
| dopamine > 15 µg/kg/min OR epinephrine > 0.1 µg/kg/min OR norepinephrine > 0.1 µg/kg/min | +4 |

4. LIVER ASSESSMENT

| Bilirubin (mg/dl) [µmol/L] | SOFA score |
|---|---|
| < 1.2 [< 20] | 0 |
| 1.2–1.9 [20-32] | +1 |
| 2.0–5.9 [33-101] | +2 |
| 6.0–11.9 [102-204] | +3 |
| > 12.0 [> 204] | +4 |

5. COAGULATION ASSESMENT

| Platelets×$10^3$/µl | SOFA score |
|---|---|
| ≥ 150 | 0 |
| < 150 | +1 |
| < 100 | +2 |
| < 50 | +3 |
| < 20 | +4 |

6. RENAL ASSESMENT

| Creatinine (mg/dl) [µmol/L] (or urine output) | SOFA score |
|---|---|
| < 1.2 [< 110] | 0 |
| 1.2–1.9 [110-170] | +1 |
| 2.0–3.4 [171-299] | +2 |
| 3.5–4.9 [300-440] (or < 500 ml/d) | +3 |
| > 5.0 [> 440] (or < 200 ml/d) | +4 |

**RESULT TABLES:**

1. In our study we took only patients with serum sodium <143mmol/L on admission. After admission there is a significant change in serum sodium with p<0.001 in ANOVA test.

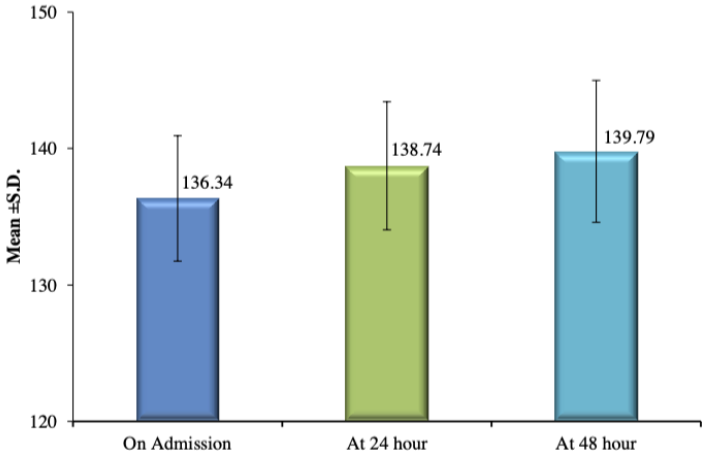


**FIG 1: Showing Serum Sodium Level at Different Time Interval**

2. We monitored serum sodium after 24 hours of patient admission, whose sodium was less than 143 at admission. In 96 patients, serum sodium (68.57%) was less than 143and 44patients (31.43%) serum sodium was ≥143

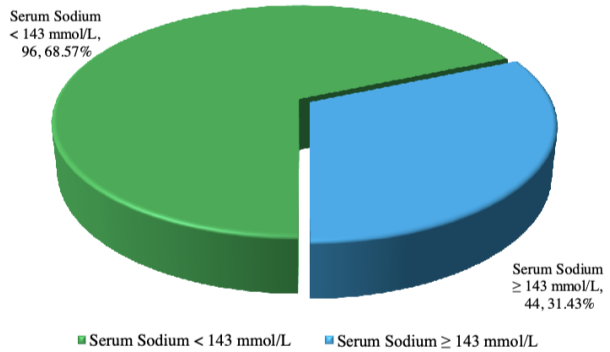


**FIG 2: Showing Serum Sodium Level after 24 hours**

3. Here we compared fluid intake in serum sodium <143 and ≥143mmol/L and the p value is 0.746. Similarly fluid balance and sodium intake is also not significant (p=0.189 and p=0.967 respectively). Serum creatinine and serum urea is also not significant on comparison (p=0.803 and p=0.213) respectively

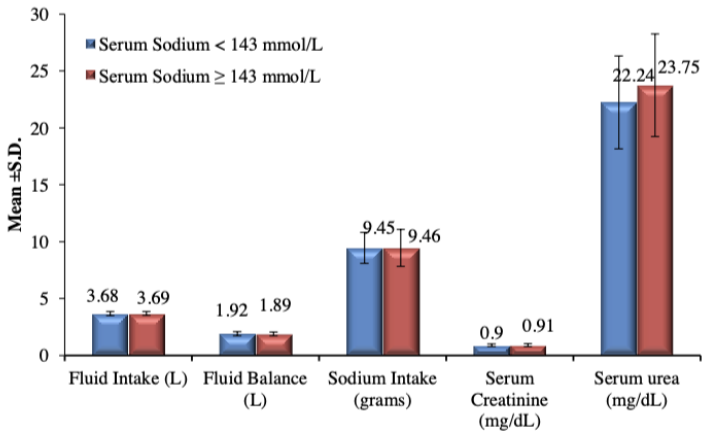


**FIG 3: Comparison of Fluid Intake, Fluid Balance, Sodium Intake, Serum Creatinine and Serum Urea at 24 hours**

4. Comparison of fluid intake, fluid balance, sodium intake, serum creatinine and serum urea at 48 hours is done in patients with serum sodium < 143 mmol /L and serum sodium ≥ 143 mmol/L. All the parameters p-value is not significant at 5% level of significance

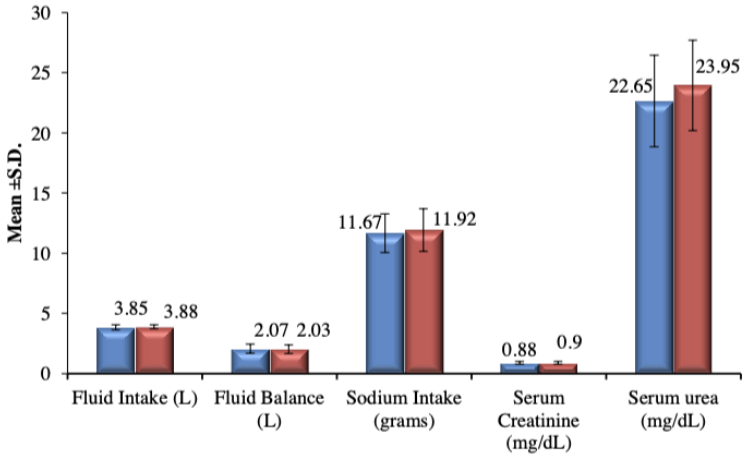


**FIG 4: Comparison of Fluid Intake, Fluid Balance, Sodium Intake, Serum Creatinine and Serum Urea at 48 hours**

5. In our study we observed that after 48 hours SOFA score is significance with P value.0.005*

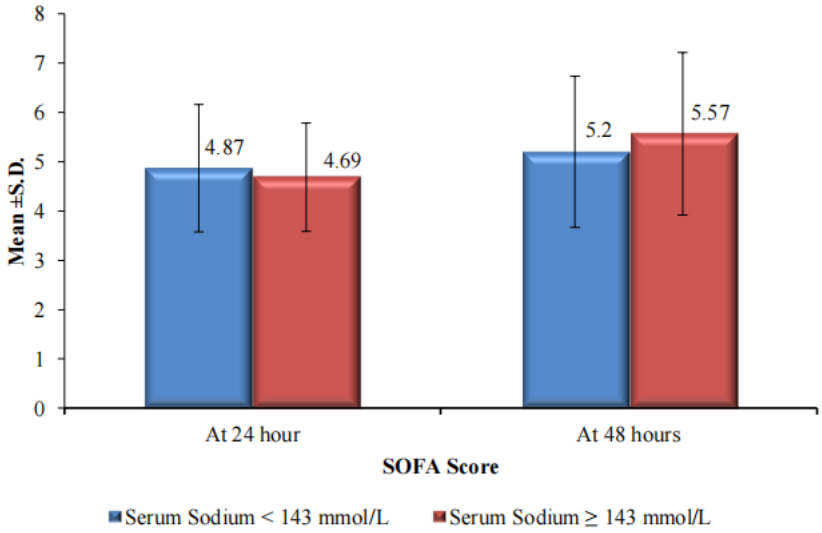


**FIG 5: Comparison of SOFA Score at 24 hours and 48 hours**

## CONCLUSION

In the study ICU acquired hypernatremia is not purely explained by sodium intake or fluid balance. This lack of association between ICU acquired hypernatremia and sodium intake and or fluid balance suggests other factors unaccounted for in the current paradigm. Hence IAH is not seems to be the primary iatrogenic complication. Severity of disease is an independent risk factor for both IAH and low renal sodium excretion. It may be due to other contributing factors such as sodium handling in third compartment. Hence prospective study about handling and distribution of sodium and sodium balance including hormonal activity should be needed to reveal the complex etiology of IAH. In our study development of IAH could not be explained by sodium intake or fluid balance though previous studies indicated that.

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