International Journal of Life Sciences, Biotechnology and Pharma Research Vol. 14, No. 2, February 2025 Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_14.2.2025.209

ORIGINAL RESEARCH

Study of serum sodium in acute myocardial infarction and its correlation with severity and complications of myocardial infarction

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Received: 11 January, 2025 Accepted: 28 January, 2025 Published: 17 February, 2025

ABSTRACT

Aim: To evaluate serum sodium levels in patients with acute myocardial infarction (AMI) and determine their correlation with disease severity and in-hospital complications. **Materials and Methods:** This prospective observational study included 100 patients admitted with AMI to a tertiary care hospital. Serum sodium levels were measured within 6 hours of admission and categorized into hyponatremia (<135 mmol/L), normal (135–145 mmol/L), and hypernatremia (>145 mmol/L). Severity of myocardial infarction was assessed using Killip classification and ejection fraction (EF) by echocardiography. In-hospital complications such as arrhythmias, heart failure, cardiogenic shock, reinfarction, and mortality were recorded. **Results:** Hyponatremia was present in 29% of patients, normal sodium in 67%, and hypernatremia in 4%. Patients with hyponatremia had significantly higher Killip class (p = 0.002), lower EF (mean 38.2% vs. 49.7%; p< 0.001), and increased incidence of complications including arrhythmias (p = 0.03), heart failure (p< 0.001), cardiogenic shock (p = 0.04), and mortality (p = 0.01). No significant correlation was observed between serum sodium and reinfarction. **Conclusion:** Hyponatremia in AMI patients is significantly associated with increased severity and worse in-hospital outcomes. Serum sodium may serve as a simple yet effective prognostic marker, and early correction could potentially improve clinical outcomes.

Keywords: Serum sodium, acute myocardial infarction, hyponatremia, complications, ejection fraction

INTRODUCTION

Acute myocardial infarction (AMI), commonly referred to as a heart attack, remains a leading cause of morbidity and mortality worldwide. It occurs when there is an abrupt interruption of blood flow to a portion of the heart muscle, most often due to the rupture of an atherosclerotic plaque and subsequent thrombus formation in the coronary arteries. The resulting ischemia and tissue necrosis lead to varying degrees of myocardial damage, the extent and severity of which have direct implications on short-term and long-term outcomes. Despite major advances in diagnosis, pharmacotherapy, and interventional strategies, AMI continues to pose a significant clinical burden, particularly due to its complications such as heart failure, arrhythmias, and cardiogenic shock.[1]

One of the many physiological changes that occur during and after an acute myocardial infarction is the alteration of electrolyte balance, particularly serum sodium levels. Sodium, the principal extracellular cation, plays a crucial role in maintaining fluid balance, nerve transmission, and muscle function, including that of the myocardium. Disturbances in serum sodium levels, even those that are mild and transient, can have important clinical implications, especially in patients with cardiovascular disease. Hyponatremia, defined as a serum sodium concentration below the normal range, is commonly observed in patients with AMI and has been associated with increased in-hospital mortality and a higher incidence of complications.[2]

The pathophysiology underlying sodium imbalance in the context of AMI is multifactorial. Myocardial injury leads to neurohormonal activation, including the renin-angiotensin-aldosterone system (RAAS), sympathetic nervous system stimulation, and release of vasopressin. These mechanisms, which are primarily compensatory in nature, attempt to preserve cardiac output and tissue perfusion. However, they also promote water retention and dilutional hyponatremia. Additionally, left ventricular dysfunction, often seen following AMI, contributes to reduced renal perfusion, further stimulating these pathways and exacerbating sodium and water

DOI: 10.69605/ijlbpr_14.2.2025.209

imbalance. Thus, serum sodium levels may reflect the severity of myocardial injury and the degree of neurohormonal activation, serving as a potential biomarker for risk stratification.[3]

The severity of AMI is influenced by several factors, including the extent of myocardial involvement, the duration of ischemia before reperfusion, and the presence of comorbid conditions such as diabetes mellitus, hypertension, or chronic kidney disease. Clinically, severity can be assessed using tools like the Killip classification, which categorizes patients based on the presence and degree of heart failure, or by evaluating parameters such as ejection fraction, infarct size, and levels of cardiac biomarkers. Complications arising from AMI—such as arrhythmias, heart failure, mechanical complications (e.g., ventricular septal rupture), and pericarditis—significantly impact prognosis and treatment decisions. Understanding factors that correlate with the occurrence of these complications can improve early identification of high-risk patients and guide therapeutic strategies.[4]

In this context, serum sodium presents as a simple, readily available, and cost-effective laboratory parameter that may provide insight into the patient's clinical status. Several observational studies and clinical reports have indicated a relationship between lower serum sodium levels and worse outcomes in patients with AMI. However, the precise nature of this relationship, its strength, and its implications for clinical practice remain areas of active investigation. Whether low sodium levels are a marker of disease severity or play a direct role in worsening outcomes is still debated, but their consistent association with adverse events underscores their potential utility in clinical settings.[5]

Given the widespread availability of serum sodium measurements and their inclusion in routine laboratory panels, a deeper understanding of their significance in the context of AMI could enhance patient monitoring and prognostication. If a clear and consistent correlation can be established between serum sodium levels and the severity and complications of AMI, it could offer clinicians an additional tool for early risk assessment. This, in turn, may facilitate timely intervention, closer monitoring, and more informed decision-making regarding the level of care and resource allocation.[6]

This study aims to explore the relationship between serum sodium levels and the clinical course of acute myocardial infarction. Specifically, it investigates whether serum sodium levels at the time of presentation can be used to assess the severity of AMI and predict the likelihood of developing complications during hospitalization. By evaluating this potential correlation, the study seeks to contribute to the growing body of evidence supporting the prognostic significance of biochemical markers in cardiovascular disease and to offer practical insights for enhancing patient care.

MATERIALS AND METHODS

This was a prospective observational study conducted at a tertiary care center. Ethical approval was obtained from the Institutional Ethics Committee prior to initiation of the study, and informed consent was obtained from all participants.A total of 100 patients diagnosed with acute myocardial infarction (AMI) were included in the study. Diagnosis of AMI was based on clinical presentation, ECG changes, and elevated cardiac biomarkers (e.g., troponin I or T, CK-MB), in accordance with the Fourth Universal Definition of Myocardial Infarction.

Inclusion Criteria

- Patients aged ≥18 years.
- Confirmed diagnosis of acute myocardial infarction (STEMI or NSTEMI).
- Admission within 24 hours of symptom onset.

Exclusion Criteria

- Patients with known chronic kidney disease or on dialysis.
- Patients with liver cirrhosis or decompensated heart failure prior to admission.
- Patients receiving diuretics, sodium supplements, or drugs affecting sodium balance prior to admission.
- Patients with endocrine disorders such as SIADH, Addison’s disease, or hypothyroidism.

Data Collection

On admission, a detailed history and physical examination were performed for all patients. Clinical data collected included age, sex, and the presence of cardiovascular risk factors such as hypertension, diabetes mellitus, smoking, and dyslipidemia. The time of onset and duration of chest pain were documented, along with the type of myocardial infarction—classified as ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI). The severity of heart failure at presentation was assessed using the Killip classification.

Venous blood samples were obtained within the first six hours of hospital admission. Laboratory investigations included measurement of serum sodium levels using the ion-selective electrode method, along with renal function tests, blood glucose, and lipid profile. Cardiac biomarkers, including Troponin I or T and creatine kinase-MB (CK-MB), were also analyzed to assess myocardial injury. Based on the serum sodium values, patients were categorized into three groups: hyponatremia (serum sodium <135 mmol/L), normal sodium levels (135–145 mmol/L), and hypernatremia (>145 mmol/L).

The severity of myocardial infarction was further evaluated using the Killip classification, left ventricular ejection fraction measured by two-dimensional echocardiography, and the extent of myocardial damage inferred from cardiac biomarker

DOI: 10.69605/ijlbpr_14.2.2025.209

levels. In-hospital complications monitored during the study included the occurrence of arrhythmias, heart failure, cardiogenic shock, reinfarction, and mortality.

Statistical Analysis

Data were analyzed using SPSS version 26.0. Categorical variables were presented as frequencies and percentages, and continuous variables as mean ± standard deviation. Correlation between serum sodium levels and severity/complications of MI was assessed using Chi-square test, ANOVA, and Pearson correlation coefficient as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics (Table 1)

The study population consisted of 100 patients diagnosed with acute myocardial infarction (AMI). The majority were male (72%), while females comprised 28% of the cohort. Although there was a higher proportion of males, the difference was not statistically significant (p = 0.07). Among comorbidities, hypertension was present in 54% of patients, diabetes mellitus in 46%, dyslipidemia in 42%, and smoking in 38%. None of these comorbid conditions showed a statistically significant association with serum sodium categories (p> 0.05). Regarding the type of myocardial infarction, ST-elevation myocardial infarction (STEMI) was more common, accounting for 68% of cases, while 32% had non-ST-elevation myocardial infarction (NSTEMI). A statistically significant association was observed between type of MI and serum sodium levels, with STEMI being significantly more common among patients with abnormal sodium levels (p = 0.04).

Serum Sodium Distribution (Table 2)

Serum sodium levels on admission revealed that 29% of patients had hyponatremia (serum sodium <135 mmol/L), 67% had normal sodium levels (135–145 mmol/L), and only 4% had hypernatremia (>145 mmol/L). This indicates that hyponatremia was relatively common among patients presenting with AMI, while hypernatremia was rare. These findings suggest that disturbances in sodium balance, particularly hyponatremia, may play a notable role in the clinical profile of AMI.

Correlation with Killip Class (Table 3)

Killip classification, used to assess the severity of heart failure, showed a strong correlation with serum sodium levels. Among patients with normal sodium levels, 71.6% were in Killip Class I (no signs of heart failure), compared to only 34.5% in the hyponatremia group and 50% in the hypernatremia group. In contrast, more severe heart failure (Killip Class II to IV) was significantly more common among patients with hyponatremia, with 41.4% in Class II, 17.2% in Class III, and 6.9% in Class IV. These differences were statistically significant (p = 0.002), indicating that lower serum sodium levels were associated with greater severity of heart failure on presentation.

Correlation with Ejection Fraction (Table 4)

Left ventricular ejection fraction (EF), an important indicator of cardiac function, was significantly lower in patients with hyponatremia compared to those with normal sodium levels. The mean EF in the hyponatremic group was 38.2 ± 6.1%, while it was 49.7 ± 5.8% in the normal sodium group. Patients with hypernatremia had a mean EF of 42.1 ± 4.9%. These findings demonstrate a statistically significant relationship between low sodium levels and reduced systolic function (p< 0.001), suggesting that hyponatremia may be a marker of more extensive myocardial dysfunction.

In-Hospital Complications (Table 5)

Hyponatremia was significantly associated with a higher incidence of several in-hospital complications. Arrhythmias occurred in 34.5% of patients with hyponatremia, compared to 13.4% in the normal sodium group (p = 0.03). Heart failure was seen in nearly half (48.3%) of the hyponatremic patients, a stark contrast to 14.9% in the normal group (p< 0.001). Cardiogenic shock was also more frequent in the hyponatremic group (13.8%) versus the normal group (3.0%), with a statistically significant difference (p = 0.04). Although reinfarction was more common in the hyponatremia group (10.3%) than the normal sodium group (3.0%), the difference was not statistically significant (p = 0.22). Mortality rates were also significantly higher in the hyponatremic group (17.2%) compared to 3.0% in the normal sodium group (p = 0.01). Overall, these results suggest that hyponatremia is not only a marker of disease severity but also a predictor of adverse outcomes and complications in patients with AMI.

Table 1: Baseline Characteristics of Study Population (N = 100)

Variable	Number (n)	Percentage (%)	*p*-value
Sex			
Male	72	72%	0.07
Female	28	28%	
Comorbidities			
Hypertension	54	54%	0.08
Diabetes Mellitus	46	46%	0.12
Smoking	38	38%	0.21

DOI: 10.69605/ijlbpr_14.2.2025.209

Dyslipidemia	42	42%	0.15
Type of MI			
STEMI	68	68%	0.04*
NSTEMI	32	32%	

Table 2: Distribution of Patients According to Serum Sodium Levels

Serum Sodium Category	Number (n)	Percentage (%)
Hyponatremia (<135 mmol/L)	29	29%
Normal (135–145 mmol/L)	67	67%
Hypernatremia (>145 mmol/L)	4	4%

Table 3: Correlation of Serum Sodium Levels with Killip Class

Killip Class	Hyponatremia (n=29)	Normal Sodium (n=67)	Hypernatremia (n=4)	*p*-value
I	10 (34.5%)	48 (71.6%)	2 (50%)	
II	12 (41.4%)	14 (20.9%)	1 (25%)	
III	5 (17.2%)	3 (4.5%)	0 (0%)	
IV	2 (6.9%)	2 (3.0%)	1 (25%)	0.002*

Table 4: Correlation of Serum Sodium Levels with Ejection Fraction

Serum Sodium Category	Mean EF (%) ± SD	*p*-value
Hyponatremia	38.2 ± 6.1	
Normal Sodium	49.7 ± 5.8	
Hypernatremia	42.1 ± 4.9	<0.001*

Table 5: In-Hospital Complications According to Serum Sodium Levels

Complication	Hyponatremia (n=29)	Normal Sodium (n=67)	Hypernatremia (n=4)	*p*-value
Arrhythmias	10 (34.5%)	9 (13.4%)	1 (25%)	0.03*
Heart Failure	14 (48.3%)	10 (14.9%)	1 (25%)	<0.001*
Cardiogenic Shock	4 (13.8%)	2 (3.0%)	1 (25%)	0.04*
Reinfarction	3 (10.3%)	2 (3.0%)	0 (0%)	0.22
Mortality	5 (17.2%)	2 (3.0%)	1 (25%)	0.01*

DISCUSSION

The demographic and clinical profile of the study population aligns with previously documented patterns in acute myocardial infarction (AMI). Male predominance (72%) was observed, consistent with the gender distribution reported by Kaleem et al. [5], where men were more frequently affected by AMI. While hypertension (54%) and diabetes mellitus (46%) were the most common comorbidities, no significant association was found between these risk factors and serum sodium levels, which echoes the findings of Goldberg et al.[6], who also reported comorbidities as non-contributory to sodium imbalances.

Interestingly, a significant correlation was found between ST-elevation myocardial infarction (STEMI) and low sodium levels (p = 0.04). Wang et al. [7] and Oren et al.[8] observed similar trends, highlighting that STEMI patients were more prone to hyponatremia, potentially due to more extensive myocardial injury and subsequent neurohormonal activation.

Our study revealed a 29% prevalence of hyponatremia in AMI patients, which is within the range reported in international literature. Cordova Sanchez et al.[9] reported a similar rate of admission hyponatremia in AMI patients and found that it significantly impacted outcomes including in-hospital and 30-day mortality. The rarity of hypernatremia (4%) was also observed in the SAMI-II study [10], where both low (<135 mEq/L) and high (≥141 mEq/L) sodium levels were associated with higher mortality, although hyponatremia had a more persistent association.

A significant inverse relationship between serum sodium levels and Killip class was demonstrated in our cohort, with more severe heart failure presentations (Killip Class II–IV) predominating in hyponatremic patients (p = 0.002). This supports previous findings by Goldberg et al. [6] and Mentz et al.[11], who documented that low sodium reflects advanced heart failure and neurohormonal dysregulation. The study by Troncone et al. [12] further reinforces this association, showing that hyponatremia in patients receiving cardiac resynchronization therapy predicted worsening heart failure.

Our results showed significantly lower ejection fraction (EF) in hyponatremic patients (mean EF: 38.2 ± 6.1%), indicating more extensive myocardial dysfunction. This pattern is supported by Goyal et al.[13], who linked hyponatremia with lower EF and adverse remodeling. Similarly, Oren et al. 8 emphasized that patients with low left ventricular ejection fraction and low sodium had markedly higher mortality risks. These findings underscore the utility

DOI: 10.69605/ijlbpr_14.2.2025.209

of serum sodium as a surrogate marker for systolic dysfunction.

Hyponatremia was associated with a significantly higher incidence of arrhythmias (34.5%), heart failure (48.3%), cardiogenic shock (13.8%), and mortality (17.2%)—all with statistical significance. These findings mirror those of Cordova Sanchez et al. [9], who reported a direct link between hyponatremia and adverse events during hospitalization, including increased ICU admission and new-onset heart failure. Kaleem et al. 5 and Singh et al. 14 also reported that worsening sodium levels correlated with higher mortality and complication rates in AMI and acute heart failure patients.

Moreover, Plakht et al. [10] in the SAMI-II project confirmed that sodium abnormalities during hospitalization (both hypo- and hypernatremia) were independently associated with increased mortality. Goldberg et al. [6] extended this finding to long-term outcomes, showing hyponatremia predicted future heart failure and death post-STEMI, highlighting its prognostic utility beyond hospital stay.

CONCLUSION

This study demonstrates a significant association between hyponatremia and increased severity and complications in patients with acute myocardial infarction. Patients with low serum sodium levels had higher Killip class, reduced ejection fraction, and a greater incidence of heart failure, arrhythmias, cardiogenic shock, and mortality. These findings suggest that hyponatremia can serve as an important prognostic marker in AMI. Early identification and correction of sodium imbalance may improve clinical outcomes. Routine assessment of serum sodium should be considered in the initial evaluation of AMI patients.

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