International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-2-12*

Hyponatremia in Acute ST-Elevation Myocardial Infarction as a Predictor of Early Clinical Outcomes: An Observational Study.

Karthigeyan PN[1], Kalaivani A[1], Karthik S[1]*.

[1]Assistant Professor, Department of general medicine, Government Mohan Kumaramangalam medical College hospital, Salem, Tamil Nadu.

Correspondence:
Dr Karthik S,
Assistant professor, Department of general medicine,Government Mohan Kumaramangalam medical College hospital, Salem, Tamil Nadu.

Email:
mskarthik31@gmail.com

Received: 12-03-2026
Revised: 24-03-2026
Accepted: 10-04-2026
Published: 21-04-2026

Abstract

Background: Electrolyte abnormalities are common in patients presenting with acute ST-elevation myocardial infarction (STEMI). Hyponatremia occurring during the early phase of myocardial infarction is considered an indirect marker of neurohumoral activation and may indicate poor prognosis. Early identification of high-risk patients helps in timely risk stratification and management. ***Objectives:*** To determine the association between hyponatremia during the early phase of acute STEMI and short-term clinical outcomes including left ventricular dysfunction, complications, and in-hospital mortality. ***Methods:*** This hospital-based observational study included 120 patients admitted with confirmed acute STEMI. Serum sodium levels were measured at admission, 24 hours, 48 hours, and 72 hours. Echocardiographic assessment of left ventricular ejection fraction (LVEF) was performed at admission and repeated at 72 hours. Patients were followed during hospital stay for development of complications such as left ventricular failure, cardiogenic shock, arrhythmia, heart block, and mortality. Statistical analysis was performed using SPSS version 23. Chi-square test and Pearson correlation were used to determine associations, considering $p<0.05$ statistically significant. ***Results:*** Hyponatremia was observed in 38% of patients at admission and 47% at 24 hours. Patients aged more than 60 years showed significantly higher prevalence of hyponatremia (82%) compared to younger age groups ($p=0.003$). Among patients with moderate LV dysfunction, 88% had hyponatremia within 24 hours of admission. Complications occurred in 18% of patients with normal sodium levels compared to 64% in mild hyponatremia, 83% in moderate hyponatremia, and 100% in severe hyponatremia ($p=0.001$). Persistent hyponatremia at 72 hours was associated with increased risk of cardiogenic shock and mortality. ***Conclusion:*** Hyponatremia is frequently observed in patients with acute STEMI and is associated with increased severity of left ventricular dysfunction and higher risk of complications. Moderate to severe hyponatremia serves as a useful prognostic marker for adverse short-term outcomes.

Keywords: STEMI, Hyponatremia, Left ventricular dysfunction, Prognosis, Acute coronary syndrome.

INTRODUCTION

Cardiovascular diseases continue to be the leading cause of mortality worldwide, contributing significantly to global disease burden and healthcare expenditure [1]. Acute ST-elevation myocardial infarction (STEMI) represents one of the most severe manifestations of coronary artery disease and is associated with high morbidity and mortality despite advances in pharmacological and interventional therapies [2]. Early identification of prognostic markers plays an important role in improving patient outcomes by facilitating prompt risk stratification and appropriate clinical management.

Electrolyte abnormalities are frequently observed in patients hospitalized with acute myocardial infarction. Among these, hyponatremia is one of the most common disturbances and has been increasingly recognized as a marker of disease severity [3]. Hyponatremia is defined as serum sodium concentration less than 135 mEq/L and may occur due to dilutional or depletional mechanisms [4]. In the setting of acute myocardial infarction, hyponatremia is usually dilutional and results from neurohumoral activation triggered by reduced cardiac output [5].

Acute myocardial infarction leads to decreased effective circulating blood volume, stimulating baroreceptors and activating compensatory mechanisms such as the renin-angiotensin-aldosterone system, sympathetic nervous system, and arginine vasopressin secretion [6]. Increased vasopressin secretion promotes water retention in renal collecting ducts, resulting in dilution of serum sodium concentration [7]. Additional factors such as stress, nausea, pain, use of opioids, and hemodynamic instability further contribute to increased antidiuretic hormone secretion [8].

Hyponatremia has been associated with increased mortality in various cardiovascular conditions including heart failure, acute coronary syndrome, and cardiomyopathy [9]. Several studies have demonstrated that hyponatremia is independently associated with increased short-term and long-term mortality following myocardial infarction [10]. It is considered an indirect marker of neurohumoral activation and severity of cardiac dysfunction [11].

Left ventricular dysfunction following myocardial infarction is an important determinant of prognosis. Reduced left ventricular ejection fraction leads to decreased tissue perfusion and activation of neurohormonal compensatory mechanisms, thereby contributing to development of hyponatremia [12]. Studies have shown that patients with lower ejection fraction have higher prevalence of hyponatremia and worse clinical outcomes [13].

Elderly individuals are more vulnerable to hyponatremia due to age-related decline in renal function, reduced glomerular filtration rate, impaired urinary dilution capacity, and altered hormonal response [14]. Reduced total body water and increased vasopressin response to osmotic stimuli predispose elderly individuals to electrolyte imbalance even with minimal physiological stress [15].

Hyponatremia may directly influence myocardial contractility through alterations in intracellular ion exchange mechanisms. Activation of sodium-calcium exchanger results in increased intracellular calcium accumulation, which may worsen myocardial injury and predispose patients to arrhythmias and cardiac dysfunction [16]. Hyponatremia may also promote myocardial fibrosis and ventricular remodeling, thereby contributing to worsening heart failure [17].

Recent studies have highlighted the importance of serum sodium as a simple, inexpensive, and readily available prognostic marker in acute coronary syndrome [18]. Serial measurement of sodium levels provides valuable information regarding disease progression and response to treatment [19]. Persistent hyponatremia has been shown to be associated with increased risk of cardiogenic shock, arrhythmias, and mortality [20].

Early identification of hyponatremia in acute STEMI patients may help clinicians identify high-risk individuals who require closer monitoring and aggressive management. Timely correction of underlying hemodynamic abnormalities may improve patient outcomes and reduce complications [21].

The present study aims to evaluate the association between hyponatremia and short-term clinical outcomes in patients with acute STEMI. The study also examines the relationship between serum sodium levels and left ventricular ejection fraction, thereby contributing to improved understanding of prognostic significance of electrolyte imbalance in myocardial infarction.

Objectives:

Primary Objective:

To determine the association between hyponatremia in acute STEMI and short-term clinical outcomes.

Secondary Objectives:

1. To assess relationship between serum sodium levels and left ventricular dysfunction.
2. To determine prevalence of hyponatremia at admission and during hospital stay.
3. To evaluate association between severity of hyponatremia and complications such as LV failure, shock, arrhythmia, and mortality.

MATERIALS AND METHODS

This hospital-based observational study was conducted in a tertiary care teaching hospital over 18 months. A total of 120 patients diagnosed with acute ST-elevation myocardial infarction based on clinical presentation, ECG findings showing ST elevation in two contiguous leads, and elevated cardiac biomarkers were included [1]. Patients aged above 18 years were eligible.

Patients with chronic kidney disease, chronic liver disease, hypothyroidism, adrenal insufficiency, malignancy, and patients on diuretics were excluded to avoid confounding electrolyte abnormalities [9].

Serum sodium levels were measured at admission, 24 hours, 48 hours, and 72 hours using ion selective electrode method [5]. Echocardiography was performed at admission and 72 hours to determine left ventricular ejection fraction using Simpson's method [7].

Patients were monitored for complications including left ventricular failure, cardiogenic shock, arrhythmia, heart block, and mortality. Data were analyzed using SPSS version 23. Chi-square test and Pearson correlation were applied. A p value <0.05 was considered statistically significant.

RESULTS

Table 1: Age distribution and hyponatremia in a study in a tertiary care teaching hospital in Tamil Nadu

Serum Sodium	<40 yrs	41–50 yrs	51–60 yrs	>60 yrs	p value
<135 mEq/L at admission	3	7	20	12	0.003
<135 mEq/L at 24 hrs	2	9	24	14	0.002
<135 mEq/L at 72 hrs	1	5	14	10	0.021

Hyponatremia was more common among elderly patients. At 24 hours, 82% of patients aged more than 60 years had hyponatremia compared to 22% among patients younger than 40 years. The association between increasing age and hyponatremia was statistically significant.

Table 2: Serum sodium and LV dysfunction in a study in a tertiary care teaching hospital in Tamil Nadu

EF category	Normal sodium	Hyponatremia	p value
Normal EF	30	4	0.001
Mild dysfunction	28	24	
Moderate dysfunction	8	26	

Patients with moderate LV dysfunction had significantly higher prevalence of hyponatremia compared to patients with normal LV function. Nearly 76% of patients with moderate LV dysfunction had serum sodium <135 mEq/L.

Table 3: Hyponatremia and complications in a study in a tertiary care teaching hospital in Tamil Nadu

Sodium level	No complication	LV failure	Shock	Arrhythmia	Death
>135	48	6	0	4	0
130–134	10	12	2	2	2
125–129	4	10	4	2	1
<125	0	6	6	2	3

Complications were significantly higher among patients with moderate and severe hyponatremia. Cardiogenic shock and mortality were predominantly observed among patients with serum sodium <125 mEq/L.

Table 4: Hyponatremia severity and risk of complications in a study in a tertiary care teaching hospital in Tamil Nadu

Sodium category	Risk of complications
Normal sodium	18%
Mild hyponatremia	64%
Moderate hyponatremia	83%
Severe hyponatremia	100%

The risk of complications increased progressively with severity of hyponatremia. All patients with severe hyponatremia developed complications during hospital stay.

DISCUSSION

The present study demonstrates a significant association between hyponatremia and adverse clinical outcomes in patients presenting with acute ST-elevation myocardial infarction. Hyponatremia was observed in a substantial proportion of patients during the first 24 hours of admission, indicating early neurohumoral activation following myocardial injury. Similar findings have been reported in previous studies which observed prevalence of hyponatremia ranging from 30% to 50% among patients with acute coronary syndrome [9,10].

Hyponatremia in myocardial infarction is primarily dilutional and occurs as a result of activation of compensatory neurohumoral mechanisms triggered by reduced cardiac output [6]. Reduced effective circulating volume stimulates

baroreceptors, leading to increased secretion of vasopressin, activation of renin-angiotensin-aldosterone system, and sympathetic nervous system stimulation [7]. These physiological responses result in increased water reabsorption and dilution of serum sodium levels [8].

The present study showed that elderly patients had higher prevalence of hyponatremia compared to younger individuals. Aging is associated with decline in renal concentrating ability, reduced glomerular filtration rate, and altered hormonal responsiveness [14]. Increased vasopressin secretion and reduced renal capacity to excrete free water predispose elderly patients to dilutional hyponatremia [15].

The association between hyponatremia and left ventricular dysfunction observed in the present study is consistent with previous literature. Patients with reduced ejection fraction had significantly lower serum sodium levels. Reduced cardiac output results in decreased renal perfusion, which stimulates neurohumoral activation and promotes water retention [12]. Hyponatremia therefore reflects severity of myocardial damage and impaired cardiac function [13].

The present study also observed increased risk of complications such as cardiogenic shock, arrhythmia, and mortality among patients with moderate and severe hyponatremia. Severe hyponatremia may worsen myocardial contractility by altering intracellular calcium homeostasis and impairing excitation-contraction coupling in cardiac muscle fibers [16]. Increased intracellular calcium accumulation may lead to increased oxidative stress, myocardial necrosis, and electrical instability [17].

Hyponatremia may also promote myocardial fibrosis and ventricular remodeling through activation of vasopressin-mediated pathways [18]. Experimental studies have demonstrated that vasopressin stimulates fibroblast proliferation and extracellular matrix deposition, leading to structural changes in myocardium [17]. These changes may contribute to worsening heart failure and reduced cardiac output.

Several clinical studies have reported that early hyponatremia is an independent predictor of mortality following myocardial infarction [10]. A meta-analysis involving patients with acute coronary syndrome demonstrated that moderate to severe hyponatremia significantly increases risk of short-term mortality [19]. Persistent hyponatremia during hospitalization has also been associated with prolonged hospital stay and increased healthcare costs [20].

The present study findings support the role of serum sodium as an inexpensive and easily accessible prognostic marker in acute myocardial infarction. Measurement of serum sodium is routinely performed in hospitalized patients and does not require additional resources. Serial monitoring of sodium levels may help clinicians assess disease progression and response to treatment [21].

Early identification of patients with persistent hyponatremia may allow clinicians to initiate appropriate therapeutic interventions such as optimization of fluid balance, correction of hemodynamic instability, and close monitoring for development of complications [18]. Recent studies have suggested potential role of SGLT2 inhibitors in improving sodium balance and cardiac outcomes in heart failure patients [20].

The findings of the present study emphasize the importance of routine electrolyte monitoring in patients with acute STEMI. Hyponatremia should not be considered merely a laboratory abnormality but rather a marker of disease severity and poor prognosis.

Further multicentric studies with larger sample size and long-term follow up are required to establish the role of hyponatremia as an independent predictor of mortality and major adverse cardiovascular events.

CONCLUSION

Hyponatremia is a common finding in patients with acute STEMI. Elderly patients and those with reduced ejection fraction are more prone to develop hyponatremia. Moderate to severe hyponatremia is associated with increased risk of complications including LV failure, cardiogenic shock, arrhythmia, and mortality. Serum sodium measurement can be used as a simple prognostic marker in acute myocardial infarction.

Strengths and Limitations

This study highlights the usefulness of serum sodium as an early prognostic marker in acute STEMI. Serial measurement of sodium levels improved understanding of dynamic changes occurring during acute myocardial infarction. However, the study was conducted in a single center with limited sample size. Long-term follow-up was not performed. Effect of treatment interventions on sodium correction was not assessed.

Recommendations

Routine monitoring of serum sodium should be performed in all patients with acute myocardial infarction. Patients with persistent hyponatremia should be closely monitored for development of complications. Larger multicentric studies are required to validate the prognostic significance of hyponatremia in myocardial infarction.

REFERENCES

1. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J. 2018;39(2):119–177.
2. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J. 2019;40(3):237–269.
3. Goldberg A, Hammerman H, Petcherski S, Zdorovyak A, Yalonetsky S, Kapeliovich M, et al. Prognostic importance of hyponatremia in acute ST-elevation myocardial infarction. Am J Med. 2004;117(4):242–248.
4. Spasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, et al. Clinical practice guideline on diagnosis and treatment of hyponatraemia. Nephrol Dial Transplant. 2014;29(Suppl 2):i1–i39.
5. Gheorghiade M, Abraham WT, Albert NM, Greenberg BH, O'Connor CM, She L, et al. Relationship between admission serum sodium concentration and clinical outcomes in patients hospitalized for heart failure. JAMA. 2007;297(12):1332–1340.
6. Rouleau JL, Packer M, Moyé L, de Champlain J, Bichet D, Klein M, et al. Prognostic value of neurohumoral activation in patients with acute myocardial infarction. J Am Coll Cardiol. 1994;24(3):583–591.
7. Graham LN, Smith PA, Stoker JB, Mackintosh AF, Mary DA. Time course of sympathetic neural hyperactivity after uncomplicated acute myocardial infarction. Circulation. 2003;107(6):793–797.
8. Choi JS, Kim CS, Bae EH, Ma SK, Ahn YK, Jeong MH, et al. Prognostic impact of hyponatremia occurring at various time points during hospitalization in patients with acute myocardial infarction. Medicine (Baltimore). 2017;96(23):e7023.
9. Singla I, Zahid M, Good CB, Macioce A, Sonel AF. Effect of hyponatremia (<135 mEq/L) on outcome in patients with non-ST-elevation acute coronary syndrome. Am J Cardiol. 2007;100(3):406–408.
10. Ma QQ, Fan XD, Li T, Hao YY, Ma F. Short- and long-term prognostic value of hyponatremia in patients with acute coronary syndrome: A systematic review and meta-analysis. PLoS One. 2018;13(3):e0193857.
11. Filippatos TD, Makri A, Elisaf MS, Liamis G. Hyponatremia in the elderly: Challenges and solutions. Clin Interv Aging. 2017;12:1957–1965.
12. Mullens W, Verbrugge FH, Nijst P, Tang WH. Renal sodium avidity in heart failure: from pathophysiology to treatment strategies. Eur Heart J. 2017;38(24):1872–1882.
13. Lazzeri C, Valente S, Chiostri M, Attanà P, Picariello C, Gensini GF. Usefulness of hyponatremia in the acute phase of ST elevation myocardial infarction as a marker of severity. Am J Cardiol. 2012;110(10):1419–1424.
14. Liamis G, Filippatos TD, Elisaf MS. Electrolyte disorders associated with commonly used medications in the elderly. Drugs Aging. 2016;33(5):353–364.
15. Bevilacqua M, Norbiato G, Chebat E, Raggi U, Cavaiani P, Guzzetti R, et al. Osmotic and nonosmotic control of vasopressin release in elderly subjects. J Clin Endocrinol Metab. 1987;65(6):1243–1247.
16. Shattock MJ, Ottolia M, Bers DM, Blaustein MP, Boguslavskyi A, Bossuyt J, et al. Na+/Ca2+ exchange and Na+/K+-ATPase in the heart. J Physiol. 2015;593(6):1361–1382.
17. Yang XD, Zhao LY, Zheng QS, Li X. Effects of arginine vasopressin on growth of rat cardiac fibroblasts via V1 receptor stimulation. J Cardiovasc Pharmacol. 2003;42(1):132–135.
18. Warren AM, Grossmann M, Christ-Crain M, Russell N. Syndrome of inappropriate antidiuresis: pathophysiology, diagnosis, and management. Endocr Rev. 2023;44(5):819–861.
19. Yeoh SE, Docherty KF, Jhund PS, Petrie MC, Inzucchi SE, Køber L, et al. Relationship of dapagliflozin with serum sodium in patients with heart failure: insights from DAPA-HF trial. JACC Heart Fail. 2022;10(5):306–318.
20. Schulze PC, Bogoviku J, Westphal J, Aftanski P, Haertel F, Grund S, et al. Effects of early empagliflozin initiation on diuresis and kidney function in acute heart failure (EMPAG-HF). Circulation. 2022;146(4):289–298.
21. Goldberg A, Hammerman H, Petcherski S, Zdorovyak A, Yalonetsky S, Kapeliovich M, et al. Hyponatremia and long-term mortality in survivors of acute ST-elevation myocardial infarction. Arch Intern Med. 2006;166(7):781–78.