

# CHANGE IN SERUM PHOSPHORUS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Khadiza Diluara<sup>1\*</sup>, Mohammad Taufiq-Ul-Alam<sup>2</sup>, Nazla Shamsuddoha<sup>3</sup>,  
Ananna Sarker<sup>4</sup>

## OPEN ACCESS

## ABSTRACT

### Reviewed by

**Mohammad Rafiqul Islam**

Mymensingh Medical College & Hospital  
Mymensingh, Bangladesh.

**Al Aharama Begum**

Medical College for Women & Hospital  
Dhaka, Bangladesh.

### \* Correspondence

Khadiza Diluara

Email: [drkhadizadiluara56@gmail.com](mailto:drkhadizadiluara56@gmail.com),

Orcid id: <https://orcid.org/0009-0009-1601-6895>

**Received:** 07.05.2026

**Accepted:** 09.06.2026

**Published:** July 2026

**Background:** Among the leading cause of death worldwide is Acute myocardial infarction (AMI). Disturbances in serum phosphorus level may occur during AMI, however this correlation has not been sufficiently explored. **Objective:** To evaluate phosphorus level in serum in patients with AMI and compare them with healthy individuals. **Materials and Method:** This cross-sectional, analytical study was carried out in the Biochemistry Department of Mymensingh Medical College, in collaboration with the Cardiology Department of Mymensingh Medical College Hospital, starting from July 2021 to June 2022. One hundred participants were included, consisting of 50 AMI patients and 50 apparently healthy controls. Colorimetric method using a biochemistry analyzer was used to measure phosphorus level in collected serum. Statistical analysis was performed using SPSS version 21, with  $p < 0.05$  considered significant. **Results:** Average serum phosphorus concentrations was markedly higher in AMI patients ( $6.12 \pm 1.34$  mg/dL) compared with controls ( $3.23 \pm 0.46$  mg/dL), demonstrating a highly significant difference ( $p < 0.001$ ). **Conclusion:** Study individuals diagnosed with acute myocardial infarction (AMI) exhibited elevated serum phosphorus concentrations compared with healthy subjects. Further large scale studies are recommended to clarify the effects of phosphorus with AMI.

**Keywords:** Serum phosphorus, Acute myocardial infarction, Electrolyte, Analysis, Colorimetry, Early diagnosis, Prevention.

## INTRODUCTION

### Cite this article:

Diluara K, Alam MTU, Shamsuddoha N, Sarker A. Changes in serum phosphorus in patients with acute myocardial infarction. J Med Coll Women Hosp. 2026; 22(2): 206-210.

Coronary artery disease is the foremost cause of mortality globally and commonly presents as angina and acute coronary syndromes<sup>1</sup>. Acute myocardial infarction tends to occur when prolonged myocardial ischemia results in irreversible damage to cardiac muscle tissue. Ischemia develops when oxygen supply to the myocardium becomes insufficient to meet metabolic demands, often due to impaired coronary blood flow<sup>2</sup>.

Phosphorus (P) is an essential element present in the body mainly as inorganic phosphate in extracellular fluid and as organic compounds within cells. It is necessary for maintaining energy metabolism and reactions, cellular structure, glycolysis, and oxidative phosphorylation. Phosphorus level in plasma exhibit circadian rhythm. The plasma Phosphorus levels are highest in case of infants ( $4.5$  to  $8.3$  mg/dL) and reduce with age with adult values in the range of  $2.5$  to  $4.5$  mg/dL. The homeostasis of phosphorus is maintained by hormones like parathormone, calcitonin and calcitriol as well as by fibroblast growth factor- 23 and  $\alpha$ -klotho<sup>3</sup>.

1\* Department of Biochemistry, President Abdul Hamid Medical College; Bangladesh.

Email: [drkhadizadiluara56@gmail.com](mailto:drkhadizadiluara56@gmail.com), Orcid id: <https://orcid.org/0009-0009-1601-6895>,

[Corresponding Author]

2 Department of Biochemistry, Shaheed Syed Nazrul Islam Medical College, Kishoreganj, Bangladesh.

3 Department of Pharmacology & Therapeutics, Medical College for Women and Hospital, Dhaka, Bangladesh.

4 Department of Biochemistry, President Abdul Hamid Medical College, Kishoreganj, Bangladesh.

Latest research has noted that a reduction in  $\alpha$ -klotho concentration, a rise in fibroblast growth factor and high phosphorus levels aggravate vascular calcification. Raised phosphate concentrations have been linked to calcifications in vessels, increased stiffness in the arterial wall, and adverse cardiovascular outcomes<sup>4</sup>. Also phosphate has been found to cause vascular calcification and apoptosis in time-dependent manner<sup>5</sup>. Mortality in Myocardial Infarction (MI) patients has been associated with high serum levels of phosphorus, since vascular pathologies noted to be promoted by phosphorus level rise<sup>6</sup>.

Previous studies have noted abnormal metabolism of minerals participate in development of cardiovascular disease and demise in case of those suffering from chronic kidney disease<sup>7,8</sup>. Low phosphorus levels have been linked to ventricular arrhythmia<sup>9</sup>. Very few data exist in this country in regards to the phosphorus levels to Acute Myocardial Infarction (AMI). It is necessary to build on this data since studies have found a significant role of phosphorus levels in vascular calcification, cardiovascular disease, and development of MI<sup>4,6</sup>. Therefore, to develop awareness, drawing focus on this important electrolyte, as well as to contribute to the growing body of research on this topic this study was undertaken to assess the serum phosphorus levels in AMI patients and to compare them to the phosphorus levels in normal healthy individuals.

## MATERIALS AND METHOD

This cross sectional analytical study was undertaken following approval from the Institutional Review Board. The study took place in Biochemistry Department of Mymensingh Medical College, with samples collected from the Cardiology Department of Mymensingh Medical College Hospital, Bangladesh, between July 2021 and June 2022.

A sum of 100 individuals aged 40 years–65 years were included, comprising 50 patients who were diagnosed case of AMI (case group) and 50 apparently healthy individuals (control group). Diagnosis of AMI was based on clinical presentation, physical findings, electrocardiographic changes, and cardiac biomarkers levels which is in accordance to the definition of the condition accepted universally<sup>10</sup>.

Participants who have structural cardiac diseases other than AMI, severe infection, kidney failure, carcinoma, recent gross surgery, or other serious systemic illnesses were excluded. Documented agreement was taken from all participants.

Colorimetric method with an biochemistry analyzer was used to calculate Serum phosphorus level. Blood was collected from AMI patients on the first day of hospitalization maintaining all aseptic measures and was then sent from serum analysis to obtain the serum phosphorus level values. Similarly, blood was collected from the control group and serum phosphorus level was obtained.

Data were recorded and SPSS version 21 was applied for Statistical analysis. Data were presented in Mean  $\pm$  SD and bar graph was used to show the comparison between the groups.

## RESULTS

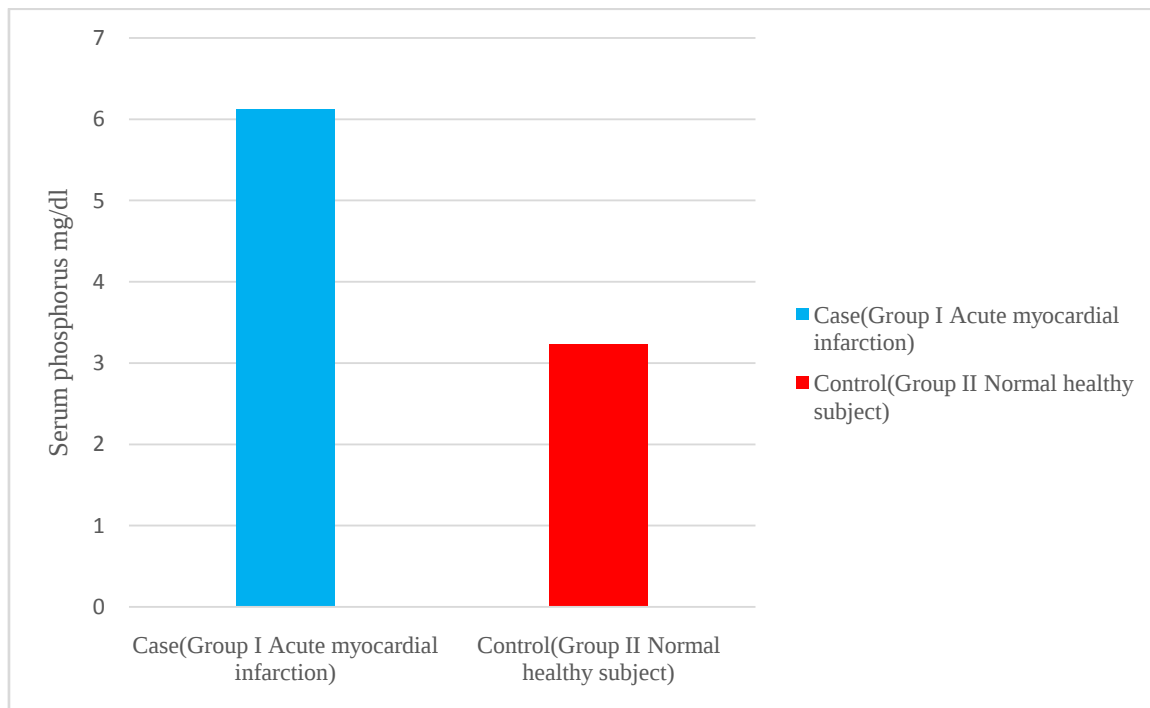
Among the participants, 84% were male and 16% were female. The average age of the case group was  $52.65 \pm 4.93$  years, whereas the control individuals was  $51.15 \pm 6.32$  years. Mean age of study group was  $52.65 \pm 4.93$  for group- I and  $51.15 \pm 6.32$  for group- II. Mean BMI was  $24.31 \pm 1.14$  for group I and  $24.23 \pm 1.20$  for group II. BMI of two groups were statistically insignificant. Some clinical parameters are shown in table 1.

**Table1: Demographic characteristics of the study subjects**

| Variables                | Group I<br>(case)<br>Mean $\pm$ SD | Group II<br>(control)<br>Mean $\pm$ SD | <i>p</i> -value |
|--------------------------|------------------------------------|----------------------------------------|-----------------|
| Age(years)               | 52.65 $\pm$ 4.93                   | 51.15 $\pm$ 6.32                       | 0.165 *         |
| BMI (kg/m <sup>2</sup> ) | 24.31 $\pm$ 1.14                   | 24.23 $\pm$ 1.20                       | 0.753*          |

Comparison of data contrast revealed that the differences between both age and BMI of AMI patients and healthy individuals was not significant ( $p > 0.05$ ) (Table 1).

Serum phosphorus was estimated. Statistical analysis was done according to data. However, serum phosphorus concentrations were substantially elevated in AMI patients in comparison with healthy controls. The mean  $\pm$  SD values were 6.12  $\pm$  1.34 mg/dL in the case group and 3.23  $\pm$  0.46 mg/dL in the control group, revealing a markedly significant difference ( $p < 0.001$ ) as displayed in Figure 1.

**Figure 1: Comparison of mean serum phosphorus levels in the study population.**

## DISCUSSION

Myocardial infarction represents a severe manifestation of acute coronary syndrome and a major contributor to global mortality. Myocardial necrosis, in a clinical setting, consistent with myocardial ischemia may be defined as myocardial infarction. Atherosclerotic plaque rupture, fissuring, erosion or a combination superimposed intra coronary thrombosis are usually the cause of acute ischemia, but not always. This may be related with increased risk of cardiac death and myonecrosis<sup>11</sup>.

The results of the current study indicate that phosphorus levels in supplied serum are significantly higher in individuals with AMI compared with controls. The mean  $\pm$  SD values of serum phosphorus were 6.21  $\pm$  1.34 mg/dl and 3.23  $\pm$  0.46 mg/dl in study group and healthy individuals respectively.

A highly significant ( $p < 0.001$ ) variation in average phosphorous levels was identified between the two groups, supporting the findings of Kendrick and Chonchol, Mathew et al., Ellamand Chico, Ayus et al.<sup>4,12-14</sup>.

In Kendrick and Chonchol's study<sup>4</sup>, serum phosphorus level was markedly higher in AMI patients than that of healthy groups ( $p < 0.001$ ). Hyperphosphatemia, particularly when related with an increased calcium-phosphorous product, plays a critical role in vascular calcification and unfavorable cardiovascular outcome. Excess phosphate facilitates pathological mineral deposition within vascular structures as well as cardiac tissues, including the myocardium and heart valves. One important mechanisms involves the phenotypic transformation of vascular smooth muscles cells (VSMCs) from their normal contractile state into osteochondrogenic-like cells, which actively promote extracellular matrix mineralization. Non-atheromatous vascular calcification contributes to arterial stiffness, elevating pulse wave velocity and cardiac workload, and thereby increasing the risk of acute myocardial infarction<sup>4</sup>.

In comparison to the result of the current study, previous investigations have reported differing observations. Geerse et al.<sup>15</sup> and Kzelden et al.<sup>16</sup> described reduced serum phosphorus concentrations in patients experiencing myocardial infarction. Their findings indicated that decreased phosphorous levels may be linked to heightened risk of acute coronary syndrome. The lower phosphorus levels observed by these studies during myocardial infarction, possibly due to redistribution of phosphate into cells during acute stress and increased energy demands<sup>14,15</sup>. Therefore, the relationship between phosphorus metabolism and AMI remains complex.

## CONCLUSION

Acute myocardial infarction continues to pose a major public health challenge worldwide. The current study reveals markedly raised serum phosphorus levels in case group compared with controls. Monitoring serum phosphorus may offer beneficial information for clinical assessment. Expanded cohort studies are recommended to further clarify the role of phosphorus in the pathophysiology and management of AMI.

## CONFLICT OF INTEREST

There is no conflict of interest.

## REFERENCES

1. Newby, DE & Grubb, NR. Cardiology. In: Ralston, SH, Penman, ID, Strachan, MWJ & Hobson, RP (ed.), *Davidsons Principles and Practice of Medicine*, 23rd edition, Churchill Livingstone Elsevier, Edinburg. 2018.444-5. p
2. Cortan, RS, Kumar, V & Robbins, SL. (eds). *Robbins pathologic basis of disease*. 9th edn, wbsaunders, philadelphia. 2012.
3. Mattoo RL. The Roles of Fibroblast Growth Factor (FGF)-23,  $\alpha$ -Klotho and Furin Protease in Calcium and Phosphate Homeostasis : A Mini-Review. *Indian J Clin Biochem*. 2014;29(1):8-12. doi: 10.1007/s12291-013-0324-1.
4. Kendrick J, Chonchol M. The role of phosphorus in the development and progression of vascular calcification. *Am J Kidney Dis*. 2011;58(5):826-34. doi: 10.1053/j.ajkd.2011.07.020.

5. Lau WL, Festing MH, Giachelli CM. Phosphate and vascular calcification: Emerging role of the sodium-dependent phosphate co-transporter PiT-1. *ThrombHaemost.* 2010;104(3):464-70. doi: 10.1160/TH09-12-0814.
6. Cao W, Li Y, Wen Y, Fang S, Zhao B, Zhang X, et al. Higher serum phosphorus and calcium levels provide prognostic value in patients with acute myocardial infarction. *Front Cardiovasc Med.* 2022;9:929634. doi: 10.3389/fcvm.2022.929634.
7. Gutiérrez OM, Mannstadt M, Isakova T, Rauh-Hain JA, Tamez H, Shah A, et al. Fibroblast growth factor 23 and mortality among patients undergoing hemodialysis. *N Engl J Med.* 2008;359(6):584-92. doi: 10.1056/NEJMoa0706130.
8. Ganesh SK, Stack AG, Levin NW, Hulbert-Shearon T, Port FK. Association of elevated serum PO(4), Ca x PO(4) product, and parathyroid hormone with cardiac mortality risk in chronic hemodialysis patients. *J Am Soc Nephrol.* 2001;12(10):2131-2138. doi: 10.1681/ASN.V12102131.
9. Vaidyanathan D, Venkatesan S, Ramadesikan VK. Serum phosphate in acute myocardial infarction. *Indian J PhysiolPharmacol.* 2000;44(2):225-8.
10. Thygesen K, Alpert JS, White HD; Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction; Jaffe AS, Apple FS, et al. Universal definition of myocardial infarction. *Circulation.* 2007;116(22):2634-53. doi: 10.1161/CIRCULATIONAHA.107.187397.
11. Fuster V, Moreno PR, Fayad ZA, Corti R, Badimon JJ. Atherothrombosis and high-risk plaque: part I: evolving concepts. *J Am Coll Cardiol.* 2005;46(6):937-54. doi: 10.1016/j.jacc.2005.03.074.
12. Mathew S, Tustison KS, Sugatani T, Chaudhary LR, Rifas L, Hruska KA. The mechanism of phosphorus as a cardiovascular risk factor in CKD. *J Am Soc Nephrol.* 2008;19(6):1092-105. doi: 10.1681/ASN.2007070760.
13. Ellam TJ, Chico TJ. Phosphate: the new cholesterol? The role of the phosphate axis in non-uremic vascular disease. *Atherosclerosis.* 2012;220(2):310-8. doi: 10.1016/j.atherosclerosis.2011.09.002.
14. Ayus JC, Mizani MR, Achinger SG, Thadhani R, Go AS, Lee S. Effects of short daily versus conventional hemodialysis on left ventricular hypertrophy and inflammatory markers: a prospective, controlled study. *J Am Soc Nephrol.* 2005;16(9):2778-88. doi: 10.1681/ASN.2005040392.
15. Geerse DA, Bindels AJ, Kuiper MA, Roos AN, Spronk PE, Schultz MJ. Treatment of hypophosphatemia in the intensive care unit: a review. *Crit Care.* 2010;14(4):R147. doi: 10.1186/cc9215.
16. Kjeldsen SE, Moan A, Petrin J, Weder AB, Julius S. Effects of increased arterial epinephrine on insulin, glucose and phosphate. *Blood Press.* 1996;5(1):27-31. doi: 10.3109/08037059609062103.