ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
Index Copernicus Value: 68 . 16
PubMed - National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 5, Issue –3, June - 2020, Page No. : 158 - 163

A comparative study of left ventricular function among acute myocardial infarction patients with normal and low serum phosphate

[1]Kamalesh Kumar Bajia, Resident ,SMS Medical College, Jaipur, Rajasthan, India
[1]Vijaiendre Singh, Resident ,SMS Medical College, Jaipur, Rajasthan, India
[1]Chaina Ram, Resident ,SMS Medical College, Jaipur, Rajasthan, India
[1]Ajay Kumar, Resident ,SMS Medical College, Jaipur, Rajasthan, India
[2]S.Banerjee, Senior Professor, SMS Medical College, Jaipur, Rajasthan, India
[3]Rohit Ishran, Asst Professor, SMS Medical College, Jaipur, Rajasthan, India
Corresponding Author: Vijaiendre Singh, Resident, SMS Medical College, Jaipur, Rajasthan, India

Citation this Article: Kamalesh Kumar Bajia ,Vijaiendre Singh ,Chaina Ram , Ajay Kumar, S. Banerjee, Rohit Ishran, "A comparative study of left ventricular function among acute myocardial infarction patients with normal and low serum phosphate", IJMSIR- June - 2020, Vol – 5, Issue -3, P. No. 158 – 163.
Type of Publication: Original Research Article
Conflicts of Interest: Nil

Abstract

Background: Phosphate depletion causes impaired energy metabolism in myocardium, leading to decreased contractility which leads to myocardial dysfunction and arrhythmias.[13] Severe acute heart failure has been described in several case reports in the presence of severe hypophosphatemia.[14] Hypophosphatemia after cardiac surgery was associated with higher requirements of inotropic support.[15] Correction of hypophosphatemia is associated with improved cardiac output.[16] Hypophosphatemia is a significant predictor of ventricular tachycardia after myocardial infarction[17] and a correlation with arrhythmias has been suggested in septic patients.[18] During correction of hypophosphatemia, phosphate may precipitate with calcium and cause hypocalcemia. It is important to keep in mind that hypocalcemia can negatively influence cardiac function as well.

Materials and methods: 32 eligible acute myocardial infarction patients presented with low serum phosphate levels were included in the study. Eligible acute myocardial infarction patient with normal serum phosphate level reported just next to each acute myocardial infarction patient with hypophosphatemia was taken as control. All Acute Myocardial infarction patients (clinical feature, ECG changes and cardiac biomarkers) with normal and low serum phosphate level were subjected to the inclusion/ exclusion criteria.

Results: Mean left ventricular ejection fraction was significantly low in low serum phosphate acute myocardial infarction patients (32.4%) compare to normal serum phosphate acute myocardial infarction patients (38.3%) The difference of mean ejection fraction of two groups was 5.9% ($p<0.001$).

Conclusions: Based on the findings of our study we concluded that normal phosphate level with acute MI (myocardial infarction) faired better than patients with

acute MI with hypophosphatemia in terms of left ventricular ejection fraction and KILLIP score.

Keywords: Hypophosphatemia, Hypocalcemia , Myocardial Infarction

Introduction

Normal organ function and many metabolic processes are dependent on intracellular as well as on extracellular electrolyte concentrations.

Phosphorus is an essential element, majority (about 80%) is a part of bones and teeth. 9%, 11% and 0.1% is found in skeletal muscle, viscera and extracellular fluid, respectively. [1] Both organic and inorganic phosphate are present in blood. 85% of the inorganic form are free phosphate ions, 10% is bound with proteins and 5% with other minerals.

Phosphorus has an important role in acid-base homeostasis, cellular and subcellular metabolism (e.g. cellular signal transduction, energy exchange) and in the cell structure as a component of the cell membranes. Phosphate is the major intracellular constituent both as a free anion and as a component of numerous compounds like enzymes, structural proteins, high energy stores (ATP, creatine phosphate) and nucleic acids, etc.[2]

Phosphate exists intracellulary at concentration close to those present in extracellular fluids. Normal values of the total serum phosphate level are 0.80 to 1.45 mmol/L (2.5 to 4.5 mg/dl).[2]

Hypophosphatemia is usually categorized as moderate (serum phosphate level of 0.32 to 0.65 mmol/L (1 to 2 mg/dl) or severe (<0.32 mmol/L (<1 mg/dl)). Moderate hypophosphatemia occurs in 2.2–3.1% of hospitalized patients, severe hypophosphatemia in 0.2–0.4% patients.[1,3]

Hypophosphatemia is known to cause generalized defect in cellular energy metabolism because of ATP depletion, a shift from oxidative phosphorylation toward glycolysis and associated tissue or organ dysfunction. [2]

After the occlusion of coronary artery various biochemical changes are observed intracellularly. Depletion of these high energy intracellular phosphates is one of the biochemical change observed following occlusion of coronary artery in experimental models. [4,5]

Depletion of this ion has a role in the genesis of myocardial stunning.[6,7]

Further studies are needed to establish this emerging hypothesis of pathogenic relationship between serum phosphate levels and myocardial performance post MI. Metabolic modification and supporting of ischemic myocardium is a new development in management of acute coronary syndromes in recent times and this knowledge of serum phosphate(serving as an indirect marker of myocardial high energy phosphate status) helps in selecting particular subset of patients who will benefit from appropriate interventions.

The aim of our study is to determine the difference in mean ejection fraction among acute myocardial infraction cases with normal and low serum phosphate level.

Materials and methods

This one year Descriptive type of observational study was carried out among 64 acute myocardial patients admitted in SMS Hospital. Where 32 patients are cases of acute myocardial infarction with low serum phosphate and remaining 32 cases of acute myocardial infarction with normal serum phosphate. Duration of study was one year (April 2016 – March 2017).

All Acute Myocardial infarction patients (clinical feature, ECG changes and cardiac biomarkers) with

normal and low serum phosphate level will be subjected to the inclusion/ exclusion criteria.

The resulting study group will be subjected to detailed history and examination followed by a ECG, 2D Echocardiography, Serum phosphate level.

The patients who satisfied the selection criteria were enrolled in study.

Inclusion Criteria

All patients with acute myocardial infarction diagnosed by clinical history, ECG changes and cardiac biomarkers with normal and low phosphate.

Exclusion Criteria

1. Patients with pre-existing cardiac failure.
2. Patient with evidence of old MI.
3. Critically ill patients.
4. Patients with deranged RFTs
5. Patients refused for consent

Sampling Procedure

32 eligible acute myocardial infarction came with low serum phosphate level would be included in the study. Eligible acute myocardial infarction came with normal serum phosphate level reported just next to each acute myocardial infarction with hypophosphatemia case would be taken for control group.

Statistical Analysis

The collected data was transformed into variables, coded and entered in Microsoft Excel sheet. Data was analyzed and statistically evaluated using SPSS-PC-17 version. Continuous data was summarized in form of Mean ± Standard Deviation. Difference in means was analyzed using student's 't' test. Count data was summarized in form of proportion. Difference in proportion was analyzed using Chi Square Test. The level of significance was kept 95% for all statistical analysis.

Results

Subjects in both groups were age matched with no significant difference (p=0.080), mean age of acute myocardial infarction patients with low serum phosphate and normal serum phosphate was 54 and 60 respectively with standard deviation of 12. (Table 1). Subjects in both groups were also sex matched with no significant difference (p=0.404), male preponderance was noted with 71.9% of patients being male. The male to female ratio in patients of acute myocardial infarction with low serum phosphate group and normal serum phosphate was 3.57 and 1.90 respectively. Diabetes and hypertension are modifiable risk factor associated with acute MI. The prevalence of diabetes in acute myocardial infarction patients with low serum phosphate and normal serum phosphate was 21.9% and 28.1% respectively which was statistically not significant (p=0.773). Prevalence of HTN in acute myocardial infarction patients with low serum phosphate and normal serum phosphate was 18.7% and 34.4% respectively which was statistically not significant (P = 0.258)

Table 1: Distribution of study subjects.

Variable	Low phosphate (n=32)	Normal phosphate (n=32)	P value
Age (yrs)	54.6	60.1	0.080
Male (%)	78.1	65.6	0.404
Diabeties (%)	21.9	28.1	0.773
Hypertension (%)	18.7	34.4	0.258

Table 2: Comparison of LVF and KILLIP score of study groups.

Group	Low phosphate (n=32)	Normal phosphate (n=32)	P value
Clinical LVF (%)	37.5	12.5	0.043
KILLIP score >1(n)	12	4	0.043

In this study clinical LVF present in 37.5% of low phosphate and 12.5% of normal phosphate acute myocardial infarction patients. KILLIP score was >1 in 12 low phosphate and 4 normal phosphate acute myocardial infarction patients. The difference in both groups for LVF and KILLIP score were statistically significant found (Table 2).

Table 3: Comparison of mean Ejection fraction (%) of study groups.

Group	N	Mean	Std. deviation
Low phosphate	32	32.4	5.7
Normal phosphate	32	38.3	7.3

t = -3.587 with 1 degrees of freedom; P < 0.001 (S)

In this study mean ejection fraction for low phosphate and normal phosphate acute myocardial infarction patients were 32.4 and 38.3 respectively. The difference in both groups for ejection fraction was statistically significant found (Table 3) (Figure 1).

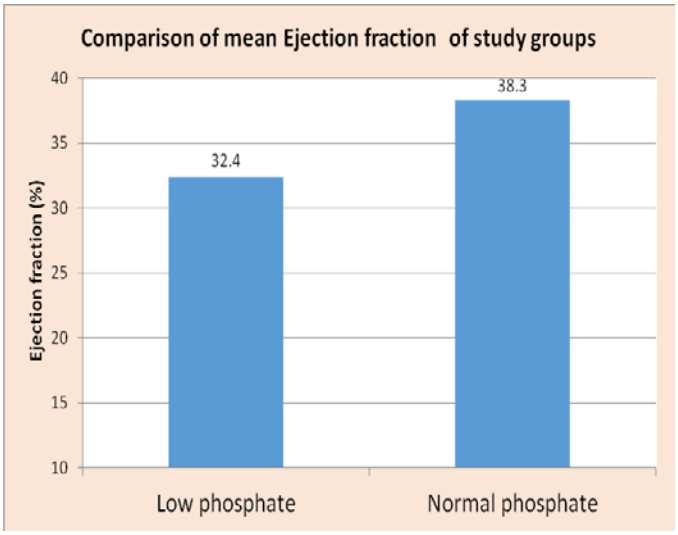

Figure 1: Low and normal phosphate acute myocardial infarction patients Ejection Fraction comparison

Table 4: Duration of hospital stay in low and normal phosphate patients in different age groups.

Age group (years) (no. of patients)	Low phosphate (no. of patients)	Normal phosphate (no. of patients)	P value
≤ 30 (1)	5 ± 0 (1)	0	-
31-50 (18)	4.75 ± 1.06 (12)	5 ± 1.1 (6)	0.646
51 – 70 (36)	7.38 ± 2.6 (16)	5.2 ± 1.24 (20)	0.002
> 70 (9)	6 ± 3.46 (3)	6.83 ± 2.32 (6)	0.675
Total (64)	6.19 ± 2.46 (32)	5.47 ± 1.57 (32)	0.168

In this study average hospital stay for low and normal serum phosphate acute myocardial infarction patients group were 6.19 and 5.47 days respectively The difference in both groups for ejection fraction was statistically significant found (Table 4).

Discussion

Phosphate depletion causes impaired energy metabolism in myocardium, leading to decreased contractility.[13] . In this study we found that patients with low serum phosphate levels (37.5%) had high risk of clinical LVF and KILLIP Score more than 1 as compared to normal serum phosphate level patients(12.5%).The difference was significant (p=0.043). Indicating that hypophosphatemia is associated with increased risk of LVF and higher KILLIP score. Similarly, Study done by Vaidyanathan D. et al [9] found a significant association between LVF , KILLIP Score and serum phosphate level in acute MI patients. He found clinical LVF and KILLIP Score more than 1 was present in 54% of low serum phosphate patients and 18% of normal serum phosphate patients (p<0.05).

In our study we found that patients with low serum phosphate levels had low ejection fraction compared to normal serum phosphate level patients. In this study mean ejection fraction in low serum phosphate patients was 32.4% with std deviation of 5.7 and in normal serum phosphate patients, mean ejection fraction was 38.3% with std deviation of 7.3. The difference of mean ejection fraction of two groups was 5.9% which was significant (p<0.001). In study done by Vaidyanathan D et al.[9] found a significant relation with serum phosphate and LVEF in acute MI patients (p<0.001).They found mean ejection fraction in low and normal serum phosphate patients was 35% and 53% respectively. The difference of mean ejection fraction was 18%.

In this study we compared the duration of hospital stay of acute myocardial infarction patients with low and normal serum phosphate. We found that average hospital stay in low and normal serum phosphate acute myocardial infarction patients was 6.19 and 5.47 days respectively which was not significant (p=0.168). However, Acute myocardial infarction patients with low serum phosphate had significantly long hospital stay in age group 51-70yrs compared to normal phosphate patients which was significant (p=0.002) . No similar observation was recorded by other author.

Conclusion

Based on the findings of our study we concluded that acute MI patients with normal phosphate levels faired better than patients with acute MI with hypophosphatemia in terms of left ventricular ejection fraction and KILLIP score.

References

1. Gaasbeek A, Meinders AE: Hypophosphatemia: an update on its etiology and treatment. Am J Med 2005, 118:1094–1101.
2. Harrison’s principals’of internal medicine,ed 19 chapter-423 2458-2460
3. Amanzadeh J, Reilly RF Jr (2006) Hypophosphatemia: an evidence-based approach to its clinical consequences and management. Nat Clin Pract Nephrol 2: 136-148.
4. Jennings RB, Hawkins HK, Lowe JE et al. Relation between high energy phosphate and lethal in myocardial ischemia in the dog. Am J Pathol 1978; 92: 187-214.
5. Komarek J, Stransova V et al. Creatinei phosphate as a drug in acute myocardial infarction. Cas Lek Cesk 1992; 24: 131(8): 248.
6. Heusch G, Schulz R. Hibernating myocardium a review. J Mol Cell Cardiol 1996; 28: 2359-2372.
7. Aoki T, Nishikawa H, Motayasu M et al. A case of stunned myocardium with marked 99mTe-PYP accumulation. Kuku Igaku 1993; 30: 61

8. The GUSTO investigators: An international randomised trial comparing four thrombolytic strategies for acute myocardial infarction. N Engl Med 1993; 329: 673-682.
9. Vaidyanathan D et al. Serum phosphate in acute myocardial infarction. Indian J Physiology Pharmacolgy 2000;44(2):225-228.
10. Yaroslavsky A, Blum M, Peer G et al. Serum phosphate shift in acute myocardial infarction. Am Heart J 1982; 104: 884-885.
11. Fuller TJ et al. (1976) Reversible changes of the muscle cell in experimental phosphorus deficiency. J Clin Invest 57: 1019–1024.
12. Fuller TJ et al. (1978) Reversible depression in myocardial performance in dogs with experimental phosphorus deficiency. J Clin Invest 62: 1194–1200.
13. Kubler W, Kate 8Am. Mechanism of early pump failure of the ischemic heart: possible role of adenosine depletion and inorganic phosphate accumulation. Am J Cardiol 1977; 40: 467.
14. Darsee JR, Nutter DO: Reversible severe congestive cardiomyopathy in three cases of hypophosphatemia. Ann Intern Med 89 : 867 – 870, 1978.
15. Cohen J, Kogan A, Sahar G, Lev S, Vidne B, Singer P: Hypophosphatemia following open heart surgery: incidence and consequences. Eur J Cardiothorac Surg 2004, 26:306–310.
16. O'Connor LR, Wheeler WS, Bethune JE (1977) Effect of hypophosphatemia on myocardial performance in man. N Engl J Med 297:901-903.
17. Ognibene A. Cinigilo R, Grefenstein A. et al. Ventricular tachycardia in acute myocardial infarction: the role of hypophosphatemia. South Med J 1994; 65-69
18. Bollaert PE et al. Hemodynamic and metabolic effects of rapid correction of hypophosphatemia in patients with septic shock. Chest 107:1698-1701, 1995.