Study of evaluation of lipid profile and phosphorus levels in acute myocardial infarction patients

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Abstract

Introduction: Myocardial infarction is the leading cause of mortality and morbidity in present days. Sometimes, especially in early period, features of MI may not be fully developed. Like chest pain is the most common presenting symptom in majority of the patients with MI. Diagnosis is based on ECG findings and result of Serum cardiac markers.

Objectives: The objective of the study is to evaluate level of serum phosphorus and lipid profile in MI patients.

Materials and Method: This study Includes 70 patients of Acute Myocardial infarction of age group 30-65 years along with 70 age and sex matched healthy controls. Fasting blood samples was taken from all participants to measure serum phosphorus and lipid profile then comparison was done between patients group and control group to see the difference of significant by calculating P- value.

Results: The mean concentration of serum Phosphorus(mg/dl) in MI patients is 3.97 ±1.51 while in control group it is 2.4±0.4. Similarly the mean concentration of S.Cholesterol(mg/dl), S.Triglyceride(mg/dl), S.HDL(mg/dl), S.LDL(mg/dl), S.VLDL(mg/dl) is 169.9±24.4, 119.9±36.9, 41.0±9.57, 108.8±23.4, 20.3±7.37 in control group and in MI patients it is 187.3±40.3, 140.4±69.11, 33.6±10.6, 129.1±30.2, 27.0±16.4 respectively.

Conclusion: serum phosphorus level might be used as a possible marker for the diagnosis of the risk of similarity to lipid profile and/or other risk factors alteration of the serum phosphorus level is recently being considered as one of the foretelling markers for the severity of cardiovascular diseases.

Keywords: Myocardial infarction, Phosphorus, Lipid profile

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Introduction

The field of mineral metabolism is at present in phase of rapid expansion. It has become apparent that not only proteins, fat and carbohydrates but also minerals are essential to life. Now, the significance of traces not only of vitamins and other active organic substances, but also of minerals is under intensive investigation. Coronary artery diseases (stable angina and unstable angina and myocardial infraction) have been progressed considerably in its diagnosis and treatment. However myocardial infarction still remains a major public health problem in developing countries. Common consequence of aging leads to the development of atherosclerosis in intimal lesions, due to vascular calcification.(1) Myocardial energy stores in the vital constituents of the intracellular anion called Phosphate/phosphorus. Occlusion in coronary artery diseases shows depletion of high energy intramyocitic phosphate at early stage in experimental animals.(2,3) In cardiovascular events the most important role in structural support of the body and providing phosphate for the extracellular and intracellular pool Inorganic phosphorus (Pi) plays an important role. Most of the phosphate in cellular is in soft tissue. Phosphate is the major intracellular anion and is a vital constituent of myocardial energy stores. It is essential for multiple and diverse biological functions, including cellular signal transduction, mineral metabolism, and energy exchange. Vascular calcification is much more complex process than originally was thought. It certainly cannot be explained fully by hyperphosphatemia alone, but phosphate, more specifically intracellular phosphate play a major role in the genesis of vascular calcification, particularly in the presence of ionized calcium. Vascular calcification is a highly regulated process involving inductive and inhibitory mechanisms. These mechanisms include: (1) loss of mineral inhibiting factors; (2) induction of bone formation; (3) cell death; and (4) circulating nucleation complexes (i.e., aggregates of calcium phosphate and proteins released from remodelling bone that may initiate ectopic mineralization). Abnormalities in mineral metabolism that enhance the calcium X phosphate product (Ca x P) may further exacerbate vascular calcification initiated by any of these mechanisms. Recent evidence implicates elevated phosphate as a major inductive factor for vascular calcification and osteopontin as an inducible inhibitor of vascular calcification, and our current understanding of their mechanisms of action.(4,5)

Materials and Method

This study was conducted at Department of biochemistry at Smt. NHL municipal medical college,

and attached V.S General Hospital, Ahmedabad, Gujarat, India.

Duration of the study: 1 year (2015-2016)

Study includes those patients who visited our hospital with first attack of acute coronary artery disease. Acute coronary artery disease is a unifying term, representing a common end result acute myocardial ischemia. It encompasses acute MI (resulting in ST elevation or non ST elevation) and unstable angina. Non ST elevation MI (NSTEM1) is established if a patient with clinical features of unstable angina develops evidence of myocardial necrosis as reflected in elevated cardiac biomarkers. Unstable angina is defined as angina pectoris or equivalent ischemic discomfort with at least one of three features: It occurs at rest (or with minimal exertion) usually lasting >10 minutes. It is severe and of new onset. It occurs with a crescendo pattern (i.e. distinctly more severe, prolonged or frequent than previously)

Inclusion criteria for cases: Fulfill the definition of acute coronary artery disease, Non-smoker, Non-alcoholic, Those who are not taking lipid-lowering drugs, Age group of 30-65 years of both sexes, informed written consent given by subject or their guardians.

Exclusion criteria for cases: Patients without written consent to participate in the studies, not fulfilling the definition of acute coronary syndrome, Patients suffering from diabetes, renal disorders were excluded from the study. Controls are apparently healthy persons selected from the bystanders and patients in Smt. NHL Municipal College, and attached VS General Hospital, Ahmedabad, Gujarat, India.

Materials and Method

Study was conducted in 70 hospitalized, diagnosed case of acute myocardial infraction of both sexes of age group 30- 65 years and 70 healthy subjects within the same age group as case and control respectively.

The following blood parameters were compared.

1. Estimation of Phosphorus:**(9)** By UV-End point method. The quantitative estimation of Phosphorus in human sera or plasma is done using Abbott Fully autoanalyzer.
2. Estimation of lipid profile:
   a) Total Cholesterol:**(10)** Enzymatic colorimetric method. Serum cholesterol was determined by end point estimation using cholesterol oxidases and peroxidase.
   b) Serum Triglyceride:**(11)** Enzymatic colorimetric method.
   c) LDL Cholesterol:(12) Direct enzymatic method
   d) HDL Cholesterol:(13) Direct enzymatic method.
   e) Estimation of VLDL cholesterol VLDL cholesterol is estimated from the total amount of Triacylglycerol. VLDL = [Triacylglycerol] /5

Statistical Analysis: All data analysis was done using Microsoft Excel and the Statistical software MedCalC version 11.5.0. Mean ± Standard deviation calculated. Results were analysed statistically for significance by Independent 't' test and chi square test. And Pearson correlation 'r' test (correlation coefficient test) was done to assess the relation of Serum phosphorus with various lipid profile parameters. Cohen's ES standards were used to calculate the p values, at, p-value <0.05, results were considered significant.

Results and Discussion

Table 1: Gender wise comparison of subjects in study

Group	Gender	Number(n)
Patients	Male	51
	Female	19
Controls	Male	60
	Female	10

Table 2: Comparison of clinical parameters in controls and patients

Parameter	Reference range	Controls	Patients	P-value
S.Phosphorus(Mg/dl)	2.5-4.5	2.4 ± 0.4	3.97±1.51	<0.01
Total cholesterol(Mg/dl)	150-240	169.9±24.4	187.3±40.3	<0.01
S.Triglyceride(Mg/dl)	<200	119.9±36.9	140.4±69.11	<0.01
S.HDL(Mg/dl)	>40	41.0±9.57	33.6±10.6	<0.01
S.LDL(Mg/dl)	<130	108.8±23.4	129.1±30.2	<0.01
S.VLDL(Mg/dl)	<35	20.3±7.37	27.0±16.4	<0.01

Graph 1: Comparison of clinical parameters in controls and patients

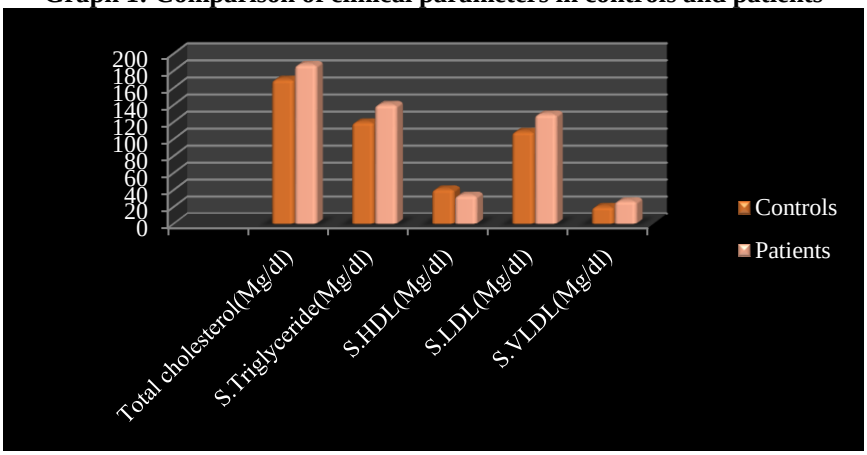
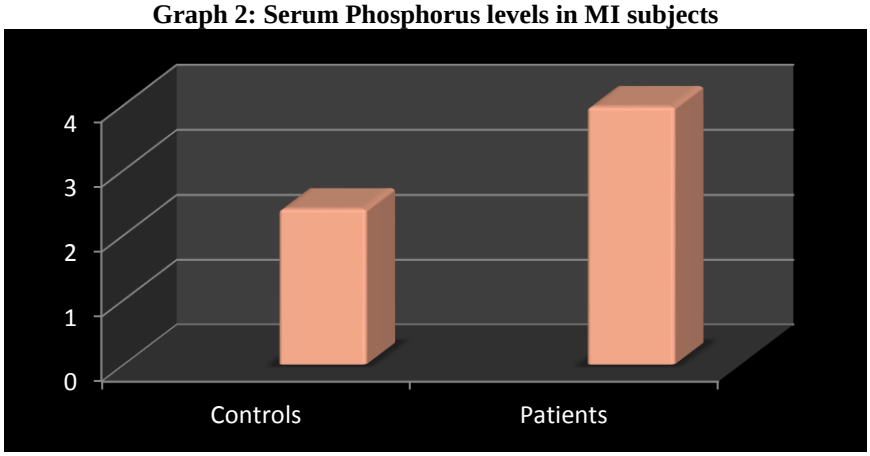


Table 3: Serum Phosphorus levels in MI subjects

Controls	Patients	p-Values
2.4 mg/dl	3.97±1.51 mg/dl	<0.0001

Graph 2: Serum Phosphorus levels in MI subjects



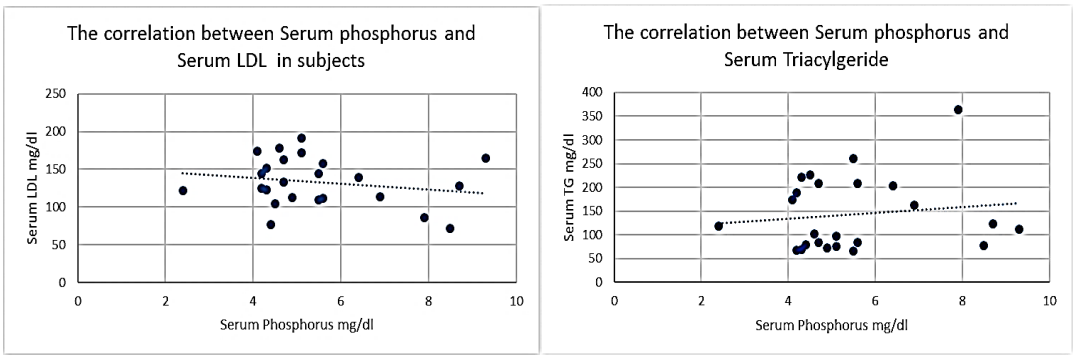


Fig. 1: Correlation of parameters with Serum Phosphorus

There has been considerable progress in the diagnosis and treatment of coronary artery disease (stable angina, unstable angina and myocardial infarction) however it still remains a major public health problem in developed countries, and it has become a major problem in developing countries.

Following myocardial infarction (MI) some proteins and enzymes labelled as cardiac markers (CPK-MB/ Troponin-T & I) are released in to the circulation in large quantity from necrotic heart muscle. These markers like CPK-MB, Troponin-T, Troponin-I and myoglobin, have specific temporal profile in relation to MI. The estimation of these markers requires costly reagents and equipment for their estimation. Assessment of CAD as well involves invasive procedure like angiography. In the rural areas with

dearth of good laboratory and healthcare services, it becomes difficult to measure these markers. The serum phosphorus level is recently being considered as one of the foretelling markers for the severity of cardiovascular diseases (CVD).[6,7]

Inorganic phosphorus (Pi) plays an important role in the structural support of the body and providing phosphate for the extracellular and intracellular pool.[8,9]

In soft tissue, most phosphate is cellular. Although both inorganic and organic phosphate are present in cells, most is organic and incorporated into nucleic acids, phospholipids, phosphoproteins, and "high-energy" compounds involved in metabolism. Adenosine triphosphate (ATP) and other phosphates, such as creatine phosphate, are involved in many energy-intensive physiological functions, such as muscle contractility, neurological functions and electrolyte transport. Phosphate is also an essential element of cyclic nucleotides (such as cyclic AMP) and NADP.[10]

In our study, Table 1 is showing the gender wise distribution of included subjects, each group consisting of 70 peoples of which control group consist of 60 male and 10 female, while Patients consists of 59 male and 19 female.

Table 2 is showing comparisons of clinical parameters in subjects from which Serum Phosphorus, S. HDL and S.LDL is showing significant result (i.e. p-value<0.0001). So, from our study is correlating well with the increase in S. Phosphorus level with decrease in S.HDL and increase in S.LDL which is one of the factors causing MI in patients.

In Table 3, it is showing the values of S. phosphorous in controls and patients.

Then, Figures is showing the Pearson correlation 'r' test (correlation coefficient test) was done to assess the relation of Serum phosphorus with various lipid profile parameters. In that Figure 1 showing correlation between Serum phosphorus and S.LDL having (r= -0.0240) mild correlation and between Serum phosphorus and S.TG having (r= -0.1049) mild correlation.

Conclusion

From Our study we would like to conclude that serum phosphorus level might be used as a possible marker for the diagnosis of the risk of similarity to lipid profile and/or other risk factors like alteration of the serum phosphorus level is recently being considered as one of the foretelling markers for the severity of cardiovascular diseases.

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