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Study to assess role of serum magnesium in acute myocardial infarction cases admitted at RVM Hospital, Siddipet district, Telangana State

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Abstract

Introduction: Acute myocardial infarction is the most important consequence of coronary artery disease and it can lead to complications such as ventricular arrhythmia and congestive heart failure.

Materials and Methods: 50 patients with acute myocardial infarction admitted in department of medicine of RVM Hospital, Siddipet District, Telangana state, during the period of 2019 to 2020 and compared with 10 healthy controls.

Discussion: The percentage of hypomagnesemic acute myocardial infarction patients in present study is 30%. Prophylactic administration of the intravenous magnesium sulphate may be considered in all cases of Acute Myocardial infarction as an adjuvant to thrombolytic therapy.

Keywords: Acute myocardial infarction, hypertension, alcoholism, stress, magnesium

Introduction

Acute myocardial infarction is the most important sequelae of coronary artery disease. Major risk factors that contribute to acute myocardial infarction are smoking, alcoholism, obesity, stress, increasing age, lack of exercise, hypertension, dyslipidaemia, diabetes and it can lead to complications such as ventricular arrhythmia and congestive heart failure ^[1]. Magnesium is an essential micronutrient for human beings and plays an important role in normal myocardial physiology. Its possible site of action includes vascular smooth muscle, platelets, and myocardial cells ^[2]. Magnesium has a lot of therapeutic potential. But it is less evident whether it is useful in patients with congestive heart failure or acute myocardial infarction. Magnesium depletion can induce hyperlipidaemias and subsequently atherogenic deposits in coronary arteries leading to atherosclerosis. Normal magnesium content in human body is around 1.7 and 2.2 meq/L. Hypomagnesemia is caused by inadequate intake of magnesium, chronic diarrhoea, malabsorption, chronic stress, alcoholism and using medication such as diuretic ^[3]. The current study was conducted to observe the changes in magnesium levels in patients with acute myocardial infarction and to find the prognostic value of serum magnesium levels with relation to the complications. The objectives of this study are 1. To know whether there is any change in the serum magnesium level in patients with acute myocardial infarction 2. Serum magnesium levels and its relation with the complications of acute myocardial infarction like ventricular arrhythmias, supraventricular arrhythmias, left ventricular failure, cardiogenic shock, bundle branch block and to compare the serum magnesium levels in acute myocardial infarction with normal serum magnesium.

Methodology

Hospital based case control study conducted with 50 patients with acute myocardial infarction admitted in department of medicine of RVM Hospital, Siddipet District, Telangana state, during the period of 2019 to 2020 and compared with 10 healthy controls.

Inclusion criteria

Patients diagnosed with acute myocardial infarction based on WHO criteria.

Exclusion criteria

Patients with history of alcoholism, cirrhosis, carcinoma, chronic diarrhoea, pregnancy, rheumatoid arthritis, renal failure, patients on diuretic therapy, patients on magnesium

containing compounds like laxatives, antacids and patients presenting to the hospital later than 24 hours of the event. All the study participants were informed about the nature and purpose of the study and informed and written consent was taken from subjects and their family members. Institutional ethical committee clearance was obtained prior to the study.

Statistical analysis

Data was spread in Microsoft excel and represented using appropriate charts and graphs. Statistical analysis carried out with SPSS software version 21.0. The association between variables was done by chi-square test/ fisher's exact test for categorical data. "*p*" values <0.05 considered statistically significant.

Results

Table 1: Showing age distribution among the study participants

Age (In years)	No. of patients with AMI
≤ 40	6
41-50	15
51-60	17
61-70	12

More number of patients in this study group belong to the age of 51-60 years and very minimum of six patients are under 40 years age group.

Table 2: Showing gender distribution among the study participants

Sex	No. of patients with AMI
Male	33
Female	17

Male predominance is observed in this study where out of 50 patients 33 are male and only 17 are females.

Table 3: Showing risk factors associated with acute myocardial infarction

Risk factor	No. of patients with AMI
Alcohol	28
Dyslipidemia	30
Hypertension	26
Diabetes	22
Smoking	35

Smoking, Dyslipidaemia and alcohol are major risk factors observed in this study participants as major risk factors followed by hypertension and diabetes.

Table 4: Number of cases and controls with hypomagnesemia and normomagnesemia

	Cases	Controls
Normal	31	10
Hypomagnesemia	19	0

There is significant association of hypomagnesemia with cases of AMI as *p* value is 0.043 (<0.05)

Table 5: Number of AMI cases with arrhythmias

	Normal	Hypomagnesemia
Total cases	31	19
Arrhythmia cases	2	6

There is significant association of hypomagnesemia with arrhythmias in cases of AMI as *p* value is 0.019

Table 6: Number of AMI cases with heart failure

	Normal	Hypomagnesemia
Total cases	31	19
Heart block cases	1	5

There is a significant association of hypomagnesemia with heart failure in cases of AMI as *p* value is 0.034.

Table 7: Number of AMI cases with heart block

	Normal	Hypomagnesemia
Total cases	31	19
Heart failure cases	0	4

There is significant association of hypomagnesemia with heart block in cases of AMI as *p* value is 0.033

Table 8: Number of AMI cases declared death

	Normal	Hypomagnesemia
Total cases	31	19
Death	0	5

There are five deaths reported in AMI patients with hypomagnesemia than in patients with normal serum magnesium levels. There is statistically no significant association seen between hypomagnesemia with death in cases of AMI as *p* value is 0.094

Table 9: Number of AMI cases with complications

	Cases without complications	Cases with complications
Normal	29	2
Hypomagnesemia	4	15

There is significant association of hypomagnesemia with complications in cases of AMI as *p* value is <0.044.

Discussion

Magnesium influences causation of AMI and also its sequelae like arrhythmias. It plays an important role in the pathogenesis of other cardiovascular diseases. Mg ions are found to be essential for the maintenance of the normal functional integrity of the myocardium [4]. Several investigations have shown that the serum magnesium level are low in the first 24 hours following AMI and later there is a gradual raise and comes to normal level in about three weeks' time [5]. The prevalence of altered serum magnesium among patients with AMI is highly variable from 0% to almost 50% of normal value [6]. Low serum Mg level was observed in the present study in first 24 h (1.89 ± 0.09 m.eq/L) in all cases, as compared to the normal healthy control (2.1 ± 0.13 m.eq/L). Similar results have been obtained by studies conducted by Giesecke D *et al.* [7], Kafka H *et al.* [8], Bordia A *et al.* [9]. The onset of AMI is found to be fatal for many patients and those who survive were found to have impaired cardiac function. Creatine kinase is an enzyme that catalyses the reversible transfer of phosphate from ATP to creatine. This reaction in the presence of Mg makes possible the storage of high energy phosphate in a more stable form than in ATP. Hence the lowering of magnesium level in the cardiac muscle may be

ascribed primarily to the damage of heart muscle. The results of this study show that there is a decrease in extracellular Mg concentration during AMI and this may be due to the fact that during infarction, the ATP production is minimized. As a result, Mg is not utilized for the production of ATP and the intracellular free Mg concentrations rise rapidly and as a consequence, the influx of calcium into the myocardium is lowered. Thus Mg acts as calcium antagonist. Effects of Mg deficiency on the heart are also complicated by intracellular potassium depletion and hypokalemia ^[10]. The most important protective effect of Mg during MI is the restriction of the cellular loss of magnesium-adenosine tri phosphate, the essential substrate for many cellular reactions ^[11, 12].

Studies conducted by MBK Choudhury *et al.* ^[13], Lakshman Lal *et al.* ^[14] reported that there is significant hypomagnesemia in patients with acute myocardial infarction which was same in the present study, i.e., significant hypomagnesemia is observed in patients with acute myocardial infarction which is 30% observed in this study. It is observed that in studies conducted by Chakraborty PK *et al.*, 1.71 ± 0.17 mg/dl and 2.16 ± 0.25 mg/dl, Lakshman Lal *et al.*, 1.01 ± 0.94 mg/dl and 2.20 ± 2.23 mg/dl the mean value of serum magnesium is less in patients with AMI compared with serum magnesium levels in controls which is similar to the results obtained in this study. A. Akila *et al.* ^[5], in the study group comprising of 50 patients, 42 were males and 8 were females with a male-female ratio of 5.25:1. The maximum incidence of acute myocardial infarction was seen in the 4th and 5th decades. The mean serum magnesium level on day-1 in all 50 patients was 1.86 ± 0.39 and the mean serum magnesium level on day-5 was 2.26 ± 0.5 . Mean serum Mg level in cases without complications 2.08 ± 0.41 mg/dl, mean serum Mg level in cases with complications 1.65 ± 0.26 mg/dl. In the study by Akila *et al.*, the serum magnesium level on day-1 was significant lower in patients with arrhythmias than those without arrhythmia ($p < 0.001$). There was an increase in serum magnesium from Day-1 to Day-5 in both those with arrhythmias and those without arrhythmias. In the present study, study group of 50 patients, 33 were males and 17 were females with a male-female ratio of 3.33:1.87. The maximum incidence of acute myocardial infarction was seen in the 5th and 6th decades. The mean serum magnesium level on day-1 in all 50 patients was 1.89 ± 0.09 m.eq/L. Mean serum Mg level in cases without complications 1.98 ± 0.28 m.eq/L, Mean serum Mg level in cases with complications 1.44 ± 0.36 m.eq/L. In studies conducted by Dyckner *et al.* ^[6], Kafka *et al.* ^[8] patients with acute myocardial infarction who have hypomagnesemia appear to have two to three fold increase in the frequency of ventricular arrhythmia in the first 24 hrs when compared to those with normal plasma magnesium level. In the present study also arrhythmias occurred in six patients in hypomagnesemia and two patients in normal magnesium i.e. 3 folds increase in occurrence of arrhythmia in hypomagnesemia compared to normomagnesemia in patients with AMI. Many studies suggest that the administration of intravenous magnesium at this time can reduce the frequency of potentially fatal ventricular arrhythmia ^[16]. The Second Leicester Intravenous Magnesium Intervention Trial (LIMIT-2) included 2,316 patients, who were randomized to receive I.V. magnesium sulfate or matching placebo. Patients received placebo or

magnesium for 5 min before initiation of thrombolytic therapy, followed by an infusion for the next 24 h. It concluded that there was 24% reduction in 28-day mortality, a 25% reduced incidence of left ventricular failure, and an improvement in long-term survival in terms of reduction of long-term mortality from ischemic heart disease (average follow-up period of 2.7 years) ^[10].

Conclusion

Magnesium treatment may reduce the incidence of ventricular fibrillation, ventricular tachycardia, severe arrhythmia needing treatment, but it may increase the incidence of profound hypotension, bradycardia, and flushing. Magnesium decreases reperfusion injury by preventing or lessening mitochondrial calcium overload in ischemic myocardial cells during the first few minutes of reperfusion and preserving intracellular Adenosine Triphosphate (ATP) and creatine phosphate reserves and inhibits platelet function, perhaps indirectly by release of prostacyclin. Thus, magnesium-infusion started early after the onset of myocardial ischemia might limit infarct size, prevent serious arrhythmias, and reduce mortality. Time is critical in management of acute myocardial infarction.

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