Journal of Diabetic Association Medical College 2023;7(1)14-17

Original Article

Low Serum Magnesium Level and Complications in Acute Myocardial Infarction

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Abstract

Background: Magnesium has been considered as an important factor in the pathogenesis of acute myocardial infarction and its complications. Magnesium ions are essential for the maintenance of functional integrity of myocardium. It also improves vascular tone, after load and cardiac output, decreases peripheral vascular resistance and cardiac arrhythmias. Serum magnesium concentration has great significance in acute myocardial infarction.

Materials and Methods: This cross-sectional descriptive study was conducted in the Department of Pharmacology & Therapeutics, Rajshahi Medical College in collaboration with the Cardiology department, RMCH between July 2019 to June 2020 to measure the serum magnesium level of the patients and to compare the levels with complications. 50 patients of acute MI included through purposive sampling technique. Data were collected using a checklist from laboratory report.

Results: The meanlevel of serum magnesium was 1.70 ± 0.37 mg/dl in complicated cases. Serum magnesium level was 2.25 ± 0.15 mg/dl in patients without any complications which was significantly higher than the patients who had multiple complications (P< 0.001). These observations suggest that in acute myocardial infarction, patients with low magnesium levels are more prone to get complications.

Conclusion: On basis of findings it can be concluded that, low serum magnesium level can play a crucialrole inprogression of adverse squeals in acute myocardial infarction.

Keywords: Acute myocardial infarction, serum magnesium level, troponin-I level

Introduction

Magnesium is an essential mineral naturally found in human beings. This is the fourth abundant micronutrient which serves as a cofactor in more than 300 enzyme systems in our body.[1] There is around 20-24 gm of magnesium present in an adult human. 60% of total body magnesium is present in bones and one third of this magnesium acts as body magnesium reservoir. Almost 35% of total magnesium is located in high metabolic tissues such as muscles, brain, heart, kidneys and liver. Magnesium in the serum represents only 1% of the total body magnesium.[2] Its diverse action includes regulation of blood pressure, glycaemic control, lipid peroxidation and maintaining cardiac physiology.[3] Magnesium is a well known mineral for maintaining the normal functional integrity and electrical stability of the myocardium. It plays a vital role in the energy balance of cardiomyocytes. Its beneficial effects also include reducing vulnerability to oxygen derived free radicals, improving endothelial function and inhibiting platelet aggregation and adhesion Magnesium is beneficial for cardiovascular system as it improves myocardial lipid metabolism, reduces cardiac arrhythmias by inhibiting calcium accumulation.[2] Low level of serum magnesium has association with atherosclerotic acceleration, inducing hyperlipidaemia and subsequent atherogenic deposits in coronary arteries. Low magnesium concentration within the myocardial cell is associated with membrane destabilization and vice versa. Deficiency of magnesium can lead to vasoconstriction and also platelet aggregation as its potent vasodilating role in muscle contraction is hamperd.[1] Magnesium deficiency plays a major role in the pathogenesis of cardiovascular diseases both on biochemical and cellular level. It activates adenosine triphospatase (ATPase) which is important for proper cell membrane function and also the source of energy for the Na^+-K^+ pump. Low magnesium level causes decrease in Na^+- K^+ pump activity leading to a increase in intracellular sodium concentrations. This increased sodium concentration into the cell alters the membrane potential

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results in arrhythmia. Additionally magnesium modulates the potassium- proton exchange mechanism thus it protects the cell from potassium loss. Intracellular hypomagnesaemia may cause increased sodium and calcium concentration into cell predisposing arterial vasospasm, increased catecholamine release and fatty acid.[2] Myocardial contractility is affected by magnesium primarily by exerting calcium mobilization. Magnesium acts as a natural antagonist to calcium by competing with calcium for the binding of troponin C and calmodulin. Intracellular calcium is recognized for cardiac excitation-contraction coupling by binding with troponin. Intracellular and extracellular magnesium both control calcium influx into the cells by inhibiting the L-type calcium channels thus prevents intracellular calcium overload and cell toxicity. In acute myocardial infarction (AMI) increased cytosolic calcium leads to an increased risk for arrhythmia.[4] In myocardial infarction, functional deficit of available magnesium occurs due to trapping of free magnesium in adipocytes. Catecholamine induced lipolysis causes soap formation when free fatty acids are released.[5] Falling in magnesium concentration would destabilize the membrane potential and make cardiac cells more excitable, thus predisposing to arrhythmias. Further fall in magnesium levels increases calcium influx which was previously inhibited by it resulting increased systemic and pulmonary vascular resistance.[6] The serum magnesium level was found low in the first 48 hours following a acute myocardial infarction in several investigations. Also levels were found lower in patients with complications, when compared with acute MI patients without complication[5].Patients of AMI who have lower level of serum magnesium have more chance to develop tachyarrythmias.[6] Hence, it is obvious that serum magnesium concentration has great significance in acute myocardial infarction. This study was an attempt to find out the relation of serum magnesium in reference to these complications.

Materials & Methods

The design of this study was descriptive cross-sectional type. It was carried out in the Department of Pharmacology and Therapeutics in collaboration with Cardiology department of Rajshahi Medical College Hospital.Ethical clearance was obtained from the ERC of Rajshahi Medical College. 50 (age 40- 65 years) clinically diagnosed acute myocardial infarction patients admitting in cardiology department of Rajshahi Medical College Hospital, Rajshahi, was included by convenient / purposive sampling technique. After meeting all inclusion and exclusion criteria and confirmed diagnosis by clinical features, electrocardiography and cardiac enzyme Troponin I; other relevant laboratory investigations were done. Then 3 ml venous blood was taken from each subject in a test tube without anticoagulant. Serum was collected after centrifuging for 15 minutes at 3000 rpm. Then serum magnesium was measured. Measuring of serum magnesium was level was carried out by spectrophotometer using Magnesium Kit**.** All relevant information's were collected and compiled. Data were processed and analyzed using SPSS (Statistical package for social sciences), version 22.0 by descriptive statistics, unpaired t-Test, Chi-square test. The level of significance was set at 5% and P- value considered < 0.05%

Results

The mean age of the respondents was 54 ± 10.3 years. The highest respondents were in age groups of 51 -65 years which occupied 64% of total study population. Numbers of male respondents were predominant occupying 84%. Majority (66%) of respondents in this study were smoker (Table 1).

Table 1: Distribution of respondents by age, sex and smoking history (n=50)

Attributes	No. of respondents with %
Age (in years)	
<40	6 (12%)
41-50	12 (24%)
51-60	16 (32%)
61-65	16 (32%)
Sex	
Male	42 (84%)
Female	8 (16%)
Smoking history	
Smoker	33 (66%)
Non smoker	17 (34%)

Large numbers of the respondents (66%) were hypertensive and 34% were normotensive. The study showed 30% respondents had past history of ischemic heart disease. However, among 42% respondent concomitant disease was found. In terms of family history of Ischemic heart disease, it found in 62% of the respondents (table 2).

Table 2: Distribution of respondents by Hypertension, Previous Cardiac problems, Concomitant disease and Family history of IHD (n= 50)

Attributes	No. of respondents with %
Hypertension	
Hypertensive	33 (66%)
Normotensive	17 (34%)
Previous cardiac problem	
Present	15 (30%)
Absent	35 (70%)
Concomitant disease	
Present	21 (42%)
Absent	29 (58%)
Family history of IHD	
Present	31 (62%)
Absent	19 (30%)

In this study it was found that 17% respondents had chest pain, 12%% had tachycardia, 8% had bradycardia, 4.5% had heart block and 2.5% had heart failure (Table 3).

Table 3: Distribution of respondents by nature of complications after AMI (n=50)

No complications	12 (6%)
Chest pain	34 (17%)
Tachycardia	24 (12%)
Bradycardia	16 (8%)
Heart block	9 (4.5%)
Heart failure	5 (2.5%)

32% Respondents had hypomagnesaemia (<1.6mg/dl) with complications whereas among 68% respondents 50% had serum magnesium ≥1.6mg/dl with complication (Table 4).

Table 4: Serum Magnesium level among complicated and un-complicated patients (n=50)

Serum magnesium level			
	Without complication 09	With complication 41	Total (n= 50)
<1.6 mg/dl	0 (0%)	16 (32%)	16 (32%)
≥ 1.6 mg/dl	9 (18%)	25 (50%)	34 (68%)

Higher serum magnesium level was observed in uncomplicated patients while patients who suffered from multiple complications had lower serum magnesium. Patients with multiple complications the serum magnesium level was 1.79 ± 0.37 and in patients without complications magnesium level was 2.25 ± 0.15. On comparison between two groups, statistically significant (P-Value = 0.01) difference was found. (Table 5).

Table 5: Mean serum Magnesium level among complicated and un-complicated patients

Serum magnesium level	(n=50)			
	n	Mean±SD	t value	p value
Without complications	9	2.25±0.15	-3.537	0.001
With Complications	41	1.79 ± 0.37	-3.537	0.001

Discussion

The age of the patients included in this study was ranging between 30 - 70 years. This study showed highest respondents in age group 51 - 60 years and age group > 60 years which occupied 16 (32%) of the population respectively. These results are similar to findings of Anjum et al. (2013) where 43.3% of patients were in age group 51-60 years.[7] The sex distribution of the study group showed male predominance. There were 42 (84%) male patients whereas 8 (16%) female. Lakshman Lal and Hiralal Murmu (2016) conducted a study where they observed similar findings showing 29 male and 11 female patients[1]. Akila et al. (2017) studied with 50 patients of acute myocardial infarction and found smoking was the most common risk factor among 35 patients.[8] In this present study, similarly we found 33 (66%) smoker among the patients and 17 (34%) were not smoker. "Subramanyam and Vakrani, (2015) subjected the serum magnesium level in 53 patients of acute myocardial infarction, showed hypertension as a high risk factor in their study.[5] Likewise this study also demonstrated that hypertension was present in 33 (66%) of the patients. In this study, positive history of ischemic heart disease was found 15 (30%) respondents and 35 (70 %) respondents had no history of ischemic heart disease. We found presence of family history of ischemic heart disease as a risk factor among 31 (62%) patients.

In case of presence of concomitant disease, this study revealed that 21 (42%) patients were suffering with other concomitant disease and 29 (58%) patients had no concomitant disease. Similar finding was found in a study conducted by Angeline et al. (2003) where it is stated that diabetes patients are more at risk of developing myocardial infarction.[9] Family history of ischemic heart disease was absent in 19 (38%) patients. This resembles the finding of Akila et al. (2017) where 10 (20%) patients having positive family history of ischemic heart disease.[8]

In this study mean serum magnesium level was 2.25 ± 0.15 mg/dl in patients without any complications. The patients with multiple complications mean serum magnesium level was 1.70 ± 0.37 mg/dl. On comparison, statistically significant difference was found (P <0.001). Lowest magnesium level of 1.64 ± 0.36 mg/dl was found in patients with heart failure followed by patients who developed bradycardia having serum magnesium level 1.69 ± 0.36 mg/dl. Comparison of serum magnesium level between patients without complications and with multiple complications groups it was found that 16 patients suffered hypomagnesaemia (<1.6 mg/dl) along with multiple complications. About 25 complicated patients had serum magnesium level ≥1.6 mg/dl. All the uncomplicated patients had serum magnesium level ≥ 1.6 mg/dl. Similar findings were found in a study conducted by Nambakam and Girish (2015).[5] Their study revealed that in complicated cases serum magnesium levels were 1.38 ± 0.03 mg/dl and in patients without complications the level was 1.73 ± 0.29 mg/dl which was statistically significant.[5] So, the fact is, measuring serum magnesium level had prognostic significance in acute MI. In attempt to find the prognostic value of serum magnesium in various complications, serum magnesium was estimated spectrophotometrically by Govind Mohan et al. (1994) in 53 acute myocardial infarction cases.[6] The study showed lower serum magnesium levels of 1.26 ± 0.19 mg/dl in 42 cases of acute myocardial infarction with complications compared to 1.41±0.13 mg/dl in 11 patients without complications.[6]

It was observed that patients who died due to arrhythmias and cardiogenic shock followed by pump failure, serum magnesium were lowest in them. Similar study by GQ Khan et al. (2002) reported low serum magnesium in 50 patients of acute myocardial infarction with mean serum Mg levels of 2.2 ± 0.24 mg/dl in controls.[11] Further, the serum magnesium level of patients who developed cardiac arrhythmias was found to be comparatively lower. Therefore they concluded that, the low level of Mg in serum can be taken as a sensitive diagnostic index in cases of acute MI.[10] Another study of serum magnesium in acute MI patients conducted by Dr. Naseem Hussain (2018) where the author found statistically significant fall in magnesium level in serum in the patients.[11]

In summary, this study demonstrated that a significant difference in serum magnesium level in patients with complications and without complications. The serum magnesium level was higher in patients who had no complications than those who suffered multiple complications. Patients who had serum magnesium level <1.6 mg/dl developed more complications. Findings showed that low level of serum magnesium was associated with increased risk of developing more adverse sequels after acute MI. Hypomagnesaemia in early periods of acute MI is responsible for poor prognosis anddetection of serum magnesium would have been a helpful alternative for taking action accordingly.

Conclusions

This study revealed the mean serum magnesium level1.70 ± 0.37 mg/dl in complicated cases and 2.25 ± 0.15 in cases without any complications which was statistically significant. Hypomagnesaemia is recognized as a significant risk parameter for hypertension, cardiac arrhythmias and other ischemic heart diseases contributing pathogenesis of AMI. So, along with other biochemical risks parameters, routine assessment of serum magnesium level estimation can be a useful choice for avoidance of adverse events. Further study with a large group of similar population can be considered in future.

Acknowledgment

First of all,we remember Almighty ALLAH, for giving us the prospect and potency to carry on and complete this research work. Thank to Pharmacology and Cardiology department of Rajshahi Medical College for providing data to complete this study. All author's contributions are equally acknowledged along with contribution of study respondentsin particular.

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