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A Study of Serum Magnesium Levels in Acute Myocardial Infarction

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Abstract

Coronary artery disease is the most common cause of premature deaths. It is characterized pathologically by atheromatous plaque which may stenose the coronary arterial system sufficiently to cause exertional myocardial ischemia. There are reports in the literature over patients with acute myocardial infraction have lower magnesium levels. Supplementation of magnesium salts may reduce the incidence of fatal and on fatal arrhythmias after an infarct and thereby decreased the mortality in AMI. The present study was undertaken to determine whether changes occur in the serum magnesium level in acute myocardial infarction and to plot its time course during the acute illness and if changes occurred, were they related to the occurrence of arrhythmias and its prognostic values. The present study was conducted on 37 patients of acute myocardial infarction admitted to ICCU. Patients presenting with chest pain suggestive of myocardial infarction, who showed E.C.G evidence of acute infarction changes were assessed by detailed history and physical examination. The infarction was confirmed by elevated CPK/SGOT levels. Serum magnesium is estimated within first 24 hours of chest pain 1st day and on 9th day as well. Eight controls were chosen in the age group of 35 to 70 years. Simultaneously serum potassium levels were estimated. The individual values of Serum Magnesium

and Potassium in controls and in patients with acute myocardial was estimated. statistical analysis was done. Of the thirty seven cases of acute myocardial infarction, ten patients were hypomagnesemic on the first day of A.M.I. In the other twenty seven cases acute myocardial infarction studied, Serum Magnesium concentrations were within normal range. It is evident from the above results, that a statistically significant decrease in mean serum magnesium levels was noted in patient with AMI as compared to control levels.

Keywords: Magnesium, Myocardial infarction, AMI, Arrhythmias.

Coronary artery disease is the most common cause of premature deaths. Its cause is unknown. But a number of risk factors have been identified including diabetes, hypertension, hypercholesterolemia, cigarette smoking, correlation of which may protect the development and progression of disease. It is characterized pathologically by atheromatous plaque which may stenose the coronary arterial system sufficiently to cause exertional myocardial ischemia, experienced by the patient as angina. Plaque rupture provides a focus for platelet deposition and thrombosis which my result in unstable angina or myocardial infarction depending on whether the thrombus is subocclusive or occlusing the coronary lumen completely. There are reports in the literature over patients

Material and methods

Selection of Patients:

The present study was conducted on 37 patients of acute myocardial infarction admitted to ICCU M.G.M hospital, Warangal who fulfilled the following criteria, included in the present study.

- 1. Patients presenting within 24 hours after the onset chest pain.
- 2. Patients showing evidence of infarction of either anterior or inferior wall were included.
- 3. Patients with history of previous infarction, chronic alcohotism and patients on diuretics were excluded.

- 4. Patients with evidence of serious diseases involving other system were also excluded.
- 5. Continuous bed ECG monitoring

Patients presenting with chest pain suggestive of myocardial infarction, who showed E.C.G evidence of acute infarction changes were assessed by detailed history and physical examination as outlined in the proforma. Subjects having hypertension, diabetes, history of smoking was noted. Type of infarction was also noted. The infarction was confirmed by elevated CPK/SGOT levels. Serum magnesium is estimated within first 24 hours of chest pain 1st day and on 9th day as well. Patients showing normal magnesium levels on admission were not subjected to repeat examination of magnesium levels on 9th day. Eight controls were chosen in the age group of 35 to 70 year. In the present study, serum magnesium is estimated by colorimetric method using calmagite. . Colorimetric determination of magnesium is done without deproteinization using calmagite. statistical analysis was done.

Results

Table 1 shows that out of 37 MI patients,10 patients had low Sr.magnesium on addmission.27 patients had normal Sr.magnesium levels. 100% patients with low magnesium level developed arrhythmias.81% patients with normal magnesium levels developed arrthymias.

Table 1:

Total Patients with	Patients with decreased Serum	Patients with normal Serum
myocardial infarction [37]	Magnesium levesl on Admission [10]	Magnesium levels on
		admission [27]
Patients developed		
Arrythmias	10 (100%)	22 (81%)
		22 (0170)

Table 2 and Table 3 shows characteristics of normomagnesic and hypomagnesic patients in relation to history of diabetes, hypertension and smoking history, and also type of MI they developed.

Table 2 . Characteristics of hypomagnesemic patients with acute myocardial infarction.

Variable	Present Study	
Males	8	
Females	2	
Male / Female Ratio	4:1	
Hypertension	6 (60%)	
Diabetes	4 (40%)	
Smoking	6 (60%)	
Anterior Wall MI	6 (60%)	
Inferior wall MI	4 (40%)	

Table 3. Characteristics of normomagnesemic patients with acute myocardial infarction.

Variable	Present Study
Males	18
Females	9
Male / Female Ratio	2 :1
Hypertension	15 (55.5%)
Diabetes	1 4 (51.85%)
Smoking	16 (59.25%)
Anterior Wall MI	16 (59.25%)
Inferior wall MI	11 (40.74%)

Table 4 shows that 10 patients had low Sr.magnesium level on first day of addmission ,by ninth day the magnesium levels increased .27 patients had normal Sr.magnesium level on addmission.Similar results were observed in other studies[7,8].

Table 5 shows Sr.magnesium levels in 8 control subjects.

Table 4. Mean and standard deviation values of Serum magnesium in patients.

Patients with low serum	Patients with low serum	Patients with normal serum
magnesium on admission	magnesium on admission	magnesium on admission
(mg%) 10 patients	(mg%) 10 patients	(mg%) 27 patients
1 st day	9 th day	1st day
1.07 ± 0.23	2.32 ± 1.78	2.32 ± 0.32

Table 5. Mean and standard deviation values of Serum magnesium in 8 Control subjects.

2.29±0.174 mg%

Discussion

Few studies have demonstrated that Serum Magnesium concentration decreases significantly during A.M.I. The cause of hypomagnesemia during the early phase of infarction is related to the increased stress induced catecholmine release, which induce enhanced lipolysis and sequestration of magnesium with free fatty acids (FFA) and adipocytes. It has been shown that magnesium depletion modifies coronary blood flow, blood clotting, and atherogenesis [9]. Magnesium cofactor in more than 300 enzymes system of the body in human cell. Its possible site of action includes vascular smooth muscle, platelets, and myocardial cells [10]. Magnesium depletion can induce hyperlipidemia and subsequently atherogenic deposits in coronary arteries leading to atherosclerosis[11].Routine use of IV magnesium is recommended within first few hours of acute myocardial infarction to reduce mortality, arrhythmias and pump failure.[12,13].Magnesium treatment reduces ventricular tachycardia and ventricular fibrillation[14]. Although the present study and many other studies have indicated a trend towards disturbed magnesium homeostasis in acute myocardial infarction, particularly in cases of pump failure, serious arrhythmias and mortality, the data seems

Intravenous magnesium therapy can therefore be recommended for use in cases of acute myocardial infarction within first few hours of the onset of infarction because of its effect on prognosis, economy.

Conclusion

Serum magnesium levels on admission were significantly low in patients of acute myocardial infarction as compared with healthy controls. Hypomagnesemia is often associated with acute myocardial infarction. Prophylactic administration of the intravenous magnesium sulphate may be considered in all cases of Acute Myocardial infarction as an adjuvant to thrombolytic therapy and in patients not suitable for thrombolysis, to prevent cardiac arrhythmias, irrespective of serum magnesium levels.

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