

SERUM MAGNESIUM : A PROGNOSTIC TOOL OF ACUTE MYOCARDIAL INFARCTION

GOVIND MOHAN AND V. K. JAIN*

*Departments of Pharmacology and Cardiology**
S. N. Medical College,
Agra - 282 002

(Received on October 16, 1993)

Abstract: In an attempt to find the prognostic value of serum magnesium in various complications, serum magnesium was estimated spectrophotometrically, in patients of acute myocardial infarction. There was a statistically insignificant difference in serum magnesium levels of complicated group of patients (1.627 ± 0.192 mg/100 mL) and those who had uneventful recovery (1.412 ± 0.132 mg/100 mL). However, this difference was highly significant when compared between control group (2.514 ± 0.16 mg/100 mL) and complicated and uncomplicated cases separately. It was also observed that serum magnesium levels were lowest in patients who died due to major arrhythmias and cardiogenic shock followed by patients who had arrhythmias (especially ventricular tachycardia) and pump failure. The routine use of iv magnesium is recommended within first few hours of acute myocardial infarction to reduce mortality, arrhythmias and pump failure.

Key words: magnesium arrhythmias myocardial infarction

INTRODUCTION

A fall in serum magnesium following acute myocardial infarction has been observed by many workers (1, 2) and magnesium deficiency is known to cause cardiac arrhythmias (3). It has been observed in many studies, that cases of myocardial infarction who have lower serum magnesium level have more chances of developing tachyarrhythmias (2, 4). However, the relationship of lowered magnesium level to the pump failure (Cardiac failure and cardiogenic shock), conduction defects and sudden death has not been well established. The present study is undertaken to find the prognostic value of serum magnesium levels in reference to these complications.

METHODS

Fifty-three patients of first acute myocardial infarction admitted to coronary care unit of S.N.

Medical College and Hospital, Agra were included in the study. Age and sex matched 30 healthy volunteers served as control. A detailed clinical evaluation was performed and patients were monitored for 48-72 hours in coronary care unit. Blood samples for enzyme estimation such as CPK and SGOT were obtained at 12, 24, and 36 hr. Routine blood count, blood sugar and blood urea, serum sodium and potassium were estimated on the day of admission. The patients with the history of carcinoma, alcoholism, cirrhosis, renal failure, chronic diarrhoea, vomiting, pregnancy, leprosy, rheumatoid arthritis, which are likely to affect the serum magnesium levels were excluded from the study. The blood samples for serum magnesium were withdrawn with a disposable plastic syringe on 1st (within 12 hr of myocardial infarction), 4th, 10th and 14th day of admission and stored in sterilized plastic vials. Serum magnesium was estimated spectro-photometrically reading absorbance at 520 nm (5).

*Corresponding Author

RESULTS

Table I shows serial mean serum magnesium levels in cases of acute myocardial infarction. The mean serum magnesium level was 2.514 ± 0.16 mg% in normal healthy control volunteers, whereas the mean serum magnesium levels in complicated and uncomplicated group was 1.267 ± 0.19 mg% and

Table II shows serial mean serum magnesium levels in cases of acute myocardial infarction with individual complications. It was observed that serum magnesium levels were lowest in patients who died (1.112 ± 0.110 mg%) followed by the patients who had arrhythmia, (1.163 ± 0.218 mg%) pump failure (1.285 ± 0.091 mg%) and conduction defect (1.613 ± 0.062 mg%).

TABLE I: Serum Magnesium levels in cases of acute myocardial infarction in comparison with normal healthy control and according to complications of MI.

Group	Number	Serum magnesium, mg/100 ml (mean \pm SE) on day			
		1	3	10	14
A Control	30	2.514 ± 0.16	-	-	-
B Acute MI (total)	53	1.382 ± 0.21	1.932 ± 0.29	2.351 ± 0.23	2.552 ± 0.15
C Acute MI with complications	42	$1.267 \pm 0.19^*$	$1.942 \pm 0.22^*$	2.365 ± 0.12	2.571 ± 0.11
D Acute MI without complications	11	$1.412 \pm 0.13^{**}$	1.894 ± 0.19	2.297 ± 0.16	2.497 ± 0.12

A Vs C *P < .001

A vs D **P < .001

1.412 ± 0.13 mg% respectively on first day of myocardial infarction, which gradually attained near normal value by 14th day. The values were not significantly different in complicated from uncomplicated group.

TABLE II: Comparison of serum magnesium levels in uneventful cases of myocardial infarction with those having various complications on day one.

Group	No. of cases	Serum magnesium in mg/100 mL (mean \pm SE)
Uncomplicated	11	1.412 ± 0.132
<i>Complicated:</i>		
Arrhythmias	28	1.163 ± 0.218
Conduction defect	04	1.613 ± 0.062
Pump Failure:	10	1.285 ± 0.091
(a) LVF	06	1.312 ± 0.122
(b) Cardiogenic shock	04	1.246 ± 0.102
Complications resulting in death	06	1.112 ± 0.110

DISCUSSION

The reported prevalence of altered serum magnesium concentrations among patients with acute myocardial infarction is highly variable from 0% to almost 50% of normal value (1). Low serum magnesium level was observed in the present study in first 12 h (1.382 ± 0.21 mg/100 mL) in all cases, as compared to the normal healthy control. This level increased slowly attaining near normal value by 14th day. Similar results have been obtained by others (6, 7). The reported unchanged serum magnesium levels in acute myocardial infarction (8) may be due to difference in time of collection of samples. Serum magnesium levels decreased to a nadir before the peak of the creatine kinase enzyme activity, i.e., within 24 h and they start normalising thereafter. This fall can be explained by a shift of magnesium from extracellular to intracellular compartment, which appears to occur by increased uptake of magnesium into adipocytes following catecholamine induced lipolysis and formation of intracellular magnesium "soap" with free fatty acids (9).

Serum magnesium levels were apparently lower in patients with complications as compared to cases who had uneventful recovery (Table I), although the difference was not significant. On further analysis, it was evident that cases who had arrhythmias, showed significantly lower levels as compared to uncomplicated cases. Similarly, the cases who died due to major arrhythmias or cardiogenic shock had very low serum magnesium. The cases with pump failure (Killip class III & IV) had lower levels of serum magnesium. Although these results were statistically not highly significant, a trend to lower serum magnesium values was seen in cases with major complications with an exception of conduction defects.

Magnesium is an important intracellular ion, which is responsible for activation of sodium potassium adenosine triphosphatase and is thought to be essential for the control of potassium flux across the cell membrane. A fall in magnesium concentration would destabilize the membrane potential and make cell more excitable, thus predisposing to arrhythmias. Further a fall in magnesium levels is likely to effect magnesium inhibited calcium influx thus increasing the systemic and pulmonary vascular resistance, i.e.,

after load, which is an important determinant of pump failure.

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 to be inconclusive because of smaller number of patients in each study.

Trials already conducted on iv magnesium therapy in acute myocardial infarction have shown reduced incidence of cardiogenic shock and arrhythmias, but no effect on mortality, however, the meta-analysis of seven trials showed a 55% reduction in odds of deaths (10) and in the recently completed LIMIT-2 study (11). There was a 24% reduction of mortality in patients of acute myocardial infarction treated with iv magnesium.

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 and safety.

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