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Alteration in Magnesium Level in Acute Myocardial Infarction

T. Angeline*, K. Ramadevi[1], Rita Mary Aruna[1], G. Mohan[1] and Nirmala Jeyaraj

PG & Research Department of Zoology, Lady Doak College, Madurai, India;
[1]Department of Biochemistry, Madurai Medical College, Madurai, India

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Sodium, potassium, calcium, zinc and magnesium levels in the serum of 31 patients diagnosed as acute myocardial infarction were analyzed on admission (within 24 Hours) and after 48 hours. The results were compared with those of 26 age matched controls. No significant difference was observed in the mean sodium, potassium, calcium and zinc levels between the cases and controls. Compared to the controls, however, the variation in the level of magnesium is highly significant at the time of admission as well as after 48 hours. When the risk factors like diabetes mellitus, hypertension, smoking and alcohol were considered, it is found that there is no significant difference between the risk groups as well as between the patients. The alteration in magnesium level in acute myocardial infarction is independent of these risk factors. Within the first 24 hours, the significant decrease in serum magnesium (35-51% fall when compared with the control group), correlates with its entry into the cell following ischemia. From this hypo-magnesemic state, it rises to 9-22 times after 48 hours. This hyper-magnesemia after 48 hours is probably due to the shift of magnesuim from the intracellular fluid compartment to the extracellular fluid compartment that follows cellular recovery. Therefore, including magnesium in the immediate management of acute myocardial infarction will be beneficial in the early recovery.

Acute myocardial infarction (AMI) is the most important consequence of coronary artery disease and it can lead to complications such as ventricular arrhythmia and congestive heart failure. Some of the major risk factors that contribute to AMI are hypertension, dyslipidemia, diabetes, smoking, alcoholism, obesity, stress and lack of exercise. Magnesium is an important factor in the physiology of the cardiovascular apparatus and in the pathogenesis of cardiovascular disease (Durlach et al., 1992). It is a trace mineral involved in several hundred chemical reactions in the body, including carbohydrate utilization, ATP metabolism, muscle contraction, transmembrane transport and the synthesis of fat, protein, and nucleic acids. It has a lot of therapeutic potential. It is less clear whether it is useful in patients with congestive heart failure or acute myocardial infarction (Swain and Kaplan-Machlis, 1999).

The aim of the present study is to observe the changes in magnesium levels during and after AMI. This change is compared with another trace element zinc and electrolytes like sodium, potassium and calcium. This will enlighten the use of magnesium infusion to minimize the complications after myocardial infarction.

*To whom all correspondences should be addressed.
E-mail: angelanand@yahoo.co.in

Materials and Methods

Patients admitted to the intensive care unit of the Vadamalayan Hospital, Madurai, South India, with documented AMI showing characteristic ECG signs and rise in total creatine-kinase activity (more than 3 times the upper reference value of our laboratory) and altered CK-MB fractions were included in this study.

Blood samples of 31 patients were collected during AMI and after 48hrs of Infarction. The collected blood samples were allowed to clot; centrifuged and the serum was separated and stored in the freezer (-20°C). The stored samples were thawed while performing the experiment. Steps were taken carefully to avoid hemolysis of the blood.

The details including age, gender, history of previous myocardial infarction and cardiovascular risk factors (smoking, dyslipidaemia, hypertension and diabetes mellitus) and medication were obtained at the time of admission and it was also made sure that none of the patients were given Mg infusions during the treatment.

The measurement of serum magnesium and zinc was performed with flame atomic absorption spectrophotometer (Elico Ltd) at 256.2 nm. The serum sodium and potassium were determined using Flame emission spectrophotometry. (Elico Ltd.) and the serum calcium was

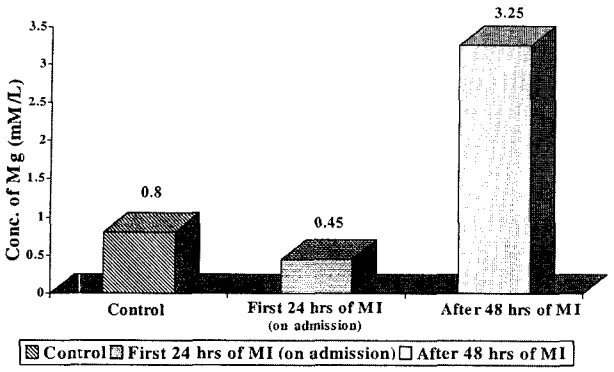

Fig. 1. Level of serum magnesium in control and in experimental (first 24 hrs+after 48 hrs of MI) groups.

estimated using O-cresolphthalein complexone method.

Analysis of variance was used to test the significance. All data were expressed as Mean ± SEM (or) percentage occurrences of a variable in both the sets. A P value < 0.01 was considered statistically significant.

Results and Discussion

In our study, no significant difference was observed in the mean sodium, potassium, calcium and zinc levels between the cases and control subjects (Table 1). However, the mean level of magnesium was found to be 0.45 ± 0.19 mM/L on admission, while after 48 hrs it was 3.25 ± 1.39 mM/L. The controls had a mean serum magnesium level of 0.80 ± 0.02 mM/L. (Table 1, Fig. 1) Compared to the controls, the variation in the level of magnesium is highly significant at the time of admission as well as after 48 hours ($p < 0.01$)

When the risk factors like dyslipidaemia, diabetes, hypertension, smoking and alcohol were considered, there is no significant difference between the risk groups as well as between patients ($p > 0.01$). Within the first 24 hours, there is 35-51% fall of serum magnesium when compared with the control group. From this hypomagnesemic state, it rises to 9-12 times after 48 hrs.

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 adenosine triphosphate (ATP) to creatine. This reaction

Table 1. Concentration of measured variables during and after 48 hrs of MI

Sample No.	Variable	Control	First 24 hrs of MI(on admission)	After 48 hrs of MI
1	Sodium (mM/L)	140 ± 1.3	142 ± 1.3**	142 ± 2.1**
2	Potassium (Meq/L)	3.8 ± 0.02	4.1 ± 0.03**	4.2 ± 0.1**
3	Calcium (mM/L)	2.40 ± 0.03	2.43 ± 0.06**	2.41 ± 0.02**
4	Magnesium (mM/L)	0.80 ± 0.02	0.45 ± 0.19*	3.25 ± 1.39*
5	Znc (mM/L)	12.3 ± 0.04	14.1 ± 0.53**	13.9 ± 0.71**

*$P < 0.01$ significant, **$P > 0.01$ not significant.

in the presence of magnesium 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 our study shows that there is remarkable decrease in extracellular magnesium concentration during AMI and this may be due to the fact that during infarction, the ATP production is minimized. As a result, magnesium is not utilized for the production of ATP and the intracellular free magnesium concentrations rise rapidly and as a consequence, the influx of calcium into the myocardium is lowered. Thus magnesium acts as calcium antagonist. The effects of magnesium deficiency on the heart are also complicated by intracellular potassium depletion and hypokalemia. (Woods and Fletcher, 1994; Jeremias et al., 2000; Rubenowitz et al., 1996).

AMI is a severe stressor and also accounts for the alteration of magnesium in the myocardium. Due to stress, the β-adrenergic receptors of adrenal gland gets stimulated and the catecholamines are released. Catecholamines decrease concentration due to a shift of magnesium into cells as a result of stimulation of β-adrenergic receptors (Woods and Fletcher, 1994; Jeremias et al., 2000; Rubenowitz et al., 1996; Strange et al., 1974; Ceremuzynski et al., 1991; Durlach et al., 1994). High catecholamines may be one of the contributing factors for the hypomagnesemic state during infarction.

The present study also indicates that the level of serum magnesium in the post AMI period is higher than that of during infarction. This has been supported by the following fact that during reperfusion, the magnesium utilization is enhanced because of ATP production, and the calcium influx leads to calcium-overload inside the myocardium (Woods and Fletcher, 1994; Seelig, 1989). During this reperfusion period, the presence of normal or high concentration of magnesium inside the cells, inturn may reduce the risk of complications after AMI. However the level of total calcium remains unaltered during and after AMI. It may be noted that in the present study only the total calcium was estimated and not the ionized form of calcium. Hence the estimation of ionized calcium may be valuable for identifying the patients who is at risk of developing AMI.

Earlier, it was observed that patients with acute myocardial infarction are magnesium deficient and this deficiency increases during the acute phase of infarction. But later it was observed that the magnesium infusion in patients with suspected AMI could prolong survival. The magnesium ion has multiple effects on the myocardium; among them are antiplatelet, antiarrythmic and coronary vasodilator effects. However, its efficacy for prolonging post-AMI survival is probably due to its ability to preserve left ventricular function by reducing calcium-mediated ischemic damage (Woods and Fletcher, 1994).

The most important protective effect of magnesium during myocardial infarction is the restriction of the cellular loss of magnesium-adenosine tri phosphate, the essential substrate for many cellular reactions (Seelig, 1989; Rubenowitz et al., 1996; Swain and Kaplan-Machlis, 1999). Studies have also reported that magnesium also limits the loss of cellular potassium and this correlation needs further study with a large sample size.

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