ORIGINAL ARTICLE

A STUDY OF SERUM HYPOMAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION

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ABSTRACT: There are reports in the literature over patients with acute myocardial infarction, who have low lower magnesium levels. Results of clinical, laboratory and epidemiological studies indicate that the association between magnesium deficiency and poor prognostic outcome in patients who have myocardial infarction. The role of magnesium in treating arrhythmias due to acute myocardial infarction (AMI) has been controversial. Most of the studies have reported a significant reduction in the mortality and frequency of arrhythmias in patients with AMI. A study was conducted on 37 patients with acute MI who presented within 24 hrs following the onset of chest pain (suggestive of Myocardial infarction). Serum Mg levels were estimated within first 24 hrs. The study was done with the main purpose of studying serum levels of Mg in these patients, in order to establish possible relation between low levels of serum Mg and increased risk & associated poor prognosis in patients with acute MI.
KEYWORDS: Hypomagnesemia, Acute Myocardial infarction, arrhythmias.

INTRODUCTION: Ischemic heart disease is the leading cause of morbidity and mortality worldwide.[1] The prevalence of myocardial infarction (MI) is more in the middle income world; (WHO report, 2004) and it is well known that males are more commonly affected than females. Magnesium is the second most abundant intracellular cation and it is vital for more than 300 enzymatic reactions which are involved in various metabolic processes in our body, but still, it is often a parameter which is overlooked by the clinicians.[2]

Hypomagnesemia is now recognized as a significant risk factor for atherogenesis, and thus for hypertension, ischemic heart disease, cardiac arrhythmias, coronary vasospasm, myocardial infarction, sudden cardiac death, and even cerebrovascular accident. Magnesium is a cardio protective element because of its βadrenoreceptor blocking action, antiplatelet action and inhibitory effect on the cardiac conducting system.[3] The ion stabilizing effect of magnesium helps in maintaining stable intra and extracellular concentrations of other electrolytes. Studies have documented significant alterations in magnesium (Mg+2) and other electrolytes in patients with Acute Myocardial Infarction (AMI).[4]

MATERIALS & METHODS: The present study was conducted to find out whether there is any change in the serum Mg levels in patients with acute phase of MI and to plot its time course during the acute illness. Serum Mg levels role in the occurrence of arrhythmias and its prognostic value in acute MI is studied. The study was conducted on 37 patients of acute MI admitted I our institute fulfilling the below mentioned criteria. Inclusive criteria – patients presenting within 24 hrs of onset of chest pain, showing evidence of infarction of either anterior or inferior wall.

Patients with previous history of infarction, chronic alcoholism, evidence of other serious disease and those on diuretics were excluded from the study. Continuous bed side ECG monitoring was performed.

Patients presenting with chest pain suggestive of MI, who showed ECG evidence of acute MI were assessed by detailed history and physical examination. The infarction was confirmed by elevated CPK/SGOT levels. Serum Mg levels were estimated within first 24hrs of chest pain on day one, and on day 9 as well. Patients showing normal Mg levels on admission were not subjected to repeat examination of serum Mg levels on day 9. Eight controls were chosen in the age groups of 35 to 70 years, simultaneously serum Potassium levels were estimated.

Serum Mg levels were estimated using calorimetric method. Calorimetric determination of magnesium without deproteinization using calmagite was performed. Elimination interferences due to calcium are around up to 3.8 mol/I (150mg/l). Range of expected values is serum of Mg is between 0.65 – 1.05 mol/I or 14 – 25.5 mg/l. Serum or heparinized plasma is the sample used for the estimation of Mg levels. Reagents used include Mg sulphate, calmagite and Reagent PH 11 EGTA.

RESULTS: A total of 37 patients were included in the study based on the inclusion criteria mentioned earlier. The study was carried out between September 2002 to October 2004 at our institution. Of the 37 patients, 27 were male and 10 were females. (Male, female ratio – 2.7). Number of deaths during the study period – 7. Total number of patients with acute MI associated with hypomagnesemia was 10 (males – 08, females – 02 male, female ratio – 4:1 and number of deaths – 02).

Range and mean of serum Mg levels are 1.90 to 2.48 and 2.15 respectively. Of the 37 patients admitted serum Mg levels in 10 patients on the first day of admission ranged between 0.82 to 1.3 mg percent. A statically significant fall in serum Mg concentration on the first day of admission was noted in these patients. In the 10 hypomagnesemia patients 8 of them developed ventricular arrhythmias. Out of these 8 patients four of them developed VPCs, two of them developed ventricular tachycardia and remaining two patients developed ventricular fibrillation. The serum Mg levels rose to 1.6 to 2.16 mg % by the seventh day of admission.

DISCUSSION: Data are accumulating that indicate that the magnesium cation may be a promising agent for protection of ischemic myocardium and modulation of reperfusion injury. Magnesium is a critical cofactor in more than 300 intracellular enzymatic processes, many of which are integrally involved in mitochondrial function, energy production, maintenance of transsarcolemmal ionic gradients, cell volume control, and resting membrane potential.[5]

Small trials reporting the use of magnesium in acute myocardial infarction have been identified intermittently for 20 years. The rationale for these studies came partly from observations of differences in heart attack rates associated with geographic variations in Mg and partly from laboratory studies showing that Mg had cardioprotective effects during ischemia and that myocardial Mg concentrations were relatively low during acute ischemia.[6]

We found serum Mg levels, independent of other risk factors, are inversely related to the incidence of acute myocardial infarction. Few studies have demonstrated that serum Mg

concentration decreases significantly during AMI. The cause of hypomagnesemia during the early phase of infarction is related to the increase stress induced catecholamines release, which induce enhanced lipolysis and sequestration of magnesium with free fatty acids and adipocytes.

The cardiovascular consequences of magnesium deficiency in animal and clinical studies have been summarized by Seelig[7] and include multifocal necrosis with calcium accumulation in mitochondria in a pattern reminiscent of myocardial ischemia and catecholamine-induced cardiomyopathy, atherogenesis, a heightened tendency to platelet aggregation, increased coronary and peripheral vascular resistances, sinus tachycardia and repolarization abnormalities, and ventricular tachyarrhythmia.

A review of epidemiologic studies has highlighted an inverse relation between the magnesium content of drinking water and ischemic heart disease–related mortality in various populations.[8] Intravenous infusions of magnesium in patients have been reported to reduce coronary and systemic vascular resistance, inhibit platelet aggregation, and terminate episodes of torsades de pointes–type ventricular tachycardia.[9]

Magnesium deficiency is known to predispose to the evolution of cardiac arrhythmias even with normal serum potassium concentration. Magnesium deficiency interferes with the function of membrane ATP-ase and thus, the pumping of sodium out from the cells and potassium into the cells is impaired. This disequilibrium of potassium between intra and extra cellular spaces may result in changes in resting membrane as well as disturbance in the repolarization phase and results in cardiac arrhythmias.

Magnesium has coronary and systemic vasodilatation and antiarrhythmic, antithrombotic and antioxidant properties and direct, myocardial protective effect in experimental and clinical models of ischemia- reperfusion injury. In the present study there was a statically significant fall in serum Mg concentration on the first day of AMI in 27.02% of patients as compared to controls.

In all the 37 patients serum potassium concentrations were normal.

CONCLUSION: All the studies showed a mean decrease in serum Mg levels in patients with AMI on first and also increased incidence of ventricular arrhythmias in the hypomagnesemia group compared to normo magnesemic group. Serum Mg levels do have an effect on the incidence of AMI independent of other risk factors. The correlation between low serum Mg levels and increased risk of development of AMI has been indicated based on the results of the above mentioned case series. The need for IV Mg infusion to counter act the transient post infarctional hypomagnesemia therefore needs to be considered.

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