PREVALENCE OF HYPOMAGNESAEMIA IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION COMPARED WITH NORMAL SUBJECTS

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ABSTRACT

Background: Magnesium, the second most abundant intracellular cation has several clinically important roles in the human body. In addition to energy production and maintenance of electrolyte balance, Magnesium (Mg^{++}) is essential for normal neuromuscular functions, excitation contraction coupling, maintenance of vascular tone, blood coagulation, as well as Ca^{++} and K^{+} transport across the plasma membrane. Mg^{++} has also important role to play in cardiovascular homeostasis. **Objectives:** To find out serum Mg^{++} levels in Acute Myocardial Infarction (AMI) patients at presentation to emergency departments and its comparison with normal subjects. **Patients and Methods:** This was a comparative study, conducted at Punjab Institute of Cardiology, Lahore from June to December 2000. A total of 125 subjects were enrolled for this study, with 88 patients having their first episode of AMI. These patients were divided into three groups A, B and C depending on age. 37 normal subjects were taken as comparative group. **Results:** There was hypomagnesemia ($p< 0.001$) in all the sub groups. Our results show that hypomagnesemia is present in all groups and as it is an important risk factor for post AMI complications. The corrective dose should not be empirical but be based on individual patient requirements, as mortality rate due to AMI and its complications is high. **Conclusion:** Hypomagnesemia was observed in all the sub groups A, B and C. Therefore, it is suggested that serum Magnesium should be estimated in each case of AMI patient and emphasis given to Mg^{++} supplementation when needed.

Keywords: Hypomagnesemia, Acute Myocardial Infarction, Serum Mg^{++}

INTRODUCTION

Magnesium (Mg^{++}), the second most abundant intracellular cation, has several critically important roles to play in the human body, like energy production, maintenance of serum sodium, serum calcium, serum potassium and smooth muscle tone in the vessel wall. Magnesium is essential for normal neuromuscular function and Ca^{++} and K^{+}transport across the plasma membrane.[1]

Evidence suggests that deficiency of Mg^{++} (called hypomagnesemia) is closely related to K^{+} deficiency and refractory potassium repletion. Although the consequences of hypokalemia are widely documented and recognized, the importance of magnesium deficiency, as a cause of potassium (K^{+}) depletion has gained clinical attention only recently.[2]

Hypomagnesemia, is present in acute Myocardial Infarction (AMI) as shift of Mg^{++} from extra cellular to intracellular compartments occurs. It is taken up by adipocytes after catecholamine induced lipolysis occurs, combined with soap formation by free fatty acids.[2]

Although the total body Mg^{++} content may not change with the onset of AMI, extra cellular Mg^{++} declines markedly, especially over the first 24 to 48 hours, after the onset of AMI. Hypomagnesemia in the initial phase of post AMI period is very critical, as ventricular tachyarrhythmia's, sudden cardiac death, and re-infarction are the usual outcome.[3]

Mg^{++} level in the extra cellular compartments is maintained by parathormone. Primary organs for Mg^{++} handling are gastrointestinal tract and the renal system. Hypocalcemia and hypomagnesemia may be concomitantly present.[4-8] Development of hypokalemia and hypomagnesemia in patient of AMI is life threatening as ventricular tachyarrhyamias and sudden cardiac death may result.[9-13]

Mg^{++} produces coronary vasodilatation and smooth muscle relaxation in coronary arteries and hence help in control of cardiac arrhythmias. These beneficial effects of Mg^{++} are due to the direct local influence of Mg^{++} & increased perfusion in turn reduces the infarct size.[14-18]

Experimental models of AMI in at least four different animal species have shown that Mg^{++} administration before coronary occlusion with concomitant perfusion for short interval time (15 to 45 minutes)

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after reperfusion reduces the infarct size and prevents myocardial stunning due to reperfusion injury. However, a 45 minute interval after reperfusion is no longer effective in reducing the myocardial damage.[19-25]

Present study was conducted to assess serum Mg^{++} levels in patients of AMI at presentation to the Emergency Department and its comparison with normal subjects.

PATIENTS AND METHODS

A total of 123 subjects were enrolled for this comparative study. 88 patients experienced acute Myocardial Infarction for the first time. These patients were classified into three groups A, B and C based on age, Group A: age 20-40 years, Group B:40-60 years and Group C:> 60years. They were compared to 37 normal subjects (control group). Study was conducted at Punjab Institute of Cardiology, Lahore, in the emergency department during June-December 2000. The patients with chronic renal failure, congestive cardiac failure, diabetes mellitus and those on diuretics were excluded.

Just after admission of each patient proper history was taken and ECG was recorded, after fulfilling the criteria specified by JCCS-2000 of AMI diagnosis. 4cc whole blood sample was taken prior to the start of treatment for estimation of serum Mg^{++}. Samples were centrifuged at 4000 RPM for 2-3 minutes, serum was isolated and Mg^{++} was estimated by colorimetric method using calmagite.

RESULTS

In this study, there were a total of 123 study subjects classified into three groups based on age. The serum Mg^{++} levels in sub group A was highly significantly low ($p<0.001$) (Table I and Figure II) as compared to the control at the time of admission and further low ($p<0.001$) after six hours of admission. Similarly, serum Mg^{++} levels in subgroup B (Table II) as well as subgroup C(Table III) were highly significantly low ($p<0.001$). Figure II shows serum magnesium levels in sub groups A, B & C at admission of AMI patients.

Table I: Value of serum magnesium in patients of AMI at the time of admission: Control versus Group A (20 - 40 years)

Serum Mg++ (Mg/dl)	Normal Subjects (n=37)	Sub group A (N=15)	P-Value
At admission (mg/dl)	2.014 ± 0.11	1.037± 0.13	P<0.001

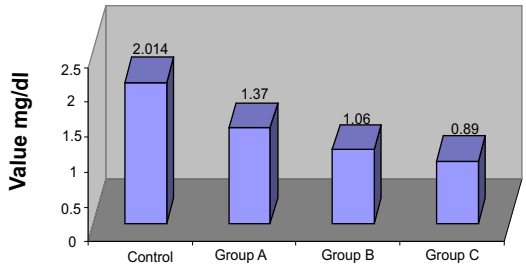
Table II: Value of serum magnesium in patients of AMI at the time of admission: Control versus Group B (40 - 60 years)

Serum Mg++ (Mg/dl)	Normal Sujects (n=37)	Sub group B (N=38)	P-Value
At admission (mg/dl)	2.014 ± 0.11	1.063± 0.59	P<0.001

Table III: Value of serum magnesium in patients of AMI at the time of admission in the Emergency ward: Control versus Group C (60 + years)

Serum Mg++ (Mg/dl)	Normal Subjects (n=37)	Sub group C (N=15)	P-Value
At admission (mg/dl)	2.014 ± 0.11	0.89± 0.22	P<0.001

Fig. II. Mean Values of Serum Magnesium in Patients of AMI at admission in Groups A, B and C versus control



DISCUSSION

Hypomagnesemia is an important risk factor for post AMI complication. It has been reported in various international studies that the serum Mg^{++} level is not only low at admission in cases of AMI but also continues to fall even for days after the onset of AMI.[22] The mean value of serum Mg^{++} levels in all three groups in our study was significantly low ($p<0.001$) as compared to the control. This finding is consistent with the above mentioned study.

It is however, apparent from this study that serum Mg^{++} plays an important role in cardiac homeostasis and its deficiency is capable of producing myocardial injury and post AMI arrhythmias.[8] There is growing evidence that hypomagnesemia acts as an important risk factor to cause serious cardiac disturbances. So, serum Mg^{++} levels should be estimated in each case of AMI and hypomagnesemia, when present, should be corrected with Mg^{++} supplements according to the

individual serum magnesium levels. This not only reduces the risks of post AMI complications but also decreases the rate of sudden cardiac death.[23] Number of cardiac arrhythmias are also significantly decreased.[10] In most of the patients in post-AMI phase, there is concomitant hypokalemia. If the serum Mg^{++} is not corrected, the hypokalemia repletion becomes refractory.[6] Magnesium should be reclassified as 5th major electrolyte of human body after Na^{+}, K^{+}, Ca^{++} and Cl^{-}.[6] As all the patients of AMI received the thrombolytic therapy, post-AMI complications could not be observed in our study. However, AMI patients in whom hypo-magnesemia was observed the intravenous infusion of Mg^{++} was given to correct it.

CONCLUSION

High prevalence of hypomagnesemia in cases of AMI, as detected in this study, highlights the importance of measuring serum Mg^{++} levels and its correction in every patients presenting with AMI, for preventing potential fatal complications.

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