Int J Pharm Bio Sci 2013 Apr; 4(2): (B) 1190 - 1195

Research Article **BioChemistry**

International Journal of Pharma and Bio Sciences **ISSN 0975-6299**

SERIAL ESTIMATION OF SERUM MAGNESIUM, CALCIUM, SODIUM AND POTASSIUM LEVELS IN MYOCARDIAL INFARCTION

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ABSTRACT

Background: Acute Myocardial Infarction (AMI) is a major public health problem. Fifty percent of deaths associated with AMI are attributable to arrythmias which result due to myocardial ionic imbalance of cations Na^{+},Mg^{+2}, Ca^{+2}, and K^{+}. Present study aims to find serial change in cation levels in MI cases and their relationship to arrythmias. Methods: Seventy five patients admitted to Intensive Coronary Care Unit (ICCU) with provisional diagnosis of acute myocardial infarction constituted the study group. They were studied serially on 1st, 3rd and 7th day of infarction. Thirty subjects without any heart disease and renal pathology, who were seen in the outpatient departments and healthy volunteers were taken as controls. S.Mg+2, Ca+2, Na+ and K+ were measured in controls and study group and compared. Results: Mean values of all the cations were lowered on 1st day of MI in study group compared to controls ($p<0.01$). A strong association between low Mg+2 and low K+ levels in MI cases and development of arrythmias is observed ($p<0.001$). Conclusions: Serial estimation of S.Mg+2 can be used as a diagnostic test for MI. Estimation of S.Mg+2 and S.K+ can be used as prognostic markers in MI.

KEYWORDS: **Acute myocardial infarction, arrythmias, Serum magnesium, Serum potassium**

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INTRODUCTION

Acute myocardial infarction (AMI) is a major public health problem in the industrialized world and is becoming an increasingly important problem in developing countries. Mortality rate in AMI cases is high within one hour of the event and is due to development of arrhythmiasAtherosclerosis is a well-known precursor of ischemic heart disease, stroke and sudden cardiac death. 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. Electrical derangements of the myocardium caused during infarction is a major cause of sudden death., and is probably directly related to myocardial ionic imbalance of Cations Na^+, Mg^{2+} , Ca^{2+} and K^+ which are important in maintaining the resting membrane potential of myocytes. The present study was therefore undertaken to note any changes in the levels of Serum Magnesium, Serum Calcium, Serum Sodium and Serum Potassium in patients with first attack of AMI serially on 1st, 3rd and on 7th day after infarction. The objective was also to study the relation if any, between the Serum Magnesium concentration and the arrhythmias recorded during the first 24 hours in Intensive Coronary Care Unit (ICCU).

MATERIALS AND METHODS

Study was conducted in 75 patients (n=75) admitted to the intensive coronary care unit (ICCU) of department of Cardiology and Acute medical care unit department of Medicine ,Osmania General Hospital, with chest pain and provisional diagnosis of Acute Myocardial Infarction (AMI). All were electrocardiographically proved cases of AMI ranging in age from 32-76 years with mean age of 54.1±11.2 (Males-45 and Females-30). Myocardial infarction was defined as two or more of following criteria: 1) typical chest pain, 2) ST elevation of 0.1 mv or more in at least one standard or two precordial leads of a 12 lead electrocardiogram (ECG) or 3) biochemical markers indicative of Myocardial ischaemia.[1] Patients without serious disease who were seen in the outpatient departments and healthy volunteers were taken as controls (n=30) with mean age of 49±9.9. Diabetics were excluded and no control subject has a history or electrocardiographic evidence of ischemic or rheumatic heart disease, hypertension, congestive heart failure or renal failure. Venous blood was collected under aseptic precautions after consent is taken and Random blood sugar (RBS), Blood Urea and Serum Creatinine were estimated to rule out diabetes and renal pathology among controls. S. Magnesium was estimated by calmagite kit method. S.calcium was estimated by o – cresol pthalein kit method and S. electrolytes were estimated by flame photometry method. Random blood sugar was measured by King and Asatoor method. Blood urea was measured by Di acetyl monoxime (DAM) colorimetric method[2] and serum creatinine by Jaffes alkaline picrate method[3] All the 75 patients were studied serially on 1st day of admission , 3rd day and 7th day after AMI. 5ml of venous blood was collected on each occasion in a plain sterile bottle and allowed to clot. Serum was separated immediately for estimation of serum creatinine, serum magnesium, calcium and electrolytes. Two ml of blood was taken in oxalate fluoride bottles on 1st day for estimation of Blood sugar and Blood urea.

RESULTS

In control group the mean serum Magnesium was 1.08 ± 0.26 mmol/litre and mean Ca^{+2} was 9.45±1.134 mg%. Mean serum Na^+ and K^+ were 137.38 ± 3.67 meq/l and 4.21 ± 0.394 meq/L respectively. Study group (n=75) is subgrouped into Ia, Ib, Ic i.e 1st day, 3rd day and 7th day

follow up study respectively. Statistical analysis was made between control and study subgroups. (Table-1). Analysis is performed by student t-test. There is significant decrease in all the parameters on 1st day of myocardial infarction ($p<0.01$) with more significance ($p<0.001$) in case of serum Magnesium and Serum Potassium levels,but there was no significant difference between control and groups Ib and Ic($p.>0.05$) in all the parameters. Intra group comparisons are showing a progressive increase in serum Magnesium, serum K^+,S.Na^+ and S.Ca^{+2} levels by 7^{th} day of follow up reaching near control values.Table-1 Comparison of Serum Magnesium, Calcium, Sodium & Potassium levels in control & Study groups (** $p<0.01$, *** $p<0.001$) Out of 75 patients arrhythmias were reported in 25 cases (n=25)(33.34%)[13 males and 12 females]. Three patients died on 7th day of follow up due to severe ventricular fibrillation. So mortality in arrhythmia cases was 12 %. Ventricular arrhythmias developed in total 17 patients (68 % of arrythmias). Supraventricular tachycardia (SVT) in 1 patient (4%). Atrio Ventricular Block (AV block) and Bundle branch blocks developed in 7 patients (28 %).

Table 1

Parameter	*Control Group (n=30)*	*Study Group (n=75) Group I*		
		Ia (1^{st} day)(n=75)	*Ib (3^{rd} day) (n=75)*	*Ic (7^{th} day) (n=75)*
Serum Magnesium (mmol/L)	*1.08 ± 0.26*	*0.74 ± 0.21***	*0.956±0.176*	*1.11±.21*
Serum Calcium (mg/dl)	*9.45 ± 1.13*	*7.04 ± 0.84***	*8.6±0.84*	*9.34±0.97*
Serum Sodium (meq/L)	*137.38 ± 3.67*	*129.1 ± 10.12***	*136 ±6.55*	*139±4.05*
Serum Potassium (meq/L)	*4.21 ± 0.39*	*2.92 ± 0.76****	*3.41±0.59*	*3.69±0.601*

Comparison of parameters in cases with and without arrhythmias shows a significant lowering of S.Mg^{+2} in arrhythmic cases with mean value 0.70 ± 0.24 when compared to mean value incases without arrythmias (p<0.01)

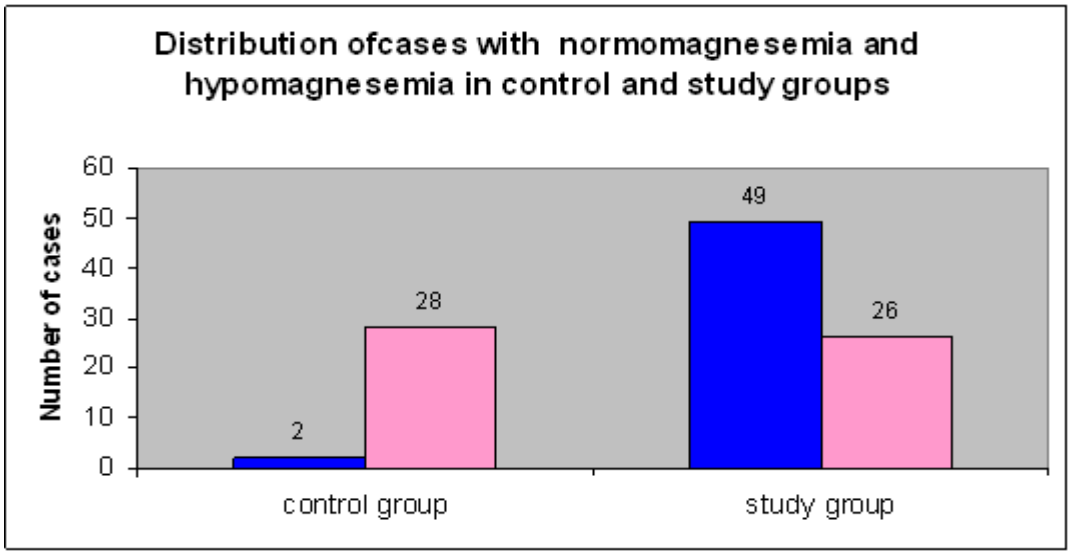
Table 2

Groups	S.Magnesium (mmol/L)	S.Calcium (mg/dl)	S.Sodium meq/L	S.Potassium meq/L
With out Arrythmias (n=50)	0.78 ± 0.26	7.92 ± 0.88	129.4± 10.5	2.94 ± 0.75
With Arrythmias (n=25)	0.70 ± 0.24**	8.34 ± 1.09	128.6± 11.08	2.87 ± 0.52*

Comparison of Serum Cation levels in cases with & without arrythmias

There is also significant lowering of S.K^+ in cases with arrhythmias ($p<0.05$). But there is no significant change in other parameters. (Table-2) Hypomagnesemia[4] is seen in 65.3 % of study group as against only 6.67% in controls ($P<0.001$] and hypokalemia in 60 % of cases compared to only 10 % in controls ($p<0.05$). (Fig-1)

Figure 1



Occurrence of arrhythmias in hypokalemic cases was 51.11 % and that in normokalemic cases 6.66 %..This shows a significant occurrence of arrhythmias in patients with hypokalemia. Out of 25 cases with arrhythmias 18 cases (72%) showed hypomagnesemia and 7 cases (28 %) demonstrated normal serum magnesium levels. This shows a significant association between S.Magnesium levels and arrhythmias ($p<0.01$).Similarly $S.K^+$ concentration was less in 23 cases out of 25 (92 %) and normokalemia was seen in two cases (8 %) showing significant correlation between arrhythmias and $S.K^+$ levels.

DISCUSSION

The results of this study demonstrates a significant lowering of the Serum Mg^{+2} levels on the 1st day of Myocardial infarction in accordance with several previous studies, [5,6,7,8] but in contrast to some other studies.[9,10] Magnesium depletion is an etiological factor in the development of IHD.[11,12,13] Flink et al[5] presented a study of the relation between free fatty acids (FFA) and $S.Mg^{+2}$ in patients with AMI. They demonstrated a rise in FFA and a concomitant decrease in $S.Mg^{+2}$ levels.Both parameters returned to normal on the 3rd day of observation. These results were interpreted as a binding of Mg^{+2} to FFA, which will yield an insoluble complex. One may speculate whether the rise in catecholamines in AMI activates the enzyme adenyl cyclase, which brings about an increased synthesis of c-AMP, c-AMP then induces lipolysis, thus increasing FFA which according to Flink et al, could chelate Mg^{+2} and thus gives a lowered serum Mg^{+2} level.The reduced $S.Mg^{+2}$ levels in AMI do not however reflect a total body Mg^{+2} deficiency. This is evidenced by the return to normal values within 2 weeks of the AMI event. [5,14]In the present study the mean $S.Mg^{+2}$ level was lowered on 1st day of infarction and its serial determination on subsequent days showed a progressive rise which came to near normal on 7th day of follow up. This is in accordance with Nath et al.[7] Hughes , Tonks and Kotia et al reported significantly low levels of $S.Mg^{+2}$ in AMI cases.[6] as $S.Mg^{+2}$ is utilized to depress clotting by substrate competition with calcium (Anstall et al, 1959) and to inhibit platelet aggregation (Heinrich cited by Browne, 1969). Both these processes are exaggerated in AMI. In this study low potassium $\leq$3.5 meq/L is reported in 39 cases i.e (70% of patients) on the 1st day of AMI. This is in accordance with previous studies.[15] The fall in serum potassium levels in AMI is related to high circulating catecholamine levels.[16] This is an acute stress effect and is

due to shift of potassium from extracellular to intracellular space and is a result of stimulation of beta-2 adreno-receptors linked to Na/KATPase. [15] Decreased Magnesium causes hypokalemia by promoting cellular and renal loss of K^+. Low calcium levels are observed in the present study on 1st day of MI. This finding supports the statement cited in Drug facts and comparison. [17] stating that hypocalcemia follows low Serum levels of magnesium. Hypomagnesemia causes hypocalcemia by its effect on parathyroid hormone.[23] Low Sodium levels found on 1st day of MI is because of use of diuretics or due to imbalance of Na^+-K^+ ATpase activity due to lowered S.Magnesium levels. [11]Magnesium has been shown to have a depressing action on both the S.A and the A.V node[19] and it also blocks the sympathetic ganglionic transmission at the stellate ganglion, thus slowing the heart. Previous studies [8, 20] found a significant decrease in the S.Mg^{+2} levels in AMI, but no relation to arrythmias. In this study 33.34 % of AMI cases developed arrythmias with ventricular arrhythmias contributing to 68 % of arrythmias. Increased incidence of ventricular arrythmias in AMI cases with significant hypomagnesemia of ≤ 0.8 mmol/L is due to effects of magnesium on membrane ATPase resulting in changes in the potassium kinetics. The prolongation of QT interval observed in these cases is mediated through disturbances in the membrane transport of potassium. A prolonged QT interval is known to predispose to aberrant conduction, reentry and ventricular fibrillation (VF). [21, 22] A low Serum Potassium affects the cell resting potential, which is mainly determined by the ratio of extra to intracellular Potassium. If this ratio is decreased through a fall of S. K^+ with no or a smaller fall of intracellular Potassium, the resting potential of excitable cells will decrease, which results in hyperpolarisation.[23]

SUMMARY AND CONCLUSIONS

There is a significant decrease in Serum Magnesium levels on 1st day of MI which has progressively increased to near control values by 7thday. Hence serial estimation of S. Magnesium can be used as a diagnostic test for MI. There is also significant lowering of S. Potassium levels on 1st day of MI which is due to factors like diuretics, disturbed gastrointestinal absorption or due to acute stress effect of Myocardial Infarction which is usually transient and may resolve without Potassium supplements. Other electrolytes like S.Na^+ and S.Ca^{+2} also showed low values which resulted due to lowered magnesium levels. In present study a strong association between low Mg^{+2} and low Potassium levels in MI cases and development of arrhythmias is observed. Hence these cation estimations can be used as prognostic markers in MI cases. As repletion with Mg^{+2} alone results in increased levels of both K^+ and Mg^{+2},this study suggest's supplementation of Magnesium to patients who present with MI ,which also corrects hypokalemia and thus alleviates development of arrhythmias .

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