

# ACUTE MYOCARDIAL INFARCTION; SERUM MAGNESIUM AND ELECTROLYTE LEVELS AT PRESENTATION IN EMERGENCY DEPARTMENT

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**ABSTRACT... Background:-** Magnesium, the second most abundant intracellular cation have several critically important roles in human body. In addition to energy production and maintenance of electrolyte balance, magnesium ( $Mg^{++}$ ) is essential for normal neuromuscular function, excitation contraction coupling maintenance of vessel tone, blood coagulation as well as  $Ca^{++}$  and  $K^+$  transport across the plasma membrane. **Study Design:** Case control study. **Material and Methods:-** A total of 125 subjects enrolled for this study, 88 were AMI patients having their first episode. 37 subjects were taken as control group. These patients were classified into three groups depending on the age of the subjects. **Aims and objectives:-** To find out serum magnesium and electrolyte levels in AMI patients at presentation to Emergency Department. **Discussion:-** Hypomagnesaemia is a common clinical finding in cases of acute myocardial infarction at their presentation to the emergency department of Punjab Institute of Cardiology, Lahore. It is very critical as ventricular tachyarrhythmias cardiac arrest, sudden cardiac death, re-infarction are the usual outcome. **Conclusion:-** There is Hypomagnesemia ( $P < 0.001$ ) in all the subgroups while for  $Na^+$  and  $K^+$  ( $P > 0.05$ ). Our result explains the contribution of hypomagnesaemia as a minor risk factor for AMI. Therefore it is suggested that serum magnesium should be estimated in each case of AMI patient. Importance may be given to  $Mg^{++}$  supplementation where needed. The dosage should not be empirical but be based on individual patient requirement.

**Key words:-** Hypomagnesemia, AMI, Serum  $K^+$ , Serum  $Na^+$ , Hypokalemia.

## INTRODUCTION

Magnesium ( $Mg^{++}$ ) is the second most abundant intracellular cation, have several critically important roles in the body. In addition to the energy production-adenosine triphosphate (ATP), maintenance of serum sodium, serum calcium, serum potassium, smooth muscles tone in the vessel wall magnesium is essential for normal neuromuscular function and  $Ca^{++}$  and  $K^+$  transport across the plasma membrane.

Evidences suggest that deficiency of magnesium called hypomagnesaemia is closely related to the potassium

deficiency and refractory potassium repletion.

Although the consequences of hypokalemia are widely documented and recognized. It is only recently that the importance of Magnesium deficiency as a cause of potassium ( $K^+$ ) depletion has gained clinical attention<sup>1</sup>.

Hypomagnesemia is present in acute myocardial

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infarction (AMI) as shift of magnesium from extra cellular to intracellular compartments occur as it is taken up by adipocytes after catecholamine induced lipolysis and combined with soaps formed by free fatty acids<sup>2</sup>. Although the total body  $Mg^{++}$  contents 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 sudden cardiac death and re-infarction are the usual outcome<sup>3</sup>.

Magnesium level in the extra cellular compartment is maintained by parathormone (PTH) while the primary organs involved for  $Mg^{++}$  handling are gastrointestinal tract and the renal system. Hypomagnesemia is not uncommon in patients with AMI. Hypokalemia and Hypomagnesemia are concomitantly present<sup>4,5</sup>.

There is high incidence of Hypomagnesemia in subjects due to the development of acute myocardial infarction, which leads to hypokalemia. The probable explanation of this finding may be the catecholamine storm which induces lipolysis and free fatty acids soap formation<sup>6,7,8,9</sup>. This catecholamine storm accompanied by the sympathetic over-activity pushes  $Mg^{++}$  as well as  $K^+$  intracellularly thus causing a relative deficiency of both these cations in the extra cellular compartment.

Developments of both states of hypokalemia and Hypomagnesemia are patients of AMI are life-threatening. Ventricular tachyarrhythmia and sudden cardiac death occurs in this period<sup>10,11,12,13</sup>.  $Mg^{++}$  produces coronary vasodilatation and helps to control cardiac tachyarrhythmia. These beneficial effects of  $Mg^{++}$  are due to the direct local influence of  $Mg^{++}$  myocardial perfusion which in turn reduces the infarct size<sup>14,16,17</sup>.

Experimental models of AMI in at least four different animal species have shown that supplemental administration of  $Mg^{++}$  before coronary occlusion, during coronary occlusion concomitant with reperfusion for a short time interval (15-45 minutes) after reperfusion reduces the infarct size and prevents myocardial

stunning due to reperfusion injury.

However, delayed administration of  $Mg^{++}$  beyond a specified time (15-60 minutes), after reperfusion is no longer effective in reducing myocardial damage<sup>21,22,23,24,25</sup>.

### AIMS AND OBJECTIVES OF THE STUDY

1. Study of Serum  $Mg^{++}$ ,  $Na^+$  and  $K^+$  levels in patients of AMI at presentation to the Emergency Department.
2. Study role of low levels of  $Mg^{++}$  in AMI patients especially in development of post AMI arrhythmia, re-infarction or sudden cardiac death.
3. Any correlation in Hypomagnesemia and hypokalemia in post AMI phase.

### MATERIAL AND METHODS

A total of 125 subjects enrolled for this study, 88 were AMI patients having their first episode. 37 subjects were taken as control group. These patients were classified into three groups as compared to the control group. Study was conducted in Emergency Department of Punjab Institute of Cardiology, Lahore in June-December, 2000.

Patients preferably having first episode, males of almost all ages were included in the study. Patients of known/IHD, diabetes mellitus, chronic renal failure, hypertension or taking diuretic or digoxin for any purpose were excluded from the study. The study group was further divided into three subgroups A, B and C depending on age of the patients.

Just after the admission of such selected patients of AMI, ECG was recorded after fulfilling the JCCS-2000 criteria of AMI diagnosis, thorough general physical examination systemic examination was made and proforma properly filled. Keeping in view of the inclusion and exclusion criteria before initiation of the specific therapy for AMI, 4CC blood sample was taken in all such patients for the estimation of serum  $Mg^{++}$ ,  $Na^+$  and  $K^+$  such samples were centrifuged at 4000 rpm for 2-3 minutes, serum isolated and preserved and proper record was maintained in each case.

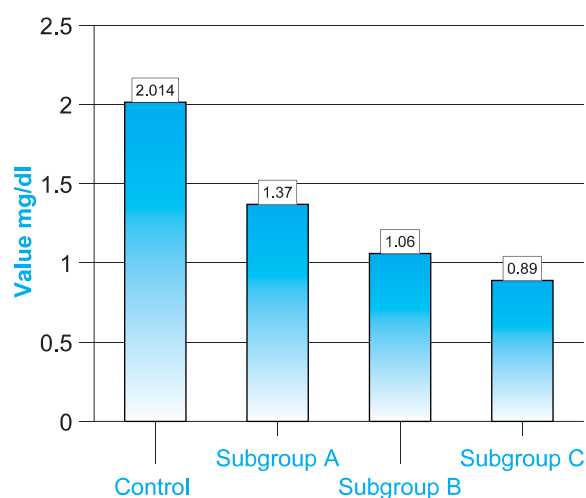
After the collection of all these samples (in June-December, 2000), serum  $Mg^{++}$  was estimated by colorimetric method using calmagite while estimation of serum  $Na^+$  and  $K^+$  was done on principle based on the potential developed in the selective electrodes and reference electrodes by using standardized methods of estimation.

## RESULTS

The serum magnesium levels in subgroup A was highly significantly low ( $P < 0.001$ ) (Table I & Fig I) as compared to the control at the time of admission. Serum magnesium level in subgroup B ( $P < 0.001$ ) also showing highly significant result (Table No. II & Fig I) while in subgroup C the results were highly significant ( $P < 0.001$ ) (Table III & Fig. I).

Serum sodium and potassium levels in subgroups A and B were non significant ( $P > 0.05$ ) while in subgroup C, there was hypokalamic tendency (Table IV, V and VI & Fig. II).

**Fig-1. Mean values of serum magnesium in patients of AMI at admission in subgroup A, B and C versus control.**



**Table I- Value of serum magnesium in patients of AMI at the time of admission in the Emergency Ward control versus Group A**

Serum $Mg^{++}$ (mg/dl)	Control (n=37)	Subgroup A (n=15)	P-Value
At admission (mg/dl)	2.014 ± 0.11	1.376 ± 0.13	$P < 0.001$

*Shows  $P < 0.001$  highly significant.*

**Table-II. Value of serum magnesium in patients of AMI at the time of admission in the hospital control versus versus Group B**

Serum $Mg^{++}$ (mg/dl)	Control (n=37)	Subgroup B (n=38)	P-Value
At admission (mg/dl)	2.014 ± 0.11	1.063 ± 0.59	$P < 0.001$

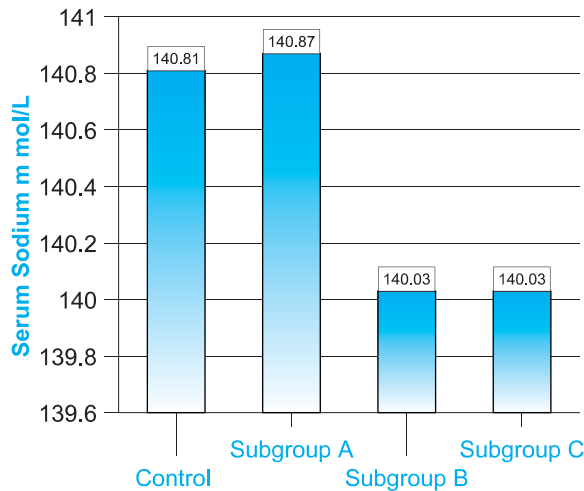
*Shows  $P < 0.001$  highly significant.*

**Table-III. Value of serum magnesium in patients of AMI at the time of admission in the Emergency Ward control versus Group C**

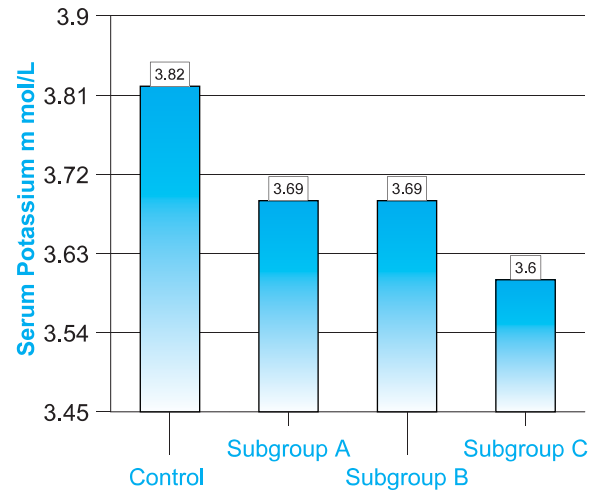
Serum $Mg^{++}$ (mg/dl)	Control (n=37)	Subgroup C (n=15)	P-Value
At admission (mg/dl)	2.014 ± 0.11	0.89 ± 0.22	$P < 0.001$

*Shows  $P < 0.001$  highly significant.*

**Fig-2. Mean values of serum sodium in patients of AMI versus control in subgroup A,B and C.**



**Fig-3. Mean values of serum potassium in patients of AMI versus control in subgroup A,B and C.**



**Table-IV. Value of serum Na<sup>+</sup> and serum K<sup>+</sup> in patients of AMI in subgroup A versus control**

Serum Electrolyte	Control (n=37)	Subgroup A (n=15)	P-Value
Serum Sodium Na <sup>+</sup> m mol/L	140.81 ± 0.62	140.87 ± 1.1	P = 0.97 NS
Serum K <sup>+</sup> m mol/L	3.822 ± 0.1	3.699 ± 0.17	P = 0.53 NS

*Both values of Na<sup>+</sup> and K<sup>+</sup> in serum of controls and subgroup A are non- significant*

**Table-VI. Value of serum Na<sup>+</sup> and serum K<sup>+</sup> in patients of AMI at admission in subgroup C versus control**

Serum Electrolyte	Control (n=37)	Subgroup A (n=15)	P-Value
Serum Sodium Na <sup>+</sup> m mol/L	140.81 ± 0.62	140.03 ± 0.84	P=0.14 NS
Serum K <sup>+</sup> m mol/L	3.822 ± 0.1	3.609 ± 0.016	P=0.41 Sig

*Both values of Na<sup>+</sup> and K<sup>+</sup> in serum of controls and subgroup C are non- significant*

**Table-V. Value of serum Na<sup>+</sup> and serum K<sup>+</sup> in patients of AMI at admission in subgroup B versus control**

Serum Electrolyte	Control (n=37)	Subgroup A (n=15)	P-Value
Serum Sodium Na <sup>+</sup> m mol/L	140.81 ± 0.62	140.87 ± 0.84	P=0.46 NS
Serum K <sup>+</sup> m mol/L	3.822 ± 0.1	3.699 ± 0.76	P=0.09 NS

*Both values of Na<sup>+</sup> and K<sup>+</sup> in serum of controls and subgroup B are non- significant*

**DISCUSSION**

Serum magnesium in cases of AMI at presentation to the Emergency Ward has been significantly decreased (P<0.001). It has been observed in various international studies that the serum magnesium Mg<sup>++</sup> level is not only low at admission in cases of AMI but also continues to fall even for days after the onset of AMI. (Woods 1993, LIMIT, Tril 1993). The mean value of the serum magnesium level in all three groups A,B and C were significantly low (P<0.001) as compared to control. This finding is also in consistency with the studies of Manthey et al (1981) who have shown that serum magnesium is also low in our studygroup.

It is however, apparent from this study that serum magnesium plays an important role in the cardiac homeostasis and its deficiency is capable of producing myocardial injury and postAMI arrhythmias (Boom 1999). There is growing evidence that Hypomagnesemia acts as an important risk factor to cause serious cardiac disturbances and the drug digitalis toxicity (Seelig MS, 1989). The findings of this study is important as it is a common clinical practice to order supplemental potassium therapy for cardiac patients receiving K<sup>+</sup> losing diuretics, particularly when digitalis preparations are being administered concomitantly.

Considerations should be given to Mg<sup>++</sup> supplementations for such patients since Hypomagnesemia besides causing cardiac arrhythmias also predisposes to digitalis intoxication the supplemental dosage should not be empirical but should be based on the need of the individual patient and also after periodic estimation of serum magnesium levels (Zipes 2002).

Considering the significant low levels of serum magnesium in the patients of AMI (P<0.001) in the present study, it is therefore suggested that it is the responsibility of the attending clinician that the use of diuretics should be done carefully and preferably undertaking prior estimation of serum magnesium Mg<sup>++</sup> (Iseri and French, 1984).

The levels of serum sodium in all subgroups of all AMI are non significant, while (P = 0.041) significant in group C, the maintenance of serum K<sup>+</sup> within normal range in cardiac patients is gaining increasing importance as hypokalemia predisposes of a number of cardiac arrhythmias (Brawnwald 2002). In most of the patients in post AMI phase there is concomitant Hypokalemia. If the serum Mg<sup>++</sup> is not corrected, the hypocalcaemia repletion becomes refractory (Barkley 2004).

It is a routine clinical practice to correct the acidosis in patients of AMI by administration of intravenous bicarbonates. The excessive use of bicarbonates can itself decrease serum K<sup>+</sup> to its shift into the cell particularly into the acidosis patients (Freitag and Miller, 1980). In

view of the recent study, great care should be exercised to correct the acidosis in the patients of AMI and preferable should be accompanied by repeated estimation of serum K<sup>+</sup> to avoid the dangerous complications of hyperkalemia (Nordehang and Vonder L, 1983). Zipes et al (2001) time period crucial for the estimation of serum Mg<sup>++</sup> should be done as early as possible, after arrival of the patient to Emergency Ward. Magnesium should be regarded as 5<sup>th</sup> electrolyte of human body after Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>++</sup> and Cl<sup>-</sup> (Barkley 2004).

### CONCLUSION

1. Serum magnesium levels in all the cases of AMI in all subgroups A,B and C were significant low (P<0.001) at the time of presentation of such patients to the emergency ward as compared to control.
2. There was hypokalemic tendency in patients with AMI above the age of 60 years (group C) while the results of serum Na<sup>+</sup> and K<sup>+</sup> were non significant in group A and B.
3. The postAMI complications were not observed in such cases as thrombolytic therapy was given and also intravenous serum magnesium given to hypomagnesaemia patients.

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