Serum Magnesium Level in Type II Diabetes Mellitus

*Maula MG,[1] Sarkar CR,[2] Zahid AR,[3] Ahmed A,[4] Aktar MS[5]

Non-insulin dependent diabetes mellitus (NIDDM) or type II diabetes mellitus is 90% to 95% of all the diagnosed diabetes mellitus. Hypomagnesaemia is associated with type II DM but a significant population suffering from this metabolic disorder is not aware about it. This cross sectional study was conducted in the Department of Physiology, Rangpur Medical College, Rangpur, from July 2010 to June 2011 to measure serum magnesium level in non-insulin dependent diabetes mellitus. A total number of 100 subjects were selected, among which 50 were patients of type II diabetes mellitus and 50 were age matched apparently healthy non-diabetic subjects for comparison. Mean serum magnesium level in type II diabetes mellitus was significantly ($p < 0.001$) lower than that of the healthy non-diabetic subjects.

[Dinajpur Med Col J 2013 Jul; 6 (2):123-127]

Key words: Serum magnesium, Type II DM

Introduction

Diabetes mellitus is a syndrome of impaired carbohydrate, fat and protein metabolism caused by either lack of insulin secretion or decreased sensitivity of the tissues to insulin.[1] Although diabetic patients can have a reasonably normal lifestyle, its late complications result in reduced life expectancy and major health costs. These include macro vascular diseases leading to an increased prevalence of coronary artery disease, peripheral vascular disease, stroke and micro vascular damage causing diabetic retinopathy, nephropathy and contributing to diabetic neuropathy.[2] There are two types of diabetes mellitus: a) Type I diabetes is caused by lack of insulin secretion. b) Type II diabetes is caused by decreased sensitivity of target tissues to the metabolic effect of insulin. This reduced sensitivity to insulin is often called insulin resistance.[1] Type II DM is a more complex condition than type I DM, because there is a combination of resistance to the action of insulin in liver and muscles together with impaired pancreatic B-cells function leading to relative insulin deficiency.[3] Type II DM is on track to become one of the major global public health challenges of the 21st century.[4]

1. **Dr. Md. Golam Maula, Assistant Professor, Department of Physiology, Barind Medical College, Rajshahi.* drzahid63@gmail.com
2. *Dr. Chandra Rani Sarkar, Professor, Department of Physiology, Rangpur Medical College, Rangpur.*
3. *Dr. A.T.M. Zoadur Rahim Zahid, Associate Professor, Department of Physiology, Rangpur Medical College, Rangpur.*
4. *Dr. Afroza Ahmed, Assistant Professor, Department of Physiology, Rangpur Medical College, Rangpur.*
5. *Dr. Mst. Sultana Aktar, Lecturer, Department of Physiology, Rajshahi Medical College, Rajshahi.*

* *For correspondence*

Magnesium is the fourth most abundant cation in the extra cellular body fluid and second in the intracellular environment. It serves as a co-factor for all enzymatic reactions that require ATP and as a key component in various reactions that require kinase.[5] In type II DM patients may result in defective tyrosine kinase activity at the insulin receptor level and exaggerated intracellular calcium concentration. Both events are responsible for the impairment in insulin action and a worsening of insulin resistance in type II DM.[6] Magnesium is an essential ion involved on multiple levels in insulin secretion, binding and activity and is a critical cofactor of many enzymes in carbohydrate metabolism.[7] Magnesium depletion has a negative impact on glucose homeostasis and insulin sensitivity in patients with type II DM, as well as on the evolution of complications such as retinopathy, thrombosis and hypertension. Moreover low serum magnesium is a strong independent predisposing factor for insulin resistance and developing of type II DM.[8]

There is a link between diabetes mellitus and magnesium deficiency.[9] A good number of studies in other countries [4-9] have demonstrated high prevalence of serum magnesium deficiency in type II diabetic patients but no such published data is available in Bangladesh. So, the present study has been designed to measure serum magnesium level in Type II DM patients. It will be helpful to increase the awareness about serum magnesium deficiency in type II DM patients. So, they can take preventive supplementation for hypomagnesaemia and thus to reduce the risk for the development of type II DM.

Methods

This cross sectional study was carried out in the Department of Physiology, Rangpur medical college, Rangpur, from July 2010 to June 2011. The protocol was approved by the ethical committee of Rangpur Medical College, Rangpur. Total number of 100 subjects of 40 to 60 years of age was included in this study. 50 patients of newly diagnosed type II diabetes mellitus were taken as experimental group (group B), 50 were age and sex matched apparently healthy non diabetic subjects were considered as control (group A). Patients with presence of hypertension (diastolic B.P>90 mm of Hg and systolic B.P>140 mm of Hg), nephropathy, neuropathy, retinopathy and history of any heart diseases were excluded from the study. Patients of diuretic users were also excluded. Controls were selected from the community by personal contact and the experimental patients were taken from the Out Patient Department in Rangpur Diabetic Association, Rangpur. After selection of the subjects, the purpose of the study was explained to each subject. When they agreed for participation, then an informed written consent was taken. Detailed family and medical history were taken. Thorough physical examinations were done and all information was recorded in a prefixed questionnaire. At the first day, all study procedures were maintained and advised the subjects to be overnight (8 -10 hours) in fasting state and instructed them to attend next day at 8.00 AM at the diabetic centre, Rangpur. Five ml of fasting venous blood from the ante-cubital vein and urine sample were collected from each subject. After that 75 gm glucose with 300 ml water was given to all the subjects. Again blood and urine samples were obtained at the end of 2 hours after ingestion of glucose. Serum magnesium was measured by photoelectric colorimetric calmagite method. Biochemical tests were done in the laboratory of the Department of Biochemistry of Rangpur Medical College, Rangpur. Data were expressed as Mean ± SD. For statistical analysis independent sample‘t’ test was

performed by using SPSS – 15.0 versions for windows.

Results

Patients characteristics are shown in the table I. Mean serum magnesium level was significantly lower ($p<0.001$) in experimental group B than that of control group A (Table II)

Table I: Sex and mean age, height, weight, BMI of the subjects in group A and B

Group	Age (Yr) Mean (L-H)	Sex	Height (cm) Mean (L-H)	Weight (Kg) Mean (L-H)	BMI (kg/m^2) Mean (L-H)
A	47.54 (38-65)	M-31 F-19	161.02 (147-173)	68.76 (46-74)	26.57 (22.74-30.55)
B	48.08 (40-70)	M-29 F-21	157.18 (140-173)	71.1 (63-83)	28.35 (24.80-32.33)

Group A: Control – Healthy non-diabetic subjects.
Group B: Experimental – Patients of type II diabetes mellitus.
L-Lower value
H-Higher value
M-Male
F-Female

Table II: Mean ± SD of serum magnesium level in group A and B

Group	Serum magnesium level(mmol/L)	Significance of difference (A vs B)
A (n=50)	8420 ± .09495	
B (n=50)	6320 ± .09355	P< 0.001 ***

*** Highly significant
n = Number of subjects.
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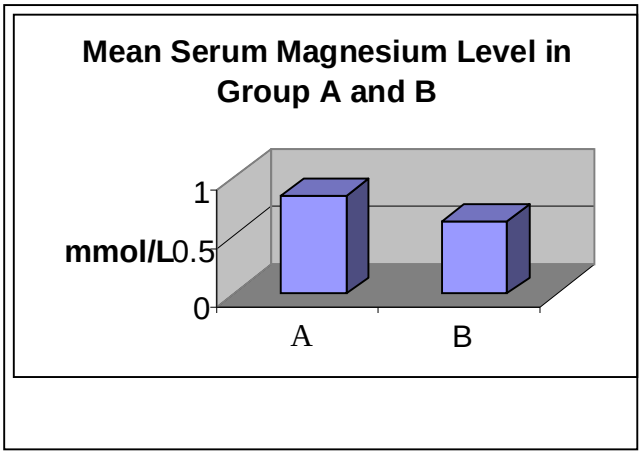

Fig. 1: Mean Serum Magnesium Level

Discussion

In the present study mean serum magnesium level in healthy control group was within normal range and also similar to those reported by the various investigators from different countries.[5-9] Mean serum magnesium level was significantly lower ($p < 0.001$) in type II diabetic patients than that of control. Similar findings were also reported by different researchers in different countries.[5-18] Various mechanisms have been proposed by different investigators for the decreased serum magnesium level in type II diabetes mellitus. Some suggested that decreased serum magnesium level in type II DM may be related to increased urinary excretion of magnesium due to osmotic diuresis.[10-15] It has been suggested that the osmotic action of glucosuria depress the net tubular reabsorption of magnesium.[15] Other investigators reported that decreased mean serum magnesium level in type II DM may be due to increased urinary loss, lower dietary intake and impaired absorption of magnesium.[5-8]

It is interesting that the induction of magnesium deficiency has been shown to reduce insulin sensitivity in individuals without diabetes, whereas magnesium supplementation during a 4-6 week period has been shown to improve glucose handling in elderly individuals without diabetes. Magnesium is reabsorbed maximally in the thick ascending limb of loop of Henle and distal convoluted tubules. Insulin has been implicated in enhancing magnesium reabsorption in these segments. Insulin resistance in diabetic state can promote magnesium excretion at these nephron segments. So, reduced serum magnesium level in Type II DM may be related to increase urinary loss of magnesium.[5]

Though various suggestions made by different investigators as mentioned above as possible causes of reduced serum magnesium level in type II DM but it is difficult to comment on exact mechanisms in this type of study as urinary magnesium level was not studied. So, in the present study it may be concluded that significantly lower serum magnesium level in type II diabetes mellitus may be related to increase urinary loss of magnesium.

Conclusion

In this study, serum magnesium level is reduced in type II diabetes mellitus as compared to healthy non diabetic control. Magnesium depletion reduces insulin sensitivity and may increase risk of secondary complications. Therefore, it may be prudent in clinical practice to periodically monitor serum magnesium concentration in type II diabetic patients. If serum magnesium is low, an intervention to increase dietary intakes of magnesium may be helpful to minimize diabetes related other complications.

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