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Original Article

Biochemistry Section

Serum Magnesium Level in Type 2 Diabetes Mellitus Patients with and without Complication and its Correlation with Poor Glycaemic Control

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ABSTRACT

Introduction: Diabetes Mellitus (DM) is the most challenging health problem in the world. Understanding the pathogenesis, maintaining sugar levels and preventing its long term complications are still the major goals of research in diabetic patients. Emerging scientific evidences has disclosed relationship between serum magnesium concentration in patients of Type 2 DM (T2DM), its complications and poor glycaemic control. Hypomagnesemia may be a factor implicated in pathogenesis of poor glycaemic control and diabetic complications.

Aim: The present study was undertaken to assess level of serum magnesium concentration in T2DM patients with and without complications and find out correlation between serum magnesium concentrations with glycaemic control.

Materials and Methods: A cross sectional study was conducted which included 150 cases divided into 3 groups. In group I, there were 50 normal healthy controls. In group II, 50 patients having DM type 2 without any complications and with good glycaemic control were included. In group III, 50 patients with DM type 2 with one or more chronic complications and having poor glycaemic control were selected. The significance of difference in serum magnesium concentration between all three groups was tested by One-way ANOVA test.

Results: Statistically significant decrease was observed in magnesium concentration (with p-value <0.001) in Group III cases compared to Group I and Group II. There was a statically significant negative correlation between serum magnesium concentration and Fasting Blood Glucose (FBS), Postprandial 2 Hour Blood Sugar (PP_2BS) and HbA1c levels.

Conclusion: Serum magnesium concentration was lower in patients with T2DM with complication (Among 56% in Group III compared to 26% in Groups II and 8% in Group I patients). Serum magnesium level and poor glycaemic control show a negative correlation. This indicates a key role of magnesium in metabolic derangement in diabetic patients, its glycaemic control and its complications.

Keywords: Glycated Haemoglobin, Hypomagnesemia, Metabolic derangement

INTRODUCTION

The DM is one of the most common noncommunicable diseases in world including India. Complications from diabetes, such as coronary heart diseases, peripheral vascular diseases, brain stroke, diabetic neuropathy, amputations, diabetic nephropathy, diabetic retinopathy, cataract etc result in increasing morbidity and mortality. Due to its complications, patients often get admitted to the hospital. It is amongst the toughest health issue to deal with which needs a multidisciplinary approach to detect and treat its potential complications [1].

DM is defined as a group of common metabolic disorders that share common phenotype of hyperglycaemia. Hyperglycaemia not only defines the disease but is the cause of its many symptoms and long term complications. The factors causing hyperglycaemia include reduced insulin secretion, insulin resistance, decreased glucose utilisation and increased glucose production. Insulin resistance plays a key role among patients of T2DM. The metabolic disturbances associated with DM causes secondary pathological changes leading to microvascular (retinopathy, nephropathy and neuropathy) and macrovascular (coronary heart disease, peripheral arterial disease and cerebrovascular disease) complications [2].

The development of complications is linked to the accumulation of glycation products in tissue proteins which is directly related to glycaemic control. Thus it is very important to maintain glycaemic control in patients with DM [3]. Optimal monitoring of glycaemic control involves measurement of plasma glucose (fasting and post prandial) and HbA1c levels. The patient's glucose measurements provide short-term glycaemic control, whereas the HbA1c reflects long term glycaemic control [2]. HbA1c is formed by glycosylation of haemoglobin by nonenzymatic process. High concentration of glucose can increase process of glycation and increase concentration of HbA1c level. American Diabetes Association recommends twice per year measurements for patients who are meeting treatment goals and quarterly measurements for those whose therapy has changed or who are not meeting treatment goals. Less than 7% should be HbA1c for DM patients which is considered as good glycaemic control [4].

Magnesium is an essential intracellular cation which has a fundamental role in many physiological functions in body. It is an enzyme activator for neuromuscular excitability, cell permeability and serves as regulator for ion channels and mitochondrial functions which is important for cell proliferation and apoptosis [5]. Magnesium is involved in many kinases reactions that need ATP and also in protein synthesis. Magnesium also has fundamental role in carbohydrate metabolism. It is a co factor in glucose transport mechanism across the cell membrane and for various enzymes in carbohydrate metabolism [6]. It plays an important role in the regulation of blood glucose levels as it is essential for insulin secretion, binding to its receptor and activity [7]. Also, magnesium has antioxidant properties by scavenging the oxygen radicals through regulating the rate of spontaneous dismutation of super oxide anions [8].

Magnesium deficiency is common in diabetic patients, 25-39% of diabetics have low concentrations of serum magnesium [9-11]. Magnesium deficiency also has a negative impact on glucose regulation and insulin sensitivity in T2DM patients [12,13]. So affects glycaemic control in diabetes and evolution of complications such as retinopathy, arterial atherosclerosis and nephropathy [14,15]. Many large studies have reported a reduction in T2DM risk in

patients with increased magnesium intake in patients [10,16-18]. In humans, several experimental metabolic studies have suggested that magnesium supplementation has beneficial effects on glucose metabolism, insulin action and insulin sensitivity [19].

After going through all the research works in similar segments [9-11,20-25], we concluded that there are studies showing significant difference in magnesium level in diabetic patients [23,26], but it was not found in our studies. In this study it gives better analysis for serum magnesium level in diabetic patients in case of poor glycaemic control, which was supported by few studies only [12-14].

So to avoid misconception and to understand the spectrum of work done we have conducted this study.

Only few studies [14,26-29] have shown magnesium level and its correlation with FBA, PP_2BS and HbA1c, in which our study adds scope for future contribution and give a strong base for justification to fill such gap and help in providing potential contribution in improving knowledge and practice in medicine.

The present study was undertaken with an aim to estimate serum magnesium level in T2DM with and without complications and compare it with non diabetic healthy control subjects. Also, to find out correlation between serum magnesium concentrations in patients with poor glycaemic control (FBS, PP_2BS and HbA1c).

MATERIALS AND METHODS

This study was a hospital based cross sectional study conducted at civil hospital, Ahmedabad between June 2015 to September 2015. We studied and analysed already available data, so only permission from medical superintendent was required for this as per our institute policy for data analysis. We took permission from medical superintendent for this prior to starting study in on 12th May 2015. No intervention was done, so no ethical permission was required.

The study involved those patients coming to diabetic OPD for routine check up. All parameters were analysed from already collected sample for their routine lab tests. Neither any additional test was done nor any additional sample was collected. The participants were informed about the aim of the study, informed consent was taken for participation and for blood collection. Emphasis was given that participation in study was absolutely voluntary. A proper history taking based on age,sex, duration and type of DM, type of treatment for DM, any complication and its related history), other diseases like hypertension, history of smoking if any. Body Mass Index (BMI) was calculated by measuring weight (kg) and height (sq mt) by formula Weight in kg/ Height in sq meter.

Empirically, it is considered enough sample size, if number of subjects in each group is more than 30. During our study period of 4 months, we got approximately 50 patients in each group which fulfilled inclusion criteria as mentioned. The study comprised 150 subjects which were divided into 3 groups according to following inclusion criteria:

Group I: Control group (n=50) which consisted of age and sex matched healthy subjects and free from any illness which could affect parameters under study. They were taken from general population.

Group II: DM type 2 patients without any complication with good glycaemic control (n=50). (Already known case of DM type 2 on treatment was taken for study. They were already diagnosed based on FBS >126 mg% & PP_2BS >200 mg% in venus plasma)

Group III: DM type 2 patients with one or more chronic vascular complications (e.g., diabetic nephropathy, diabetic retinopathy, heart disease, diabetic neuropathy) and had poor glycaemic control (n=50).

The cut-off value to differentiate between good and poor glycaemic control was based on HbA1c level. The HbA1c level more than 7% was considered as poor glycaemic control.

The patients with type 1 DM, critically ill patients (patients with significant hepatic and renal disease, haematological malignancy, chronic kidney disease, acute cerebrovascular accidents, acute myocardial infarction etc.,), gestational DM, patients receiving magnesium supplements, diuretics, patients with history of alcohol abuse and patients refusing to give informed consent for the study etc., were excluded from this study.

About 5 mL of venous blood was drawn from each volunteer using a disposable vaccutainer system in fasting condition (in plain, EDTA and fluoride) by trained phlebotomist at OPD blood collection center. Postprandial 2 hour sample (in fluoride) was collected under proper aseptic condition.

FBS & PP_2BS estimated by End Point Glucose Oxidase and Peroxidase (GOD POD) method (Mfg: Spinreact).

HbA1c concentration was measured by Immuno turbidimetric method (Mfg: Spinreact on HbA1c analyser Mfg by Tulip).

Serum Magnesium concentration was done by Calmagite– EGTA colorimetric method (Mfg: Spinract).

Other parameters like cholesterol (by CHOD POD method, Mfg by crest biosystem), triglyceride (GPO PAP method, Mfg crest biosystem), Creatinine (Jaffe's, Mfg by Transgeniks), ALT (UV kinetic, Mfg by spinreact) were assessed. All biochemical investigations were performed on fully automatic biochemistry analyser (Model: XL 640 Mfg by Transasia Bio-Medicals Ltd.,).

STATISTICAL ANALYSIS

The data for this study was statistically analysed by using GraphPad InStat version 3.1 software & Microsoft excel. The significance of difference in serum magnesium concentration between all three groups was tested by One-way ANOVA test. The correlation between serum magnesium concentration (mg/dl) and HbA1c (%), FBS (mg/dl) and PP_2BS (mg/dl) in patients having poor glycaemic control was tested by Pearson's correlation. The p-value less that 0.05 was considered as significant. The cut-off value to differentiate between normomagnesemia and hypomagnesemia was 1.6 mg/dl [30].

Prevalence was calculated as percentage of total patient in particular group.

RESULTS

Mean age of patient in Group 1, Group 2 and Group 3 are 54±5, 57±6 and 58±5 years, respectively. Among Group 2, 26 were males and 24 are females. Among Group 3, 30 were males and 20 females. Among Group 1, 27 were males and 23 females. Comparison of other variables between study groups like hypertension, smoking, average duration, total cholesterol level, triglyceride level, creatinine level etc., given in [Table/Fig-1].

	Group I	Group II	Group III
Number	50	50	50
Average age (years)	54	57	58
Average duration of DM (year)	-	6.12	11.23
Prevalence of Hypertension (%)	-	56	84
Prevalence of smoking (%)	-	26	48
Average BMI (kg/m^2)	23.56±5.13	26.63±6.34	26.77±8.12
Mean Serum total cholesterol (mg/dL)	165	178	215
Mean Serum triglycerides (mg/dL)	105	134	154
Mean Serum Creatinine (mg/dL)	0.56	0.78	1.6
Mean Serum ALT (U/L)	23	32	43

[Table/Fig-1]: Comparison of other variables between study groups.

As shown in [Table/Fig-2] mean serum magnesium concentration in Group-I, Group II and Group III was 1.92, 1.84 and 1.43 mg/dl, respectively. The mean serum magnesium concentration between

Group 1 and Group 3 as well as between Group 2 and Group 3 was statistically highly significant (with p-value <0.001). But the difference between Group 1 and Group 2 was not significant (p-value >0.5) [Table/Fig-3].

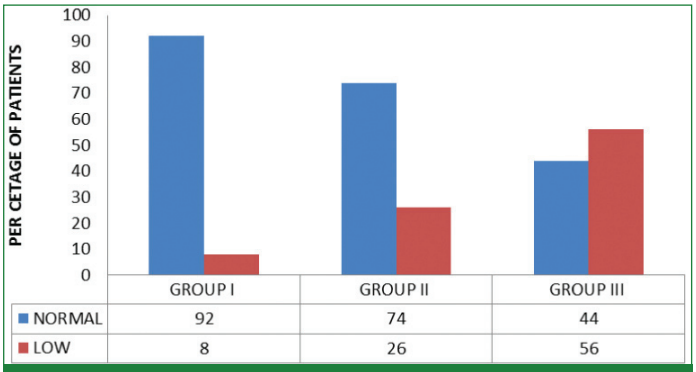
	Group I	Group II	Group III
Number	50	50	50
Mean	1.92	1.84	1.43
Standard Deviation	0.25	0.30	0.38
Standard error of mean	0.035	0.043	0.054

[Table/Fig-2]: Serum magnesium concentration (mg/dl) in Group I, II & III.

	Mean difference	q value	p-value
Group I vs Group II	0.08620	1.919	>0.05
Group I vs Group III	0.4894	10.894	<0.001
Group II vs Group III	0.4032	8.975	<0.001

[Table/Fig-3]: Results of One-way ANOVA test.

As shown in [Table/Fig-4], the prevalence of hypomagnesemia among Group II and Group III cases was 26% and 56%, respectively. The Group I patients had 8% cases with decreased value of serum magnesium concentration level.

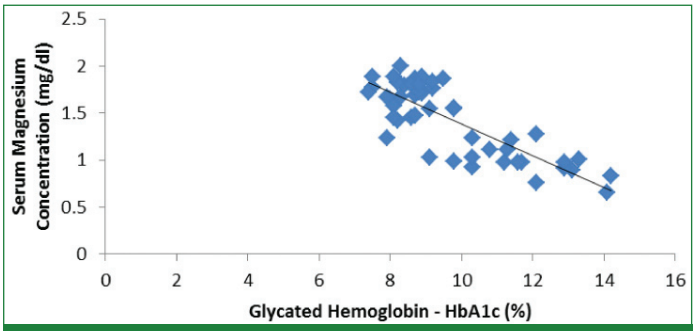


[Table/Fig-4]: Bar diagram showing prevalence of low magnesium level in study groups (%)..

[Table/Fig-5-8] shows negative correlation between serum magnesium concentration with HbA1c, FBS and PP_2BS in cases with poor glycaemic control and these values were statistically significant (p<0.0001).

		HbA1c (%)	FBS (mg/dL)	PP_2BS (mg/dL)
Serum magnesium concentration (mg/dl)	Sample size	50	50	50
	Correlation coefficient r	-0.8185	-0.6373	-0.6203
	Significance (p-value)	<0.0001	<0.0001	<0.0001

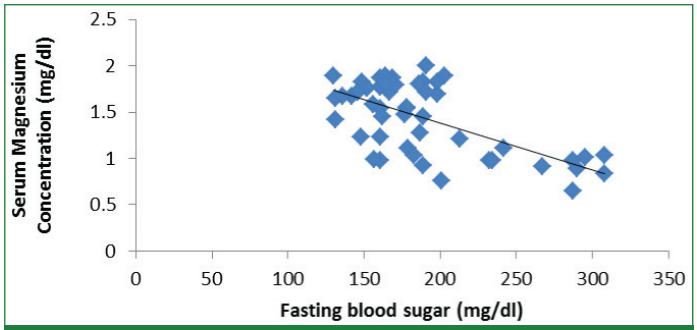
[Table/Fig-5]: Correlation between serum magnesium concentration (mg/dl) and HbA1c (%), FBS (mg/dl) and PP_2BS (mg/dl) in Group III patients.



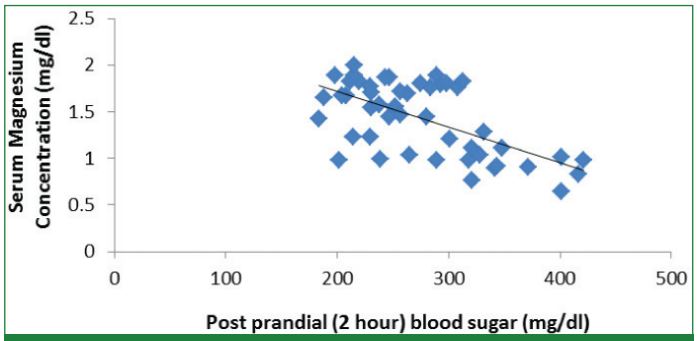
[Table/Fig-6]: Scatter dot diagram shows correlation between serum magnesium concentration (mg/dL) and HbA1c %.

DISCUSSION

Present study was undertaken to assess level of serum magnesium concentration in T2DM patients with and without complications.



[Table/Fig-7]: Scatter dot diagram shows correlation between serum magnesium concentration (mg/dL) and FBS (mg %).



[Table/Fig-8]: Scatter dot diagram shows correlation between serum magnesium concentration (mg/dL) and PP_2BS (mg %).

The cross sectional study consisted of 150 patients out of them 50 patients having T2DM without any complication and with good glycaemic control (Group II), 50 patients with T2DM with one or more vascular complications and poor glycaemic control (Group III) and 50 normal healthy controls (Group-I) were selected.

In our study, we found mean serum magnesium concentration in Group III, Group II and Group I were 1.43, 1.84, 1.92 mg/dL, respectively. Statistically, significant decrease in magnesium concentration in Group III cases (p<0.0001) was observed compared to Group I and Group II. We did not observe decrease in magnesium concentration in Group II cases that were in good glycaemic control compared to healthy control. The prevalence of decreased serum magnesium concentration in Group III and Group II were 56% and 26%, respectively whereas in Group I was only 8%. Also, we found significant negative correlation between serum magnesium concentration and FBS, PP_2BS and HbA1c in cases having poor glycaemic control (Group III). This relationship was statistically significant (p-value<0.0001).

As per this study, the plasma magnesium concentration was from 25-39%, which is in accordance with previous studies [9-11].

Walti MK et al., reported that hypomagnesemia in type 2 diabetic mellitus patient is seen in 37.6% cases versus 10.9% cases in nondiabetic controls [20]. Hypomagnesemia in diabetics can be due to osmotic losses by kidney along with glycosuria, impaired intestinal absorption and redistribution of magnesium from plasma to red blood cells due to effect of insulin [20].

Serum levels of magnesium have been found by several investigators to correlate inversely with FBS, PP_2BS and HbA1c% [14,16,27,28].

Hypomagnesemia is reported to be both a cause and result of poor glycaemic control. Various studies have shown that magnesium deficiency enhances insulin resistance [31].

Nadler JL et al., concluded that insulin sensitivity reduces after induction of magnesium deficiency [12]. Conversely, Mc Nair P et al., indicated that hyperglycaemia and osmotic diuresis due to glycosuria may lead to increased urinary magnesium excretion and hypomagnesemia in diabetics with poor glycaemic control. Insulin treatment and control of hyperglycaemia has been shown

to correct renal magnesium loss in diabetics [32]. Abdul Wahid et al., found 34% prevalence. Diabetics with hypomagnesemia had poor glycaemic control. Hypomagnesemia was significantly associated with diabetic retinopathy [33].

Arpaci D et al., have shown low magnesium concentration in patients of DM with complication. Also, weak negative correlation was found between serum magnesium concentration and HbA1c levels and between serum magnesium level and urine proteinuria. So magnesium concentration affects glycaemic regulation and nephropathy both [34].

Also, poor glycaemic control in type 2 diabetic patients is a known risk factor for magnesium deficiency. A statistical significant negative correlation between magnesium level and fasting blood glucose, HbA1c is found [16,29]. Some studies have shown reduced magnesium level could have a role in pathogenesis of microvascular complications of DM [35].

Low magnesium levels result in defective tyrosine kinase activity at the insulin receptor level which impairs insulin action and deteriorates insulin resistance in type 2 diabetic patients. It has been associated with increased levels of TNF-α which lead to post receptor insulin resistance [21,36]. Insulin activates sodium magnesium exchange at membrane and regulates the intracellular magnesium levels which explains low cellular magnesium levels due to insulin resistance [22]. Hypomagnesaemia leads to increase in free oxygen radical formation and decrease in the antioxidant properties contributing to the oxidative stress in T2DM [8].

Parlapally RP et al., showed decreased serum magnesium levels in diabetics compared to controls. Also they had shown a low level of serum magnesium in uncontrolled diabetics [23]. El-said NH et al., shown hypomagnesaemia is closely linked to T2DM and it is strongly correlated to glycaemic control [26]. There results were in contrast to our result where we find no significant difference between diabetic person with good glycaemic control and healthy group. Our result is in support of results given by Naila M et al., and Zargar AH et al. They did not report any significant difference in serum magnesium level in type 2 diabetic patients when compared to healthy controls [24].

We find strong negative correlation between magnesium level and glycaemic control. This was in agreement with Mishra S et al., and Karim R et al., who found significant negative correlation between serum magnesium levels and fasting blood sugar in T2DM [37,38]. In contrast Walti MK et al., and other studies did not find such correlation between serum magnesium levels and HbA1c [20]. The association between low serum magnesium levels and increased HbA1c levels suggests important role of magnesium in progress and development of complications in diabetic patients especially in patient with uncontrolled diabetes.

Previous studies reported improvement in insulin sensitivity and metabolic control in type 2 diabetic patients who received magnesium supplementation [17,18]. Contradictory results about the correlation between serum magnesium and glycaemic control and the effect of oral magnesium supplementation on metabolic parameters of type 2 diabetic patients may be related to different study designs and different populations which need further large scale studies [25].

As there were lots of reports [14,26-29] linking low magnesium level with poor glycaemic control and diabetic complications, attention and understanding of this matter among clinicians is important which may improve patient outcome. Thus, present study has shown the functional role of magnesium in pathogenesis of DM and introduces a relatively new concept on the implication of magnesium deficiency in diabetic complications & its association with poor glycaemic control.

Limitation(s)

Due to small sample size of this study and no follow-up done, being a cross-sectional study, further interventional study is required to analyse therapeutic role of magnesium supplementation in patient with T2DM to improve glycaemic control and prevention of complications related to it.

CONCLUSION(S)

Serum magnesium concentration was lower in patients with T2DM with complication. A negative correlation was seen with serum magnesium and poor glycaemic control. This indicates a key role in magnesium in metabolic derangement in subjects with diabetes. In future studies should be done to evaluaye the role of serum magnesium in modifying effect of insulin and glycaemic control in DM.

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