e-ISSN: 0975-5160, p-ISSN: 2820-2651

Available online on www.ijtpr.com

International Journal of Toxicological and Pharmacological Research 2023; 13(6); 270-279

Original Research Article

Serum Magnesium Levels in type II Diabetes Mellitus Patients with Special Reference to Long Term Complications of Diabetes (Study of 100 Patients)

Pooja Jariwala[1], Darshak Makawana[1], Patel Mittalben Maheshbhai[2], Madhvi Kareliya[3*]

[1]MD, Medicine, Assistant Professor, P.D.U Government Medical College, Rajkot, Gujarat, India

[2]Senior Resident, GMERS Medical College, Vadnagar, Gujarat, India

[3]MD, Pharmacology, Assistant Professor, P.D.U Government medical college, Rajkot, Gujarat, India

Received: 22-04-2023 / Revised: 25-05-2023 / Accepted: 27-06-2023

Corresponding author: Dr. Madhvi Kareliya

Conflict of interest: Nil

Abstract:

Background and Objectives: Hypomagnesemia can be a consequence of hyperglycemia (as seen in increased urinary magnesium excretion along with glycosuria) and a cause of insulin resistance. The association between diabetes mellitus and hypomagnesaemia is compelling for its wide-ranging impact on diabetic control, its macro and micro-vascular complications, and ultimately on the therapy.

Objectives: To estimate prevalence of hypomagnesaemia in patients with type 2 DM and to correlate the serum magnesium concentrations with micro and macrovascular complications of diabetes – retinopathy, nephropathy, neuropathy and ischemic heart disease.

Material and Method: An observational study carried out for one year in 100 participants diagnosed with Type 2 diabetes mellitus in the age group of 30–80 years in medicine department, were included in the study. Serum Magnesium levels of all diabetic patients were investigated and compared with long term diabetes complications like diabetic retinopathy, nephropathy and neuropathy. Serum Magnesium levels were also compared with comorbid conditions like hypertension, Lipid profile and HbA1C for poor glycaemic control. Statistical analysis were carried out for correlation and test of significance.

Result: Among 100 participants, males were 47% and females were 53 % with mean age of 55.04 ± 11.2 years. The average duration of diabetes in study population was 5.5 years and most of the patients were received oral hypoglycemic drugs for their diabetes treatment. 29% of participants had hypomagnesemia (serum Mg≤ 1.7mEq/L). Among the patients having hypoglycemia, 23.3% of participants were having hypertension, 31% were having retinopathy, 37.9% were having nephropathy and 69% were having neuropathy. In our study, retinopathy and neuropathy were significantly correlated with hypoglycaemia (p value <0.05). Among lipid profile, Serum triglyceride and serum HDL cholesterol were significantly and inversely correlated with hypomagnesemia. Poor glycemic control in patients with hypomagnesemia were also established due to significant correlation between High HbA1C level and hypomagnesemia.

Conclusion: Hypomagnesemia was significantly associated with diabetic retinopathy and neuropathy, poor glycemic control and lipid profile abnormalities. Benefits of Mg supplementation on metabolic profile in diabetic subjects have been found in most, but not all

clinical studies, and larger prospective studies are needed to support the potential role of dietary Mg supplementation as a possible public health strategy in diabetes risk.

Keywords: Diabetes Mellitus, Hypomagnesemia, Diabetic Retinopathy, Diabetic Neuropathy.

Introduction

Magnesium is a cofactor in the glucose-transporting mechanism of the cell membrane and various enzymes in carbohydrate oxidation. It is also involved at multiple levels in insulin secretion, binding and activity. The almost universal involvement of magnesium in a wide variety of cellular processes critical to glucose metabolism, insulin action and cardiovascular functions. [1]

Hypomagnesemia has been related as a cause of insulin resistance, also being a consequence of hyperglycemia, and when it is chronic leads to the installation of macro and microvascular complications of diabetes, worsening the deficiency of Magnesium.[2]

Intracellular magnesium concentration has also been shown to be effective in modulating insulin action (mainly oxidative glucose metabolism), offset calcium-related excitation-contraction coupling, and decrease smooth cell responsiveness to depolarizing stimuli. Magnesium also helps correct abnormal lipoprotein patterns. Improved insulin sensitivity from magnesium replacement can markedly reduce triglyceride levels.[3] There were several studies were performed to evaluate the role magnesium in insulin sensitivity, glucose regulation and protection from type 2 diabetes. In one such a large Japanese study([4] (the Hisayama Study), researchers found that magnesium intake was a significant protective factor against type 2 diabetes in the general Japanese population, especially among those "with insulin resistance, low-grade inflammation and a drinking habit." And in the Framingham Offspring cohort (2006), it was concluded that higher magnesium intake improved insulin sensitivity and reduced type 2 diabetes risk.[5]

However, limited attention has been drawn to the impact of magnesium deficiency on late diabetic complications, including cardiovascular disorders and osteoporosis. So, the present study was undertaken with an aim to estimate prevalence of hypomagnesaemia in patients with type 2 DM and to correlate the serum magnesium concentrations with micro and macrovascular complications of diabetes – retinopathy, nephropathy, neuropathy and ischemic heart disease.

Material & Method

This was an observational study carried out in 100 participants in medicine department at tertiary care teaching hospital with duration of one year (December 2013 to December 2014). Patients diagnosed with Type 2 diabetes mellitus patients in the age group of 30–80 years, including patients with nephropathy, neuropathy and retinopathy were included in the study. Patients having type 1 diabetes mellitus. Patients taking lipid lowering agents, magnesium containing antacid, chronic diuretics or the Patients having metabolic/other endocrine disorders, renal failure, ischemic heart disease, acute and chronic sepsis and patients having chronic diarrhea were excluded from the study. All these conditions can alter the serum magnesium level in the body.

After taking written informed consent all patients were be subjected to detailed history taking – including duration of diabetes, treatment mode, symptoms suggestive of diabetic neuropathy, associated diseases such as hypertension

and ischemic heart disease was obtained followed by physical and neurological examination and ECG. Retinopathy was assessed by direct ophthalmoscopy. Blood samples were collected for measurement of fasting blood glucose and serum magnesium. Postprandial blood sugar was measured two hours after a standard meal.

Blood urea, serum creatinine and 24-hour urinary albumin were estimated. Serum magnesium was estimated by Calmagite dye method. HbA1C estimation was carried out by a modified calorimetric method.

All data were analyzed using appropriate statistical tests by using MedCalc version 14.10.20.

A p value <0.05 was considered statistically significant with 95% confidence interval. Serum Magnesium levels of diabetic patients were compared with long term complications of diabetes.

Result:

Among 100 participants there were 47 men and 53 women. Mean age was 55.04 years. Highest number of patients were seen between age group of 41-50 and 61-70 years. Age group wise distribution of male and female was given in figure no 1.

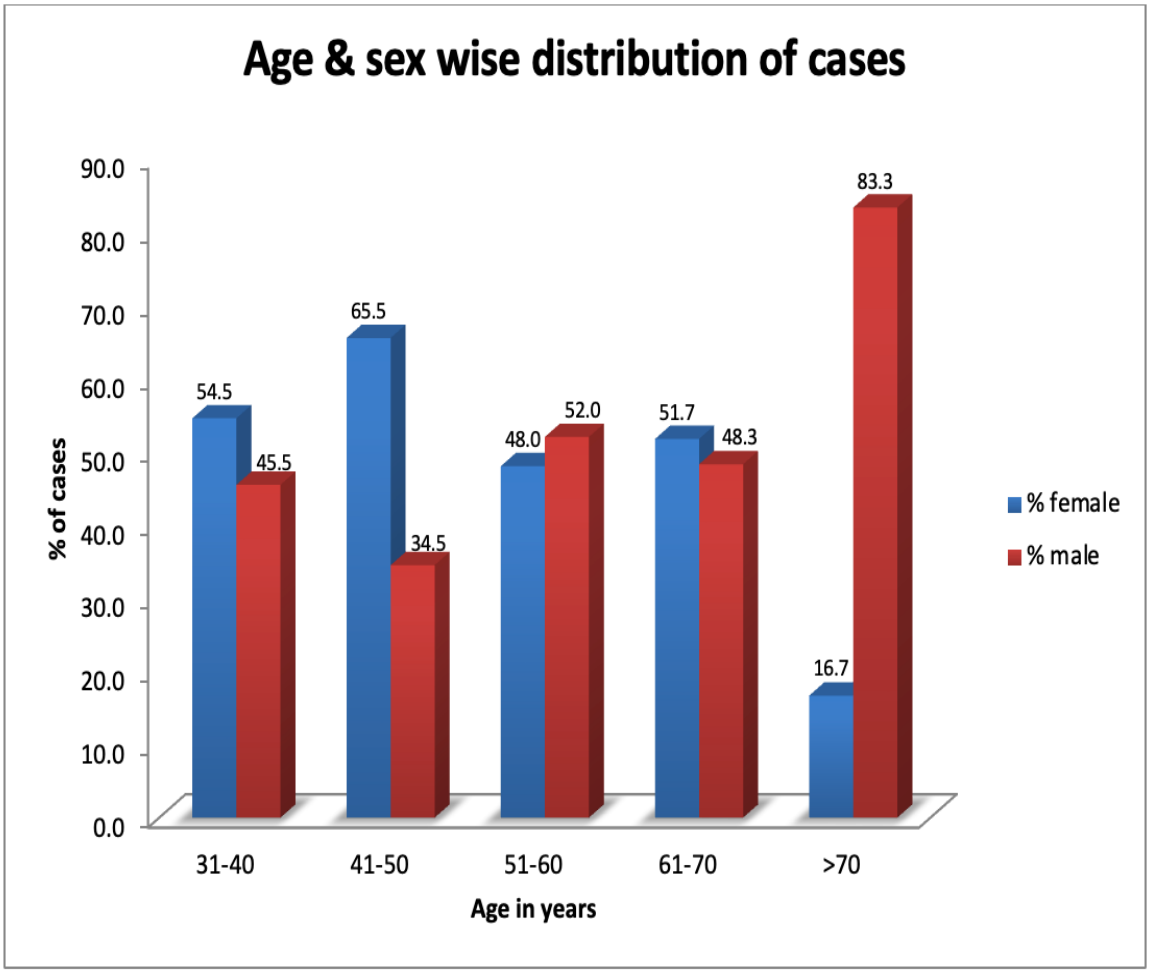

Figure 1: Age group and gender wise Distribution of Patients (N=100)

Baseline characteristics were given in Table no 1. The average duration of diabetes in study population was 5.5 years. The mode of diabetic treatment and presence of comorbid condition were shown in table no 1.

Table 1: Baseline data of study participants (N=100)

Baseline Characteristics	No. of patients (N=100)
Age (Years)	55.04 (Mean)
Duration of diabetes (years)	5.5yrs (Mean)
1. Mode of treatment used in diabetic patients	
• Insulin	4
• Oral hypoglycemics	88
• Insulin and oral hypoglycemics	1
• Diet only	4
2. Comorbidities	
• Hypertension	39
• Ischemic heart disease	NONE
• Non-proliferative diabetic retinopathy (NPDR)	15
• Proliferative diabetic retinopathy (PDR)	3
• Diabetic neuropathy	50
• Diabetic nephropathy - Microalbuminuria	27
• Diabetic nephropathy - Macroalbuminuria	4
• Poor glycemic control (HbA1c > 7.5)	80

Patients were divided into those with Mg≤ 1.7mEq/L as hypomagnesaemia, and those with Mg>1.7 mEq/L as normomagnesemia. Among total patient, 29(29%) patients had hypomagnesaemia and 71 (71%) patients had normomagnesemia. None had hypermagnesemia defined as S. Mg > 3.5 mEq/L. Hence the prevalence of hypomagnesemia among type 2 diabetics was 29%.

In this study, duration of diabetes and patients having hypomagnesaemia were correlated with each other. This correlation was found significant at P value 0.016. So , the duration of diabetes significantly longer with low serum magnesium concentration. (Figure 2)

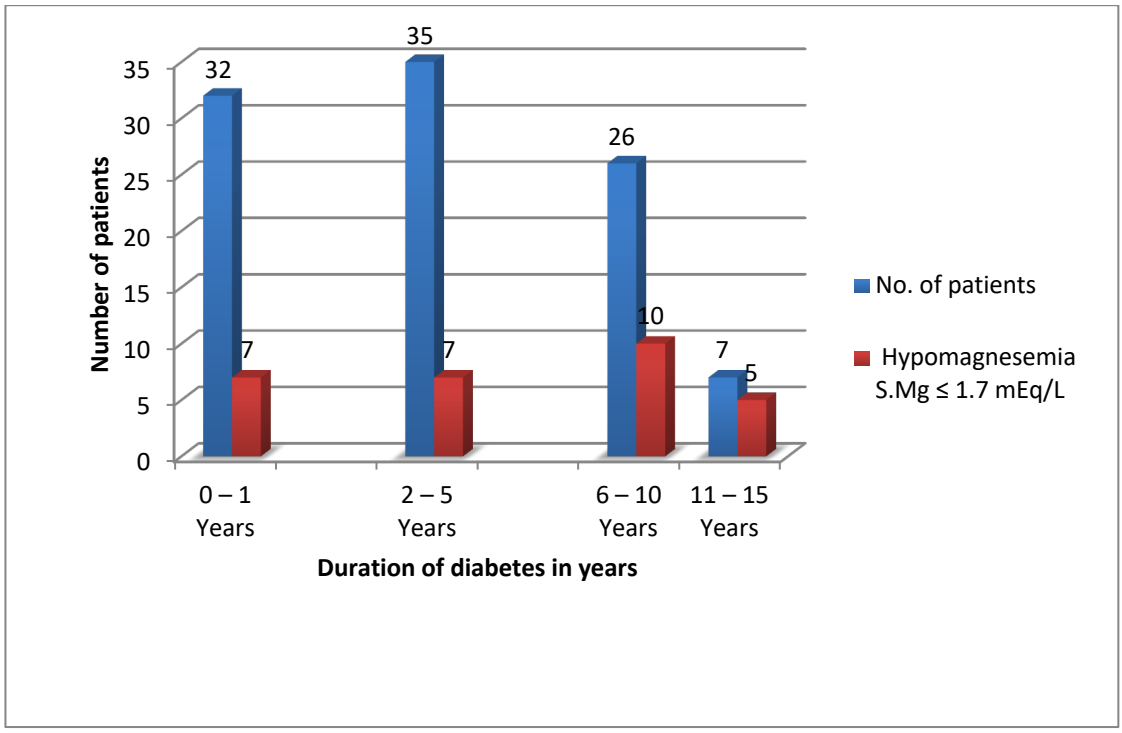

Figure 2: Prevalence of hypomagnesemia and duration of diabetes

In this study, Mode of diabetic treatment and hypomagnesemia were compared with each other and it was observed that higher prevalence of hypomagnesemia was seen in patients treated with insulin and those who were on diet only. However, the difference was statistically not significant (p value 0.913) (Table 2)

Table 2: Prevalence of hypomagnesemia and mode of diabetic treatment

Mode of diabetic Treatment	No. of patients	No. of patients with hypomagnesemia (Mg≤ 1.7 mEq/L)	frequency (%)
Insulin	4	3	75.00
Oral hypoglycemic drugs	88	23	26.13
Insulin + Oral hypoglycemic drugs	1	0	0
Diet only	4	3	75.00

Analysis of correlation between serum Mg level and diabetes control was assessed. For diabetic control HbA1C, FBS (Fasting Blood Sugar) and PP2BS (Post Prandial Blood Sugar) are investigated in all the patients. There was a significant inverse correlation was observed with hypomagnesemia and HbA1C levels with P vale of 0.038 which was statistically significant. (Table 3)

Table 3: Correlation of hypomagnesemia with diabetes control HbA1C, FBS & PP2BS

Variables	S Mg ≤ 1.7 mEq/L		S Mg >1.7 mEq/L		Total		P value
	Mean	**±Sd**	**Mean**	**±Sd**	**Mean**	**±Sd**	
HbA1C (%)	10.4	2.2	7.5	2.5	9.8	2.4	0.038*
FBS mg/dL	202.3	70.1	216.6	81.7	212.5	78.4	0.35
PPBS mg/dL	285.4	67.6	308.7	93.4	302.0	87.0	0.17

In this study, Hypertension, Retinopathy and Nephropathy like comorbid conditions is also assessed with serum level of Magnesium. It was found that Retinopathy and Neuropathy was significantly correlated with hypomagnesemia. (Table no 4)

Table 4: Correlation of comorbid conditions with Serum Mg level

Comorbid condition		No. of patients with Serum Mg level ≤ 1.7 mEq/L	No. of patients with Serum Mg level >1.7 mEq/L	P value
Hypertension	With without	7 (23.3%) 23 (76.7%)	33 (45.7%) 37 (54.3%)	0.060
Retinopathy	With Without	9 (31.0%) 20 (69.0%)	9 (12.7%) 62 (87.3%)	0.031*
Nephropathy	With without	11 (37.9%) 18 (62.1%)	20 (28.2%) 51 (71.8%)	0.34
Neuropathy	With without	20 (69.0%) 9 (31.0%)	30 (42.3%) 41 (57.7%)	0.0002*
*P value ≤ 0.05 statistically significant				

ECG changes is also assessed with serum magnesium level. Though none of them had 2D Echo suggestive of Ischemic Heart Disease (IHD). Here, no statistical difference was noted between hypomagnesemia and patients with ECG changes. (P value 0.26) Among 29 patients having hypomagnesemia, none has having ECG changes while 3 patients out of 79

patients (normomagnesemia) having ECG changes.

Among the lipid profile abnormality, a significant inverse correlation was found between hypomagnesemia and Serum triglycerides and with Serum HDL levels. In case of hypomagnesemia, Serum triglycerides and Serum HDL level was increased. (table 5)

Table no. 5: Correlation of hypomagnesemia with lipid profile (serum cholesterol, serum triglycerides, serum HDL, serum LDL and serum VLDL)

Lipid Profile	Serum Mg ≤ 1.7 mEq/L		Serum Mg >1.7 mEq/L		Total		P value
	Mean	± Sd	Mean	± Sd	Mean	± Sd	
S. Cholesterol mg/dL	187.5	55.2	190.2	45.5	189.4	48.2	**0.79**
S. Triglycerides mg/dL	259.6	100.1	219.2	94.8	230.9	97.6	**0.059***
S.HDL mg/dL	34.9	10.5	38.6	6.3	37.5	7.9	**0.055***
S.VLDL mg/dL	39.5	19.8	36.7	19.5	37.5	19.5	**0.51**
LDL mg/dL	118.2	127.1	104.2	34.9	108.3	74.3	**0.47**

Discussion

There are several reports suggesting high prevalence of low plasma magnesium concentrations among subjects with diabetes and possible association of hypomagnesemia (≤1.7mEq/L) with long term complications of diabetes, prompted this study.

In the present study, among 100 patients, serum concentration of magnesium was found low in 29 patients (29%). Which is similar to the several studies which reported prevalence of low plasma magnesium status in type-2 diabetics ranged from 25 to 39%. [2]

A meta-analysis of 7 of these studies by Larsson SC et al.[6], which included 286,668 patients and 10,912 cases of diabetes over 6 to 17 years of follow-up, found that a 100 mg/day increase in total magnesium intake decreased the risk of diabetes by a statistically significant 15%. Another meta-analysis of 8 prospective coho rt studies, by Schulze MB et al.[7] that followed 271,869 men and women over 4 to 18 years found a significant inverse association between magnesium intake from food and risk of type 2 diabetes; the relative risk reduction was 23% when the highest to lowest intakes were compared.

Several studies have reported increased urinary magnesium excretion in type 1 and type 2 diabetes. Recently a specific tubular defect in magnesium reabsorption in thick ascending loop of Henle is postulated. This defect results in reduction in tubular reabsorption of magnesium and consequently hypomagnesemia. The reason for this tubular defect in diabetics in unclear. Insulin treatment has been shown to correct renal magnesium loss in diabetics. Increased urinary magnesium excretion due to hyperglycemia and osmotic diuresis may contribute to hypomagnesemia in diabetes. [8]

In this study, there was no correlation between urine Mg concentration and glycosylated hemoglobin or fasting plasma glucose level. The data suggest that intracellular Mg depletion without significant hypomagnesemia is related to increased urinary Mg loss in patients with diabetes. The urinary Mg loss is not correlated with the degree of metabolic control.[1,8]

Our results have not shown any significant associations between sex, age but duration of diabetes had significant correlation with serum magnesium levels.

Significant differences, in serum magnesium concentrations have been reported between the insulin treated and

non-insulin treated diabetics. Yajnik et al.[9] reported that insulin treated diabetics have significantly lower serum magnesium levels compared to non-insulin treated ones. In the present study prevalence of hypomagnesemia in insulin treated diabetics (75%) was higher than in noninsulin treated (50%). However, the difference was statistically significant. While Walti MK et al.[10] have reported that diabetes treatment (insulin or oral hypoglycaemic drug) did not significantly predict hypomagnesemia.

In a the study by Alzaida et al.[11] have found that cellular uptake of magnesium is normally stimulated by insulin. So, insulin treatment may enhance cellular magnesium uptake and result in increased prevalence of hypomagnesemia.

In the present study no significant correlation was found between incidence of hypertension and hypomagnesemia. Magnesium deficiency may contribute to high blood pressure, but the most common underlying cause is typically related to insulin resistance. Research published in 1998 in the journal Diabetes reported that nearly two-thirds of the test subjects who were insulin resistant also had high blood pressure.[12]

In the present study, no correlation was found between incidence of ECG changes and hypomagnesemia. However, Ischemic heart disease was excluded from the study. Several observational studies have associated lower serum levels of serum magnesium with higher risk of coronary artery disease. Liao F et al[13], as part of Atherosclerosis risk in communities' study, over 4 to 7 years of follow-up study (N=319) suggest that low magnesium concentration may contribute to the pathogenesis of coronary atherosclerosis or acute thrombosis. However, a low serum magnesium predisposes to coronary artery diseases is not known. Abraham et al. 1987[14], proposed the role of s. magnesium in treatment of fatal arrythmias in acute MI, showing its deficiency predisposing in pathogenesis of MI.

Magnesium deficiency has been found to be associated with diabetic microvascular complications. In the present study, significantly higher prevalence of hypomagnesemia was observed in diabetics with microvascular complications.

Hypomagnesemia has been reported in patients with diabetic retinopathy[15]. Lower magnesium levels predicted a greater risk of severe diabetic retinopathy. Our observation was revealed statistically significant difference is prevalence of hypomagnesemia in diabetics with retinopathy and without retinopathy (31%; $P< 0.005$).

The mechanism by which hypomagnesemia predisposes to diabetic retinopathy is not clearly defined. Grafton et al.[16] have proposed the inositol transport theory to explain this association. But the exact reason remains unclear. Dipankar Kundu et al. in 2013[17] concluded that hypomagnesemia and albuminuria individually or in conjunction serve as indicators for dysglycemia and could be used as marker for the risk of development of diabetic retinopathy. Like other diabetic microangiopathies, diabetic nephropathy was not significantly associated with prevalence of hypomagnesemia in our study. These results are similar to those reported by Sakaguchi Y et al.[18] 2012 in which they have included 455 chronic kidney disease (CKD) patients (144 with type 2 diabetic nephropathy and 311 with nondiabetic CKD) and participants were categorized based on serum Mg level into Low-Mg (serum Mg level ≤1.8 mg/dL) and High-Mg (serum Mg level >1.8 mg/dL) groups. From the subjects with type 2 diabetic nephropathy, 102 progressed to ESRD during follow-up (median, 23 months) and the Low-Mg group had a 2.12-fold higher risk of ESRD than the High-Mg group (95% CI 1.28-3.51; P = 0.004). In contrast, 135 of the nondiabetic CKD subjects progressed to ESRD during

follow-up (median, 44 months). No significant difference in outcome was found between the Low- and High-Mg groups of this population (adjusted hazard ratio, 1.15; 95% CI 0.70-1.90; P = 0.57), concluding that hypomagnesemia is a novel predictor of ESRD in patients with type 2 diabetic nephropathy.

In contrast, Corsonello et al[19] demonstrated significantly lower serum magnesium in type 2 diabetics with nephropathy compared to a normoalbuminuric group. They argued that in diabetics with nephropathy, serum magnesium might be reduced because of lower serum albumin concentration, as 30% of serum magnesium is bound to proteins, mainly albumin.

In our study, 15 patients had microalbuminuria and 3 patients who had macroalbuminuria had 24-hour albumin excretion less than 1.5 mg. This should not lower plasma albumin, because plasma contains macro amounts (35-52 g/L) of albumin.

In our study, we found statistical significance with diabetic neuropathy with hypoglycemia. The patients with diabetic neuropathy had a slightly higher prevalence of hypomagnesemia compared to those without neuropathy (69% v/s 31%) and the difference was statistically significant.

Experimental studies have also shown that hypomagnesemia inhibits prostacyclin receptor function, producing an imbalance between prostacyclin and thromboxane effects. In present study among the lipid profile abnormality, a significant inverse correlation was found between hypomagnesemia (S.Mg ≤ 1.7) and Serum triglycerides P= 0.059, and Serum HDL cholesterol levels P= 0.055, but no correlation was found with magnesium levels and Serum cholesterol. In the study done by Hamid Nasri et al.[20] 2008, had no significant correlation between serum Mg with serum HDL and Triglyceride were found, which needs further investigation

Serum levels of magnesium have been found by several investigators to correlate inversely with fasting blood gluscose concentration and the percentage of HbA1C[21] . The present study revealed no statistically significant correlation between serum magnesium levels and fasting blood sugar and HbA1C.

However, patients with poor glycemic parameters (FBS>130 mg/dl or HbA1C > 7%) had a statistically significantly correlation with P= 0.034,of hypomagnesemia compared to overall prevalence in diabetics [31%]. Tilal Abd El-Hameed Seedahmed et al.[22] studied a significant reduction in the mean of the plasma levels of magnesium of the diabetic group when compared with the control group (p. value <0.05), whereas HbA1C were increased in diabetic patients (type 2) compared to the control group (p. value <0.05). However, there was a strong negative correlation between the plasma levels of magnesium and HbA1C levels in diabetic patients, also there was a weak negative correlation between the plasma levels of magnesium and duration of disease.

Schlienger et al. [23] studied the influence of glycemic control (glycemic control evaluated by HbA1C) on various trace elements and reported significantly reduced plasma magnesium levels in patients with poor control of diabetes. Higher percentages of HbA1C indicate poor glycemic control in the previous months.

Hypomagnesemia is reported to be both a cause and result of poor glycemic control. Magnesium is a cofactor in both glucose transporting mechanisms of cell membrane and various enzymes important in carbohydrate oxidation. In addition, magnesium deficiency has been shown to promote insulin resistance in multiple studies. Nadler et al.[2] have reported that insulin sensitivity decreases even in nondiabetic individuals after induction of magnesium deficiency. Like-wise, elderly subjects were shown to have improved

glucose tolerance when they received magnesium supplements. Thus, hypomagnesemia by itself results in poor glycemic control. Conversely, hyperglycemia and osmotic diuresis may lead to increased urinary magnesium excretion and hypomagnesemia in diabetics.

However, high prevalence of hypomagnesemia is reported in type – 2 diabetics with good glycemic control. So, although poor glycemic control is associated with magnesium deficiency, it is not simply induced by hyperglycemia and is not corrected by improvement in metabolic control alone.

Limitations

Because of small sample size, inherited bias and errors do apply to this study. Association of Ischemic heart disease with hypomagnesemia could not be studied as those patients were excluded. Long term follow-up of patients after replacement of oral magnesium was not carried out.

References

1. Hans, Chetan P., et al. "Magnesium Deficiency and Diabetes Mellitus." Current Science, JSTOR 2002;83(12): 1456–63.,
2. Nadler JL, Buchnan T, Natarajan R, Antonipillai I, Bergman R, Rude RK. Magnesium deficiency produces insulin resistance and increased thromboxane synthesis. Hypertension. 1993; 21: 1024-9.
3. Paolisso G, Barbagallo M. Hypertension, diabetes mellitus, and insulin resistance: the role of intracellular magnesium. Am J Hypertens. 1997 Mar;10(3):346-55.
4. Ninomiya T. Japanese Legacy Cohort Studies: The Hisayama Study. J Epidemiol. 2018 Nov 5;28(11):444-451.
5. Wawrzyniak, A.J. Framingham Offspring Study. In: Gellman, M.D., Turner, J.R. (eds) Encyclopedia of Behavioral Medicine. Springer, New York, NY. 2013.
6. Larsson SC, Wolk A. Magnesium intake and risk of type 2 diabetes: a meta-analysis. J Intern Med 2007; 262:208-14.
7. Schulze MB, Schulz M, Heidemann C, Schienkiewitz A, Hoffmann K, Boeing H. Fiber and magnesium intake and incidence of type 2 diabetes: a prospective study and meta-analysis. Arch Intern Med 2007;167:956–65.
8. Djurhuus, Mogens & Skøtt, P & Vaag, Allan & Hother-Nielsen, Ole & Andersen, Pernille & Parving, H & Klitgaard, N. Hyperglycaemia enhances renal magnesium excretion in Type 1 diabetic patients. Scandinavian journal of clinical and laboratory investigation. 2000; 60:403-9.
9. Yajnick CS, Smith RF, Hockaday TDR, Ward NI. Fasting plasma magnesiu m concentration and glucose disposal in diabetes. BMJ 1984; 288: 1032-4.
10. Walti MK, Zimmermann MB, Hurrell RF. Low plasma magnesium in type-II diabetes. Swiss Med Wkly. 2003; 133: 289-92.
11. Alzaida A, Dinneen SF, Moyer TP, Rizza RA. Effects of insulin on plasma magnesium in noninsulin dependent diabetes mellitus – evidence for insulin ressitance. J Clin Endocrinol Metab. 1995; 80: 1376-81.
12. Resnick LM. Cellular ions in hypertension, insulin resistance, obesity, and diabetes: a unifying theme. J Am Soc Nephrol 1992;3:S78–S85.
13. Liao F, Folsom AR, Brancati FL. Is low magnesium concentration a risk factor for coronary heart disease? The ARIC study. Am Heart J. 1998; 136: 480-90.
14. Abraham AS, Rosenmann M, Kramer M. Magnesium in the prevention of lethal arrhythmias in acute myocardial infarction. Arch Intern Med. 1987; 147: 753-5.
15. Kareem I, Jaweed SA, Bardapurkar JS, Patil VP, et al. Study of magnesium, glycosylated hemoglobin and lipid

profile in diabetic retinopathy-Indian J Clin Biochem. 2004 Jul;19(2):124-7.
16. Grafton G, Baxter MA, Sheppard MC. Effects of magnesium on sodium dependant inositol transport. Diabetes. 1992; 41: 35-9.
17. Dipankar Kundu, Manish Osta, Tridibeswar Mandal, Ujjwal Bandyopadhyay, Debes Ray, and Divyendu Gautam, et al. Serum magnesium levels in patients with diabetic retinopathy-J Nat Sci Biol Med. 2013 Jan-Jun; 4(1): 113–116.
18. Sakaguchi Y, Shoji T, Hayashi T, Suzuki A, Shimizu M, Mitsumoto K, Kawabata H, Niihata K, Okada N, Isaka Y, Rakugi H, Tsubakihara Y, et al. Hypomagnesemia in type 2 diabetic nephropathy: a novel predictor of end-stage renal disease-Diabetes Care. 2012 Jul;35(7):1591-7.
19. Corsonello A, Ientile R, Buemi M, Cucinotta D, Mauro VN, Macaione S, Corica F. Serum ionized magnesium levels in type 2 diabetic patients with microalbuminuria or clinical proteinuria. Am J Nephrol. 2000 May-Jun;20(3):187-92.
20. Lipids in association with serum magnesium in diabetes mellitus patients Hamid Nasri, Hamid-Reza Baradaran-Bratisl Lek Listy 2008; 109 (7)
21. Kareem I, Jaweed SA, Bardapurkar JS, Patil VP, et al. Study of magnesium, glycosylated hemoglobin and lipid profile in diabetic retinopathy-Indian J Clin Biochem. 2004 Jul;19(2):124-7.
22. Corsonello A, Ientile R, Buemi M, Cucinotta D, Mauro VN, Macaione S, Corica F. Serum ionized magnesium levels in type 2 diabetic patients with microalbuminuria or clinical proteinuria. Am J Nephrol. 2000 May-Jun;20(3):187-92.
23. Halder, Subrata & Roy, Koushik & Biswas, Kaushik & Biswas, Priyankar & Sarkar, Ramita. (2016). Study of relation of serum magnesium level with glycemic control in diabetes mellitus. International Journal of Research in Medical Sciences. 4429-4433.