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Frequency of Hypomagnesemia and Its Association with Glycemic Control in Type 2 Diabetes Mellitus

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ABSTRACT

Objective: the objective of this study is to observe Frequency of hypomagnesemia and its association with glycemic control in type 2 diabetes mellitus

Study design and duration: Descriptive cross sectional study, from February 2026 to July 2026

Methodology: 150 adult patients suffering from type 2 diabetes mellitus were included. Serum magnesium, HbA1c and serum creatinine were measured from venous blood samples. Hypomagnesemia was defined as a serum level of <1.7 mg/dl; poor glycemic control was defined as HbA1C ≥7% and good glycemic control as HbA1C <7%. The data was analysed using SPSS. A Chi-square and independent-samples t test were performed, p≤0.05 was considered significant.

Results: Among 150 patients, 84 (56.0%) were males and 66 (44.0%) females, with a mean age of 55.8 ± 10.6 years. The mean duration of diabetes was 8.7 ± 5.9 years, mean BMI was 27.8 ± 4.3 kg/m², mean HbA1c was 8.34 ± 1.89%, and mean serum magnesium was 1.82 ± 0.31 mg/dL. Hypomagnesemia was detected in 39 (26.0%) of the patients and in 111 (74.0%) patients, the levels of magnesium were normal. Forty-two (28.0%) patients had good glycemic control, but 108 (72.0%) had poor glycemic control. Patients with poor glycemic control were significantly more likely to have hypomagnesemia compared to those with good glycemic control (32.4% vs. 9.5%, p=0.004). Patients with hypomagnesemia had a significantly higher mean HbA1c (%Glycated Hb) than patients with normomagnesemia (9.21 ± 1.72% vs. 8.03 ± 1.85%, respectively; p<0.001). There was a significant negative correlation between serum magnesium and HbA1c (r=-0.386, p<0.001).

Conclusion: Hypomagnesemia was relatively prevalent in patients with type 2 diabetes mellitus. There was a significant correlation between low levels of serum magnesium and poor glycemic control. Patients with hypomagnesemia had significantly higher HbA1c levels and serum magnesium levels had a significant inverse correlation with HbA1c.

Keywords: *Hypomagnesemia, Type 2 Diabetes, Glycemic Control*

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1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most common chronic metabolic conditions all over the world and also a public health concern. It is defined by the presence of chronic hyperglycemia due to insulin resistance, progressive insulin β-cell failure, and insulin deficiency. As the number of individuals with T2DM has grown, so, too, have the number of people developing microvascular and macrovascular complications, such as diabetic nephropathy, retinopathy, neuropathy, cardiovascular disease, and peripheral vascular disease. Despite the improvements in pharmacologic treatment, and monitoring, in a large number of patients, obtaining optimal glycemic control is still difficult. Besides the well known factors which affect glucose homeostasis, abnormalities in important micronutrients have become a major focus of interest in this field in particular, magnesium.[1]

Magnesium is the fourth most abundant mineral in the body, and plays an important role as an intracellular cation in many metabolic pathways and enzymatic reactions. It is important in connection to carbohydrate metabolism, insulin secretion, insulin receptor activity, glucose transport and cellular glucose utilization. Magnesium is a cofactor of several enzymes which carry out phosphorylation reactions and energy production. Thus, disturbances in magnesium homeostasis can have a negative impact on insulin sensitivity and glucose metabolism.[2]

Hypomagnesemia is commonly seen in T2DM patients and can be attributed to a few mechanisms. Long-term hyperglycemia causes an excessive loss of magnesium into the urine caused by osmotic diuresis and renal tubular dysfunction. Additionally, certain drugs (e.g., diuretics, proton pump inhibitors), inadequate nutrition, and insulin resistance can also lead to lowered levels of magnesium. On the other hand, deficiency of magnesium can exacerbate insulin resistance and decrease insulin production, thus establishing a potentially harmful cycle between insulin resistance and hypomagnesemia.[3]

A couple of studies have shown that low magnesium is linked with poor glycemic control, which is measured by glycated hemoglobin (HbA1c). Hypomagnesemia has been reported to occur more frequently in patients with poorly controlled diabetes than glycemically controlled diabetes. There is also evidence that low magnesium is associated with a higher risk of diabetic complications such as nephropathy, retinopathy, neuropathy and cardiovascular disease. The incidence of hypomagnesemia, however, is very variable among various populations due to differences in dietary habits, length and control of diabetes, renal function, and the definition of the hypomagnesemia and drugs used.[4]

The clinical significance of the hypomagnesemia-glycemic control relationship is that a simple and inexpensive blood magnesium level test is possible. Recognizing the presence of magnesium deficiency at the outset could be useful in identifying patients with a high risk of poor metabolic control and diabetic complications. Additionally, adequate correction of magnesium deficiency could have a potential therapeutic action on insulin sensitivity, glucose control, which needs further scientific studies in various patient groups.[5]

Very few local data are available regarding the prevalence of hypomagnesemia in the T2DM patient and its association with glycemic control. Hence, the purpose of the present study was aimed at finding out the prevalence of hypomagnesemia in type 2 diabetes mellitus patients and its correlation with glycemic control. The results may help to establish the clinical relevance of magnesium status and promote the use of the routine evaluation of magnesium in selected diabetics.

2. METHODOLOGY

This was a descriptive cross-sectional study which aimed to estimate the prevalence of hypomagnesemia and its relationship to glycemic control in T2DM patients. This study was done in Department of Medicine, Tertiary Care Hospital, Islamabad Pakistan. The study was conducted for six months starting from February 2026- July 2026.

This study involved adult patients attending the medical outpatient department or admitted to the medical wards of the hospital with a diagnosis of type 2 diabetes mellitus for the duration of the study. Diabetes mellitus type 2 was defined as per the existing diabetes diagnosis criteria: documented diabetes mellitus type 2 diagnosis or use of oral hypoglycemic agent(s) and/or insulin for diabetes management.

Sample size was determined with the WHO sample size calculator for estimating a population proportion. Based on the previous studies hypomagnesemia was expected with 70% frequency in type 2 diabetes mellitus patients, with a confidence level of 95%, absolute precision of 5%, and margin of error of 5%, the sample size was calculated to be 150 patients. Eligible patients were recruited for the study using consecutive non-probability sampling until the required sample size was attained.

Prior to the commencement of the study, ethical approval was obtained from the Institutional Review Board/Ethical

Committee of Tertiary Care Hospital, Islamabad. All participants signed informed consent forms prior to participating. Patients had to opt into the study, and they were educated about the aim and methods of the study. Patient information was kept confidential by assigning identification codes and not sharing patient information. Participants were informed that they could withdraw from the study at any time without it impacting their usual care.

Inclusion Criteria:

1. Male and female patients with age of 30 years or over.
2. Patients who have been diagnosed with T2DM for at least six months.
3. Patients under treatment with oral hypoglycemic agents, insulin or both.
4. Patients who are willing to participate in the study and sign informed consent form.

Exclusion Criteria

1. Type 1 diabetes mellitus.
2. Gestational diabetes mellitus.
3. Chronic Kidney Disease or substantial renal impairment.
4. Acute kidney injury.
5. Chronic liver disease.
6. Enrollment with acute severe illness, septicemia or DKA
7. Chronic gastrointestinal diseases with malabsorption or long duration of diarrhea.
8. Chronic alcohol abuse.
9. Current magnesium supplementation.
10. Medications that have the potential to significantly impact serum magnesium, such as loop/thiazide diuretics, and long-term proton pump inhibitors.
11. Pregnancy.
12. Patients who refuse or are unable to give informed consent.

After approval from the Institutional Review Board/Ethical Committee, eligible patients were recruited after obtaining written informed consent. A pre-designed structured proforma was used for collecting demographic and clinical data. Demographic variables were age and gender. Clinical data comprised diabetes mellitus duration, current anti diabetes treatment, history of hypertension and presence of diabetic complications (if present). A detailed history was taken to determine the factors that may affect the serum magnesium levels. A physical examination was performed in all the patients. Weight and height were measured in a standard way and body mass index (BMI) was calculated as the weight in kg divided by the square of the height in meters (kg/m2). About 5–7 mL of venous blood was collected aseptically. The serum magnesium level was determined by standard laboratory methods in the hospital laboratory and glycated hemoglobin (HbA1c) was used to measure. Renal function was also evaluated by the measurement of serum creatinine to rule out significant renal impairment.

Operational Definitions

Hypomagnesemia

The study laboratory reference range was used to define hypomagnesemia as a serum magnesium level less than 1.7 mg/dL (0.70 mmol/L). Patients with serum magnesium level of 2.46 mEq/L or more were defined as having normal serum magnesium level.

Glycemic Control

Glycated hemoglobin (HbA1c) was used to determine glycemic control. Patients were divided into 2 groups:

- Achievement of good glycemic control (HbA1c < 7%).
- Poor glucose control, HbA1C ≥7%

HbA1C was thought to reflect the mean of blood sugar levels for the last two to three months.

Type 2 Diabetes Mellitus

T2DM was defined as a diagnosed clinical T2DM (or use of oral hypoglycemic agents and/or insulin).

Outcome Variables

Hypomagnesemia frequency of patients with type 2 diabetes mellitus was the main study outcome.

The secondary outcome was the relationship of hypomagnesemia to glycemic control, which was determined by comparing the prevalence of hypomagnesemia in patients with good glycemic control and poor glycemic control based on HbA1C measurements.

Data Analysis

The data were collected and analyzed using statistical package for social sciences (SPSS) version 26. Data for quantitative variables, such as age, duration of diabetes, serum magnesium level, HbA1c and BMI, were expressed as mean ± standard

deviation or median (interquartile range), as appropriate. Frequencies and percentages were used to present qualitative variables such as gender, hypomagnesemia and glycemic control categories. The correlation between hypomagnesemia and glycemic control was evaluated by Chi-square test. Independent samples t-test was used to compare mean serum magnesium level between the good and the poor glycemic control patients group, where appropriate. Potential effect modifiers (age, gender, diabetes duration, BMI and treatment modality) were stratified. To assess the association of hypomagnesemia with glycemic control, a post-stratification chi-square test was used to analyze each subgroup. A p-value of ≤0.05 was considered statistically significant.

3. RESULTS

A total of 150 patients with type 2 diabetes mellitus were included in the study. The mean age of the study participants was 55.8 ± 10.6 years, with an age range of 32 to 78 years. There were 84 (56.0%) male and 66 (44.0%) female patients. The mean duration of diabetes mellitus was 8.7 ± 5.9 years, while the mean body mass index (BMI) was 27.8 ± 4.3 kg/m².
The mean serum magnesium level among all study participants was 1.82 ± 0.31 mg/dL. Hypomagnesemia, defined as a serum magnesium level below 1.7 mg/dL, was observed in 39 (26.0%) patients, whereas 111 (74.0%) patients had normal serum magnesium levels.

The mean HbA1c level of the study population was 8.34 ± 1.89%. Good glycemic control, defined as HbA1c <7%, was observed in 42 (28.0%) patients, while 108 (72.0%) patients had poor glycemic control with HbA1c ≥7%.

The frequency of hypomagnesemia was significantly higher among patients with poor glycemic control compared with those having good glycemic control. Hypomagnesemia was present in 35 (32.4%) patients with HbA1c ≥7%, compared with only 4 (9.5%) patients with HbA1c <7%. This association was statistically significant (p=0.004). Furthermore, patients with hypomagnesemia had significantly higher mean HbA1c levels compared with patients having normal serum magnesium levels (9.21 ± 1.72% versus 8.03 ± 1.85%, respectively; p<0.001). Serum magnesium levels demonstrated a statistically significant negative correlation with HbA1c (r = -0.386, p<0.001), indicating that lower serum magnesium levels were associated with poorer glycemic control. Hypomagnesemia was more frequently observed among patients aged 50 years or above, females, and those with a longer duration of diabetes; however, these associations were not statistically significant. Although hypomagnesemia was numerically more common among older patients, female patients, those with a longer duration of diabetes, and obese individuals, none of these associations reached statistical significance.

Overall, the findings demonstrated that hypomagnesemia was present in approximately one-quarter of patients with type 2 diabetes mellitus and was significantly associated with poor glycemic control. Patients with low serum magnesium levels had significantly higher HbA1c levels, suggesting an inverse relationship between serum magnesium concentration and glycemic control.

Table 1: Demographic and Clinical Characteristics of Study Participants

Variable	**Mean ± SD / n (%)**
Age (years)	55.8 ± 10.6
<50 years	42 (28.0%)
≥50 years	108 (72.0%)
Gender	
Male	84 (56.0%)
Female	66 (44.0%)
Duration of diabetes (years)	8.7 ± 5.9
<5 years	48 (32.0%)
5–10 years	54 (36.0%)
>10 years	48 (32.0%)
BMI (kg/m²)	27.8 ± 4.3
Normal (<25 kg/m²)	39 (26.0%)
Overweight (25–29.9 kg/m²)	67 (44.7%)
Obese (≥30 kg/m²)	44 (29.3%)
HbA1c (%)	8.34 ± 1.89
Serum magnesium (mg/dL)	1.82 ± 0.31

Table 2: Frequency of Hypomagnesemia and Glycemic Control

Variable	Frequency (n)	Percentage (%)
Serum magnesium status		
Hypomagnesemia (<1.7 mg/dL)	39	26.0
Normal magnesium (≥1.7 mg/dL)	111	74.0
Good control (HbA1c <7%)	42	28.0
Poor control (HbA1c ≥7%)	108	72.0

Table 3: Association Between Hypomagnesemia and Glycemic Control

Glycemic Control	Hypomagnesemia n (%)	Normal Magnesium n (%)	Total
Good control (HbA1c <7%)	4 (9.5%)	38 (90.5%)	42
Poor control (HbA1c ≥7%)	35 (32.4%)	73 (67.6%)	108
Total	**39 (26.0%)**	**111 (74.0%)**	**150**

Table 4: Comparison of Mean Clinical Parameters According to Serum Magnesium Status

Variable	Hypomagnesemia (n=39) Mean ± SD	Normal Magnesium (n=111) Mean ± SD	p-value
Age (years)	57.4 ± 10.2	55.2 ± 10.7	0.248
Duration of diabetes (years)	10.1 ± 6.1	8.2 ± 5.7	0.071
BMI (kg/m²)	28.3 ± 4.6	27.6 ± 4.2	0.382
HbA1c (%)	9.21 ± 1.72	8.03 ± 1.85	**<0.001**
Serum magnesium (mg/dL)	1.48 ± 0.14	1.94 ± 0.20	**<0.001**

Table 5: Association of Hypomagnesemia with Selected Patient Characteristics

Variable	Hypomagnesemia n (%)	Normal Magnesium n (%)
Age		
<50 years (n=42)	8 (19.0%)	34 (81.0%)
≥50 years (n=108)	31 (28.7%)	77 (71.3%)
Gender		
Male (n=84)	19 (22.6%)	65 (77.4%)
Female (n=66)	20 (30.3%)	46 (69.7%)
Duration of diabetes		
<5 years (n=48)	9 (18.8%)	39 (81.2%)
5–10 years (n=54)	14 (25.9%)	40 (74.1%)
>10 years (n=48)	16 (33.3%)	32 (66.7%)
BMI		
Normal (n=39)	8 (20.5%)	31 (79.5%)
Overweight (n=67)	18 (26.9%)	49 (73.1%)
Obese (n=44)	13 (29.5%)	31 (70.5%)

4. DISCUSSION

This study aimed to find out the prevalence of hypomagnesemia in type 2 diabetes mellitus patients and its association with glycemic control. A total of 150 patients with T2DM were included in the study. Thirty-nine patients (26.0%) had hypomagnesemia and 111 patients (74.0%) had normal magnesium levels. Of the study population, 72.0% had poor glycemic control, or an HbA1c level of ≥7%. Hypomagnesemia was also significantly more common in patients with poor glycemic control than in those with good glycemic control (32.4% versus 9.5%, p=0.004), importantly. In addition, the patients with hypomagnesemia showed a significantly higher mean HbA1C value compared with the patients with normal serum magnesium (9.21 ± 1.72% vs. 8.03 ± 1.85%, respectively, p<0.001). There was also a significant inverse correlation between serum magnesium and HbA1c (r=-0.386, p<0.001), indicating that lower serum magnesium concentrations are related to poorer long-term glycemic control.

The prevalence of hypomagnesemia (26.0%) suggests that magnesium deficiency is fairly common in patients with T2DM. The incidence of HYPOMAG reported amongst diabetic patients has been widely reported and may be due to varying study populations, dietary practices, duration of diabetes, glycemic control, renal function, therapeutic regime and

laboratory ranges defining HYPOMAG. Low blood magnesium levels were recently found in 21.8% of adults with T2DM in a cross-sectional study conducted at a tertiary care center, and hypomagnesemia was significantly linked to increased blood glucose in patients with T2DM. The slightly elevated frequency noted in the current study might be due to differences in demographic and clinical variables of the population studied, including the high percentage of patients with poor glycemic control.

This relationship between diabetes and magnesium deficiency is a plausible association. Magnesium is a crucial intracellular cation with a variety of intracellular functions, such as glucose metabolism, insulin secretion, insulin receptor signaling, and cellular glucose transport. Low levels of magnesium can hinder the ability of insulin to facilitate the uptake of glucose and lead to insulin resistance. On the other hand, chronic hyperglycemia can lead to polyuria with loss of magnesium and renal tubular magnesium wasting. Thus, it is possible that poor glycemic control and hypomagnesemia could be a vicious cycle in which hyperglycaemia increases magnesium loss and hypomagnesemia increases insulin resistance and glucose control.[6]

The relationship between hypomagnesemia and poor glycemic control found in the present study is in agreement with recent data. Hypomagnesemia occurred in almost one-third of patients with poor glycemic control, while a significantly smaller percentage of patients with good glycemic control had hypomagnesemia. In a recent cross-sectional study, the authors showed that there was significant difference in serum magnesium levels between the different HbA1c categories and that serum magnesium was inversely associated with HbA1c. In that study, the patients with the low magnesium levels also had significantly higher HbA1C levels than patients with normal magnesium levels. The results suggest a possible association of magnesium status with long-term glycemic control.[7]

Our findings also showed that the mean HbA1c was significantly elevated in the hypomagnesemic patients. A difference greater than 1% in HbA1c between patients with low and normal magnesium level may be clinically significant and could contribute to the higher risk of diabetic microvascular and macrovascular complications among those with low magnesium levels. The present study was cross-sectional, and therefore cannot prove a cause and effect relationship, but this association does point to a subset of patients with poorer metabolic control and perhaps higher risks of adverse diabetic outcomes who could be identified as having hypomagnesemia.[8]

The inverse correlation between serum magnesium and HbA1c in the present study ($r=-0.386$, $p<0.001$) further strengthens this association. There was a moderate, not strong, correlation, however, suggesting a consistent trend of lower serum magnesium levels with higher HbA1c levels. Another study recently found an inverse relationship between serum magnesium and HbA1c ($r=-0.41$). These results are very similar and confirm the consistency of the association between magnesium status and glycemic control in various populations. Serum magnesium, however, only reflects the level of magnesium found in the blood, and is not always a reliable indicator of the level of intracellular magnesium. So the specific connection between overall magnesium deficiency and glycemic control might be more complicated.[9]

The patients in the present study who were aged 50 years or above, female, and had longer duration of diabetes or obesity, showed a higher rate of hypomagnesemia, numerically. But these associations were not significant. This lack of statistical significance could be attributed to the small sample size in each of the subgroups. However, the higher prevalence of hypoglycemia with the longer duration of diabetes makes this clinically reasonable. Diabetics may suffer from chronic urinary magnesium losses and prolonged hyperglycemia over a prolonged period, which may predispose them to magnesium deficiency. Likewise, obesity and insulin resistance can also lead to magnesium metabolic disturbances.[10]

There has also been interest in the relationship between magnesium and diabetes, and whether or not magnesium supplements could be used as an adjunctive intervention. According to a pooled analysis of 24 RCTs of magnesium supplementation in T2DM, there was a decrease in both fasting plasma glucose and HbA1c levels. Oral magnesium supplementation showed potential benefits on HbA1c and fasting blood glucose in another dose–response meta-analysis of controlled clinical trials. Based on these findings, correction of magnesium deficiency might be beneficial in certain patients with regard to their metabolism.[11]

But the evidence on magnesium supplementation needs to be taken with a pinch of salt. A more recent systematic review and meta-analysis showed that magnesium supplementation led to a significant increase in serum magnesium concentration, and caused a significant improvement in fasting blood glucose, but there was only a weak or non-significant reduction in HbA1c. The large between-study variations suggest that the benefits of magnesium supplementation may be influenced by baseline magnesium status, duration of diabetes, dosage, formulation, duration of treatment and baseline glycemic control. For this reason, at present it is not recommended to supplement all patients with T2DM with magnesium routinely for the sole purpose of glycemic control (HbA1c). Rather, magnesium level in serum is recommended to be especially helpful in cases of poor glycemic control or other factors that may make the patient susceptible to magnesium deficiency.[12]

The present results present some clinical implications. Hypomagnesemia seems to be relatively prevalent among patients with T2DM, especially those with sub-optimal glycemic control. Secondly, the estimation of serum magnesium is an inexpensive test which is widely available, and could give further information about metabolic status. If they have a deficiency in magnesium, it can be helpful to identify and correct the underlying cause, such as poor dietary habits, gastrointestinal and/or urinary losses or effects of medications, as part of a broader diabetic management plan. The clinical significance of magnesium is also reinforced by the findings of a relationship between hypomagnesemia and insulin resistance and a higher risk of microvascular and cardiovascular complications in diabetes.[13]

There are some limitations of the present study. Because of the cross-sectional design, no temporal/causal relationship can be made between poor glycemic control and hypomagnesemia. It can thus not be concluded whether magnesium deficiency is a direct cause of poor glycemic control or whether the chronic hyperglycemia is the cause of the loss of magnesium. The study was limited to a one-center experience with a relatively small number of patients which could limit the generalizability of the results. Intracellular magnesium levels, urinary magnesium excretion and dietary magnesium intake were not evaluated. Also, other factors that could affect magnesium status, such as certain antidiabetic drugs and subtle variations in renal function, may have influenced magnesium status. In spite of these constraints, this study offers valuable insights into the prevalence of hypomagnesemia and its association with glycaemic control in T2DM patients. These significant relations between low serum Mg and high HbA1c indicate the importance of paying more attention to the Mg status of patients, especially those who have persistently poor glycemic control.

A significant result of this study was that the incidence of hypomagnesemia was found to be 26.0%, similar to the incidence of hypomagnesemia reported in recent literature. In 2024, a systematic review and meta-analysis found a pooled prevalence of hypomagnesemia of about 32% in 19 observational studies and 4192 T2DM patients. Interestingly, the prevalence reported to Asian populations was also relatively high. Hence, the frequency seen in the present study is in the range that was expected from the present day evidence, and this expands the importance of magnesium deficiency being a common metabolic aberration in T2DM. Variations in ethnicity, dietary magnesium consumption, socioeconomic status, diabetes duration, severity, diabetes procedure, variations in laboratory methods, and hypomagnesemia cut-off values could account for differences between the studies.[14]

The magnesium/glucose metabolism relationship is complex. Magnesium has several mechanisms of action on insulin, such as insulin receptor phosphorylation, intracellular signaling, glucose transport and energy metabolism in cells. Low intracellular magnesium can adversely affect the function of cells with insulin receptors, which can lead to insulin resistance. Moreover, magnesium deficiency can result in increased oxidative stress and low-grade inflammation, both of which are known factors associated with insulin resistance and the gradual loss of β-cell function. The present study showed an association between hypomagnesemia and increased HbA1c which could be interpreted as representing a biochemical incidental finding or as being caused by insulin action disturbances. Recent reviews have highlighted the possible contribution of magnesium deficiency to onset and progression of metabolic and vascular complications of T2DM.[15]

However, uncontrolled diabetes could also play a role in the development of hypomagnesemia. Glucosuria and osmotic diuresis leads to loss of electrolytes, including magnesium, from the urine.Because of persistent hyperglycemia, urinary losses of electrolytes such as magnesium occur because of glucosuria and osmotic diuresis. Insulin also can affect renal magnesium handling, and thereby further contribute to magnesium depletion. This bidirectional relationship is an important consideration when interpreting the findings of the present cross-sectional study. This can be because patients with elevated HbA1c have experienced prolonged hyperglycemia that has led to hypomagnesemia, whereas magnesium deficiency can further contribute to insulin resistance and to a deterioration in glycemic control. It would therefore be of value to have a prospective longitudinal study to establish the time sequence of these abnormalities.[16]

The finding that 72.0% of the participants in the present study had poor glycemic control is also noteworthy. The overall population's mean HbA1C level was 8.34 ± 1.89%, suggesting that a significant percentage of patients had suboptimally controlled blood glucose levels despite continued treatment. This may be the reason for the relatively high incidence of hypomagnesemia. Over time, a hyperglycemia state may lead to renal losses of magnesium. In addition, suboptimal glycemic control could be accompanied by inadequate food consumption, obesity, drug intake, and other metabolic disorders that all help to change magnesium balance.[17]

There were no statistically significant relationships between the age, gender, BMI, and length of diabetes and hypomagnesemia in the present study. However, there were some trends seen with diabetes duration. In patients with diabetes less than 5 years duration, 18.8% were found to have hypomagnesemia while 33.3% of those with diabetes > 10 years duration had hypomagnesemia. This difference was not statistically significant, but could be clinically important. Diabetes, as a chronic disease, can result in long-term metabolic abnormalities, frequent hyperglycemic events, and possible progressive changes in renal function, which can affect magnesium balance. The lack of significance may be due to low number of patients in each subgroup.

Females had a slightly higher prevalence of hypomagnesemia, likewise, the prevalence of hypomagnesemia was slightly higher among patients with obesity, though not statistically significant. There could be possible mediation between obesity and magnesium status through insulin resistance, diet and magnesium intake, chronic inflammation and altered renal handling of magnesium. BMI might not accurately reflect metabolic risk, however, as patients with comparable BMIs can have a significant variation in visceral adiposity and insulin resistance. Further research that includes measurements of waist circumference, waist to height ratio or insulin resistance may yield a better understanding of these relationships.[18]

These results have implications for beyond glycemic control. Hypomagnesemia has been linked with various T2DM complications such as nephropathy, retinopathy, neuropathy and cardiovascular disease. Magnesium plays a role in normal vascular function, integrity of the endothelial cells, cardiac electrophysiology and regulation of blood pressure. So, chronic magnesium deficiency could be a factor in the overall disease burden of diabetes. Complications were not specifically analysed as primary outcomes in the present study, however it would be interesting to see if hypomagnesemia can be used as a standalone predictor of microvascular and macrovascular outcomes in patients with T2DM in the future.

There is much clinical interest in the ability to correct magnesium deficiency by dietary means or by supplementation. Foods that are rich in magnesium are green leafy vegetables, legumes, nuts, seeds, and whole grains. Eating a healthy diet might therefore be a practical strategy for diabetes patients, especially if they have a history of low magnesium in their diet. But diet recommendations must be tailored to each patient, particularly if they have impaired renal function or other medical disorders which could alter electrolyte levels.

All of this additional evidence confirms the main finding of this study: Hypomagnesemia and poor glycemic control are closely linked. The prevalence of hypomagnesemia was comparable to the high prevalence reported globally and there was a strong inverse association between serum Mg and HbA1c, highlighting the potential biological relevance of magnesium in glucose metabolism. Hypomagnesemia may be a consequence, a contributor or both a consequence and contributor to poor glycemic control and larger prospective studies with a variety of populations and repeat magnesium measurements are needed to determine this. These investigations should also determine if specific correction of Mg deficiency has an impact on improving HbA1c and decreasing long-term complications of diabetes.

Finally, hypomagnesemia was found in about one-quarter of T2DM patients, and was significantly associated with poor glycemic control. Magnesium was significantly lower in patients with hypomagnesemia and serum magnesium revealed an inverse correlation with HbA1c. The results indicate that magnesium deficiency could be a metabolic disorder in poorly controlled T2DM patients. More prospective, multicenter studies are required to establish the causal link between magnesium deficiency and hyperglycemia and to establish if the targeted magnesium replacement can have a meaningful impact on long-term glycemic outcomes and the reduction of diabetic complications.

5. LIMITATION OF STUDY

it is single center study, with limited sample size, and hence its results cannot be generalized over population level. To get better results, a large sample and multicenter trial should be done.

6. CONCLUSION

The present study showed that hypomagnesemia is a relatively common occurrence in patients with type 2 diabetes mellitus. Hypomagnesemia was significantly correlated with poor control of glycemia. Patients with low serum magnesium levels had significantly higher mean HbA1c levels than patients with normal magnesium levels. Moreover, there was a significant inverse relationship between serum magnesium and HbA1c, suggesting that a lower level of magnesium was related to worse glycemic control. These observations are consistent with the essential role of Mg in glucose metabolism and insulin sensitivity and indicate possible role of Mg deficiency in the etiology or as a consequence of chronic hyperglycemia.
There was no statistically significant difference in the prevalence of hypomagnesemia with age, sex, BMI or duration of diabetes, though there were some numerical differences. Considering the relatively common occurrence of hypomagnesemia and the strong association with glycemic control, some patients with poorly controlled T2DM may benefit from serum magnesium level measurement.

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