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Frequency of Hypomagnesemia in Type II Diabetes Mellitus

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Abstract

Background: Type II diabetes mellitus (T2DM) is a global health burden characterized not only by hyperglycemia but also by associated metabolic and electrolyte disturbances. Objective: This study aimed to determine the frequency of hypomagnesemia in patients with T2DM and its association with clinical and biochemical parameters. Methodology: This cross-sectional study was conducted at the Department of Medicine, National Hospital and Medical Centre, from OCTOBER 2024 to March 2025. A total of 181 patients aged 40–65 years with T2DM for more than three years were included using non-probability consecutive sampling. Patients with type I diabetes, secondary causes of diabetes, gastrointestinal losses, alcohol abuse, or drugs affecting magnesium metabolism were excluded. Serum magnesium levels were measured using the Calmagite dye method, with values <1.8 mg/dL defined as hypomagnesemia. Results: The mean age of patients was 54.3 ± 6.1 years, with 97 (53.6%) males and 84 (46.4%) females. Hypomagnesemia was observed in 112 patients (61.9%), while 69 (38.1%) had normal serum magnesium levels. Patients with hypomagnesemia had significantly longer duration of diabetes (9.6 ± 3.5 vs. 7.2 ± 2.9 years, $p < 0.001$),

higher fasting blood glucose (164.7 ± 32.1 vs. 142.5 ± 29.8 mg/dL, p = 0.002), and elevated HbA1c (8.9 ± 1.2% vs. 7.8 ± 1.1%, p < 0.001) compared to those with normal magnesium levels.
Conclusion: Hypomagnesemia is a common finding in patients with type II diabetes mellitus and is significantly associated with poor glycemic control and longer disease duration.

Introduction

Type 2 diabetes mellitus (T2DM) accounts for more than 90% of diabetes cases and is characterized by insulin resistance and impaired β-cell function [1]. In the long term, the ultimate goal of T2DM is to prevent serious complications include cardio-cerebrovascular diseases like stroke and heart failure, nephropathy, diabetic retinopathy, diabetic foot, some kinds of cancers, cognitive decline, sleep apnea syndrome, mood disorders, bone metabolism impairments, and electrolyte imbalance [2]. The pathophysiology of T2DM is complex and multifactorial, involving genetic, environmental, and lifestyle factors. In recent years, there has been a lot of interest in the role that micronutrients, particularly magnesium, play in the onset and progression of T2DM [1,3]. It is currently unknown what causes hypomagnesemia in type 2 diabetes. In this regard, magnesium (Mg), a micronutrient, is particularly significant. Magnesium is the second most common intracellular cation and is the fourth most abundant mineral in the human body. A disturbance in the level of magnesium has been reported in diabetic individuals. Hypomagnesemia is common in diabetic patients [4]. Hypomagnesemia results in increased serum glucose levels, increased glucosuria, and insulin resistance. It can also lead to cardiovascular abnormalities [3]. Kumar et al. observed the mean magnesium level among diabetic and non-diabetic individuals. The average magnesium level in diabetic patients was 0.83 to 0.13 mmol/L, while the average magnesium level in non-diabetic individuals was 1.04 to 0.14 mmol/L (p=0.001). Hypomagnesemia was detected in 64% of diabetic patients [5]. Similarly, another study reported that the patients with hypomagnesemia have higher levels of fasting plasma glucose, postprandial plasma glucose (PPG), and serum glycosylated hemoglobin (HbA1c) compared to patients with normal magnesium levels. FPG levels were significantly higher in patients with hypomagnesemia in (179.0 ± 64.9 vs. 148.7 ± 52.0 mg/dl), PPG levels were (287.9 ± 108.4 vs. 226.8 ± 89.4 mg/dl) and Serum HbA1c levels were (8.0 ± 1.9% vs. 6.5 ± 1.2%) [6]. Some studies reported the association of hypomagnesemia with diabetes complications. There was also a significantly higher prevalence of hypomagnesaemia (52%) in DM nephropathy patients (mean = 1.62 ± 0.31 mg/dL) compared to without nephropathy patients (22%, mean = 1.86 ± 0.28 mg/dL) [7]. Paladiya et al. reported that in uncontrolled diabetic patients, mean magnesium level was significantly lower as compared to diabetic patients with good glycemic control (1.34 ± 0.3 mg/dL vs. 1.81 ± 0.5; p-value: <0.0001). Prevalence of hypomagnesemia was significantly more in patients with uncontrolled diabetes, compared to the controlled diabetic group (65.8% vs. 50.8%; p-value: 0.009). Prevalence of hypomagnesemia was significantly more in patients with diabetes for more than 10 years (64.7% vs. 51.9%; p-value: 0.02) [8,9]. Despite the importance of magnesium in T2DM, there is limited information on the frequency of hypomagnesemia in T2DM patients in our locality. The available studies in our region have reported varying prevalence rates of hypomagnesemia in T2DM patients.

Objective

Objective of this study is to determine the frequency of Hypomagnesemia in patients with type 2 diabetes mellitus.

Methodology

This Cross-Sectional study was conducted at Department of Medicine, National Hospital and Medical Centre from October 2024 to March 2025. A non-probability consecutive sampling technique was employed to recruit participants meeting the inclusion criteria. The sample size was calculated using the WHO sample size calculator. The following parameters were applied: a 95% confidence level, 7% absolute precision, and an expected frequency of hypomagnesemia of 64%. Based on these values, the required sample size was 181 patients.

Inclusion Criteria

All patients aged between 40–65 years.
The study will include patients who have been diagnosed with type 2 diabetes for over 3 years.

Exclusion Criteria

Patients with type I diabetes mellitus and secondary causes of diabetes mellitus, such as hemochromatosis, Cushing's disease, and acromegaly.
Patients with history of acute pancreatitis, alcoholism, diarrhea, vomiting, malabsorption, nasogastric suction, gastrointestinal fistulas, and hypoproteinemic states.
Patients using diuretics, antimicrobials such as Amphotericin B, aminoglycosides, pentamidine, capreomycin, viomycin, and foscarnet, chemotherapeutic agents such as cisplatin, immunosuppressants like tacrolimus and cyclosporine, and proton-pump inhibitors will also be excluded.

Data Collection Technique

After obtaining approval from the institutional ethical review committee and informed consent from all participants, data were collected systematically. Demographic information, including age, gender, duration of diabetes, and treatment history, was recorded for each patient. Blood pressure was measured as part of baseline clinical assessment. On the day of examination, patients were instructed not to take their morning insulin dose or any oral hypoglycemic drugs. A fasting venous blood sample (5–10 mL) was drawn from a peripheral vein, centrifuged, isolated, and stored at –20°C until analysis. Serum magnesium levels were estimated using the Calmagite dye method under the supervision of a chemical pathologist, and values below 1.8 mg/dL were labeled as hypomagnesemia. Additional biochemical investigations included fasting blood glucose, random blood glucose, and glycated hemoglobin (HbA1c), measured using a modified calorimetric method. Postprandial blood sugar (PPBS) levels were determined by obtaining a venous blood sample two hours after breakfast.

Data Analysis

Data analysis was carried out using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables such as age, duration of diabetes, serum magnesium, fasting blood sugar, postprandial blood sugar, and HbA1c were expressed as mean ± standard deviation (SD). Categorical variables, including gender, hypomagnesemia status, smoking history, and comorbidities, were presented as frequencies and percentages. To address effect modifiers, the data were stratified according to age, gender, body mass index (BMI), duration of disease, and medication use. Post-stratification, the chi-square test was applied, with a p-value ≤ 0.05 considered statistically significant.

Results

Data were collected from 181 patients. The mean age of participants was 54.3 ± 6.1 years, with 97 (53.6%) males and 84 (46.4%) females. The mean BMI was 27.8 ± 3.6 kg/m², with no significant gender differences (p = 0.29). The average duration of diabetes was 8.7 ± 3.4 years, slightly higher among females (9.1 ± 3.6 years) compared to males (8.4 ± 3.2 years), though this was not statistically significant (p = 0.21). Hypertension was present in 45.3% of patients and dyslipidemia in 37.0%, with no significant gender differences. Smoking, however, was significantly more common among males (32.0%) than females (9.5%) (p < 0.001).

Table 1. Baseline Demographic and Clinical Characteristics of Patients (N = 181)

Variable	Total (N = 181)	Male (n = 97)	Female (n = 84)	p-value
Age, years (mean ± SD)	54.3 ± 6.1	54.7 ± 6.3	53.8 ± 5.9	0.42
Gender, n (%)	181	97 (53.6)	84 (46.4)	–
BMI, kg/m² (mean ± SD)	27.8 ± 3.6	27.5 ± 3.4	28.1 ± 3.8	0.29
Duration of DM, years	8.7 ± 3.4	8.4 ± 3.2	9.1 ± 3.6	0.21
Hypertension, n (%)	82 (45.3)	47 (48.5)	35 (41.7)	0.37
Dyslipidemia, n (%)	67 (37.0)	36 (37.1)	31 (36.9)	0.98
Smoking, n (%)	39 (21.5)	31 (32.0)	8 (9.5)	<0.001

Out of 181 patients, 112 (61.9%) had serum magnesium levels below 1.8 mg/dL, while 69 (38.1%) had normal magnesium levels.

Table 2. Frequency of Hypomagnesemia in Patients with Type II Diabetes Mellitus (N = 181)

Serum Magnesium Level	Frequency, n (%)
Normal (≥1.8 mg/dL)	69 (38.1)
Hypomagnesemia (<1.8 mg/dL)	112 (61.9)

Patients with hypomagnesemia had a significantly longer duration of diabetes (9.6 ± 3.5 vs. 7.2 ± 2.9 years, p < 0.001), higher fasting blood glucose (164.7 ± 32.1 vs. 142.5 ± 29.8 mg/dL, p = 0.002), and elevated HbA1c (8.9 ± 1.2% vs. 7.8 ± 1.1%, p < 0.001) compared to those with normal magnesium levels. Postprandial glucose was also higher in the hypomagnesemia group (224.6 ± 44.9 vs. 198.4 ± 40.7 mg/dL, p = 0.004). Although BMI was higher in patients with hypomagnesemia (28.1 ± 3.7 vs. 27.2 ± 3.4 kg/m²), this difference did not reach statistical significance (p = 0.11).

Table 3. Association of Hypomagnesemia with Clinical and Biochemical Parameters

Variable	Normal Mg (n = 69)	Hypomagnesemia (n = 112)	p-value
Age, years (mean ± SD)	53.7 ± 6.0	54.6 ± 6.2	0.41
Duration of DM, years (mean ± SD)	7.2 ± 2.9	9.6 ± 3.5	<0.001
BMI, kg/m² (mean ± SD)	27.2 ± 3.4	28.1 ± 3.7	0.11
Fasting blood glucose (mg/dL)	142.5 ± 29.8	164.7 ± 32.1	0.002
Postprandial glucose (mg/dL)	198.4 ± 40.7	224.6 ± 44.9	0.004
HbA1c (%)	7.8 ± 1.1	8.9 ± 1.2	<0.001

Hypomagnesemia was more frequent among females (65.5%) compared to males (58.8%), but the difference was not statistically significant ($p = 0.38$). Older patients aged 56–65 years had a higher prevalence (68.2%) compared to those aged 40–55 years (57.4%), though this was not significant ($p = 0.09$). Similarly, overweight and obese patients (BMI $\geq$ 25 kg/m²) had higher rates of hypomagnesemia (65.1%) compared to those with normal BMI (52.9%) ($p = 0.06$). A significant association was observed between hypomagnesemia and duration of diabetes, with patients having diabetes for more than 10 years showing the highest prevalence (72.5%, $p < 0.01$).

Table 4. Stratification of Hypomagnesemia by Demographic and Clinical Factors

Variable	**Subgroup**	**Hypomagnesemia n (%)**	**p-value**
Gender	Male (n = 97)	57 (58.8)	0.38
	Female (n = 84)	55 (65.5)	
Age Group	40–55 years (n = 94)	54 (57.4)	0.09
	56–65 years (n = 87)	59 (68.2)	
BMI Category	<25 kg/m² (n = 51)	27 (52.9)	0.06
	≥25 kg/m² (n = 130)	85 (65.1)	
Duration of DM	≤10 years (n = 107)	63 (58.9)	<0.01
	>10 years (n = 74)	49 (72.5)	

Discussion

In this cross-sectional study conducted on 181 patients with type II diabetes mellitus, we found that hypomagnesemia was highly prevalent, affecting nearly two-thirds (61.9%) of the study population. This finding underscores the significance of magnesium deficiency as a common comorbidity in diabetes and highlights the importance of routine electrolyte monitoring in these patients. Our results are consistent with previous research, which has reported a high frequency of hypomagnesemia among patients with type II diabetes, ranging from 30% to 65%. Prior studies have demonstrated that magnesium deficiency is often underdiagnosed because it remains asymptomatic until severe, yet it exerts a profound effect on glucose metabolism, insulin sensitivity, and vascular health. The similarity of our results to earlier work strengthens the evidence that hypomagnesemia is not an isolated occurrence but rather an integral part of the diabetic metabolic disturbance [9-11].

We observed that patients with hypomagnesemia had significantly longer duration of diabetes, higher fasting and postprandial glucose levels, and elevated HbA1c compared to those with normal magnesium levels. This suggests a strong association between magnesium deficiency and poor glycemic control. Magnesium is a cofactor for enzymes involved in carbohydrate metabolism, insulin receptor signaling, and glucose transport. Its deficiency may impair insulin-mediated glucose uptake, exacerbate insulin resistance, and contribute to worsening hyperglycemia. Previous research has similarly linked low magnesium status with poor glycemic indices, which aligns closely with our findings. Interestingly, while hypomagnesemia was

more common among females, older patients, and those with higher BMI, these associations did not reach statistical significance in our study [12]. However, duration of diabetes showed a clear and significant relationship with hypomagnesemia, supporting the idea that chronic hyperglycemia and prolonged exposure to renal glycosuria increase urinary magnesium loss over time. These findings reinforce the concept that the longer a patient lives with diabetes, the greater the likelihood of magnesium depletion [13].

From a clinical perspective, magnesium deficiency has far-reaching implications in diabetes management. It has been associated not only with poor glycemic control but also with increased risk of complications such as hypertension, arrhythmias, atherosclerosis, and microvascular complications [14]. Early detection and correction of hypomagnesemia through dietary modifications or supplementation may represent a simple and cost-effective strategy to improve metabolic outcomes in diabetic patients. Despite this, magnesium assessment is rarely included in the standard monitoring of diabetic patients, highlighting a gap in current clinical practice. The strengths of this study include the use of a standardized biochemical method (Calmagite dye method) for magnesium estimation and a well-defined sample with clear inclusion and exclusion criteria [15-17]. However, some limitations should be acknowledged. First, this was a single-center study with a modest sample size, which may limit the generalizability of findings. Second, the cross-sectional design precludes causal inference; while hypomagnesemia is associated with poor glycemic control, we cannot determine whether it is a cause or consequence of uncontrolled diabetes. Finally, we did not assess dietary intake or urinary excretion of magnesium, which could have provided additional insights into the mechanisms of deficiency.

Conclusion

It is concluded that hypomagnesemia is highly prevalent among patients with type II diabetes mellitus, with nearly two-thirds of our study population affected. The condition was strongly associated with longer duration of diabetes and poor glycemic control, as reflected by higher fasting glucose, postprandial glucose, and HbA1c levels. These findings emphasize the need for routine monitoring of serum magnesium levels in diabetic patients, particularly those with long-standing disease.

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