Original Article

Association of Serum Magnesium Levels with Glycemic Control in Patients with Type 2 Diabetes Mellitus

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Abstract

Background: Type 2 diabetes mellitus (T2DM) is a prominent metabolic condition defined by enduring hyperglycemia caused by inadequate insulin production and insulin resistance. Magnesium, an essential intracellular cation, plays a vital role in glucose metabolism, insulin signaling, and enzymatic reactions. Magnesium insufficiency is commonly noted in individuals with T2DM and has been linked to inadequate glycemic regulation, insulin resistance, and the onset of diabetes complications. Evaluation of serum magnesium could serve as a significant biochemical indicator of metabolic condition. The aim is to assess serum magnesium concentrations in individuals with type 2 diabetes mellitus and examine their correlation with glycemic regulation. **Material and Methods:** A hospital-based analytical study of a cross-sectional nature was carried out in the Department of Biochemistry, in partnership with the Department of General Medicine, lasting 8 months from July 2025 to February 2026. The analysis featured 180 subjects with an established diagnosis of type 2 diabetes mellitus, identified thru consecutive sampling. Venous blood collections were performed under strict aseptic conditions. A completely automated biochemistry analyzer was used to estimate serum magnesium levels. Standard laboratory procedures were used to assess serum creatinine, glycated hemoglobin (HbA1c), fasting blood glucose, postprandial blood glucose, and lipid profile. SPSS version 25.0 was used for the statistical analysis. A statistically significant p-value was one that was less than 0.05. **Results:** The mean age of the participants was 56.7 ± 10.5 years, with males accounting for 104 (57.8%) of the study population. Hypomagnesemia (serum magnesium <1.7 mg/dL) was detected in 66 (36.7%) patients, whereas 111 (61.7%) had normal magnesium levels and 3 (1.6%) had elevated levels. The mean serum magnesium concentration was significantly lower among patients with poor glycemic control than those with good glycemic control (1.56 ± 0.24 mg/dL vs. 2.02 ± 0.20 mg/dL; p<0.001). **Conclusion:** Individuals with type 2 diabetes mellitus often experience hypomagnesemia. Lower serum magnesium levels correlated strongly with elevated fasting blood glucose, postprandial blood glucose, and HbA1c, highlighting the potential role of magnesium deficiency in impaired glucose metabolism and poor metabolic control. Estimation of serum magnesium may serve as a simple, cost-effective laboratory parameter for identifying diabetic patients at risk of poor metabolic control.

Keywords: Glycemic control; HbA1c; Hypomagnesemia; Serum magnesium; Type 2 diabetes mellitus.

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Introduction

Due to its rapidly rising incidence and related morbidity and mortality, type 2 diabetes mellitus (T2DM), one of the most common non-communicable illnesses in the world, has become a significant public health problem.[1] Chronic hyperglycemia brought on by insulin resistance, decreased insulin production, or a combination of the two is the hallmark of the illness.[2] According to the International Diabetes Federation (IDF), diabetes affects hundreds of millions of individuals globally, with India accounting for one of the largest diabetic populations.[3]

Patients who have persistent hyperglycemia are more likely to experience macrovascular problems including peripheral arterial disease, coronary artery disease, and stroke as well as microvascular problems like diabetic nephropathy, retinopathy, and neuropathy. Thus, the key to stopping the progression of the disease and enhancing long-term clinical results continues to be maintaining optimal glycemic control.[4,5]

After potassium, magnesium is the second most common intracellular cation and the fourth most common cation in the human body. Approximately 99% of total body magnesium is present within bones, muscles, and soft tissues, while less than 1% circulates in the extracellular fluid.6 Magnesium plays an essential role in insulin secretion, insulin receptor phosphorylation, intracellular glucose transport, and post-receptor insulin signaling. Consequently, alterations in magnesium homeostasis can adversely affect glucose metabolism and insulin sensitivity.[7]

Hypomagnesemia is frequently observed in patients with T2DM, with reported prevalence ranging from 20% to 40%. Reduced serum magnesium levels in diabetics are caused by a

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number of factors, including increased magnesium loss in the urine due to osmotic diuresis, poor intestinal absorption, insufficient nutritional intake, insulin resistance, and the use of certain drugs like diuretics.[8] Magnesium deficiency has been implicated in the progression of insulin resistance, impaired pancreatic β-cell function, increased oxidative stress, chronic low-grade inflammation, endothelial dysfunction, and enhanced platelet aggregation.[9]

Reduced serum magnesium has also been linked with dyslipidemia, hypertension, diabetic nephropathy, retinopathy, peripheral neuropathy, and cardiovascular disease. Since serum magnesium estimation is inexpensive, readily available, and routinely performed in clinical biochemistry laboratories, it may serve as a useful adjunctive biomarker for evaluating metabolic control in diabetic patients.[10,11]

Despite increasing evidence regarding the association between hypomagnesemia and T2DM, routine assessment of serum magnesium is not commonly incorporated into diabetic evaluation protocols, particularly in resource-limited settings. Furthermore, regional variations in dietary habits, socioeconomic conditions, lifestyle factors, and disease characteristics may influence magnesium status, emphasizing the need for locally generated evidence. Identifying hypomagnesemia at an early stage may facilitate timely nutritional intervention, magnesium supplementation when indicated, and improved metabolic management, hence lowering the possibility of long-term issues.

Aim: To calculate serum magnesium levels and assess how they relate to glycemic management in individuals with type 2 diabetes.

Materials and Methods

Over the course of eight months, from July 2025 to February 2026, this cross-sectional analytical investigation was carried out at the Sree Mookambika Institute of Medical Sciences in Kulasekharam, Tamil Nadu, in the Department of Biochemistry in cooperation with the Department of General Medicine. Prior to enrollment, each study subject provided written informed consent. The Sree Mookambika Institute of Medical Sciences, Kulasekharam's Institutional Ethics Committee examined and approved the study protocol.

Inclusion Criteria

- Individuals who have been diagnosed with type 2 diabetes mellitus and are at least thirty years old. • Individuals receiving inpatient or outpatient care during the study period who have type 2 diabetes mellitus.
- Patients willing to participate and provide written informed consent.
- Patients with complete clinical and biochemical investigations, including HbA1c and serum magnesium estimation.

Exclusion Criteria

- Individuals suffering from secondary diabetes, gestational diabetes, or type 1 diabetes.
- Individuals undergoing dialysis, acute renal damage, or chronic kidney disease (Stage 4 or Stage 5).
- Patients with chronic liver disease, pancreatitis, thyroid disorders, or parathyroid disorders.
- Patients receiving magnesium supplements or medications known to alter magnesium metabolism.
- Patients with acute infections, sepsis, malignancy, or critical illness.
- Pregnant or lactating women.
- Patients unwilling to participate or with incomplete clinical or laboratory data.

The calculation of the sample size employed the method, reflecting a hypomagnesemia prevalence of 37.2% among type 2 diabetes mellitus patients, as established by Abdullah M et al., alongside a 95% confidence level and 7.1% absolute precision. The formula was employed to compute the sample size:

Where:

- n = Required sample size
- Z = Standard normal deviate at 95% confidence level = 1.96
- p = Expected prevalence of hypomagnesemia among patients with type 2 diabetes mellitus = 37.2% (0.372)
- q = 1 − p = 62.8% (0.628)
- d = Absolute precision = 7.1% (0.071)

The calculated minimum sample size was 178 participants. To compensate for possible incomplete data and to improve statistical precision, 180 participants were ultimately included.

The American Diabetes Association (ADA) criteria or a prior medical diagnosis with continuous treatment were used to diagnose type 2 diabetes mellitus. A predesigned and pretested data collection proforma was used to record demographic information, clinical history, length of diabetes, treatment type, body mass index (BMI), blood pressure, and related comorbidities. A detailed clinical examination was performed for all participants.

Under aseptic precautions, approximately 5 mL of fasting venous blood was collected from each participant. Blood samples were centrifuged within one hour of collection, and the serum was analyzed immediately in the Clinical Biochemistry Laboratory. Fasting blood sugar (FBS), postprandial blood sugar (PPBS), serum creatinine, and lipid profile were estimated using standard enzymatic methods on Beckman Coulter AU480. Glycated hemoglobin (HbA1c) was measured using immunoturbidimetric assay as per laboratory protocol. Serum magnesium was estimated by the Xylidyl Blue colorimetric method, which is routinely employed for quantitative determination of serum magnesium. Two-level Internal Quality Control and participation in External Quality Assurance Scheme (EQAS) were followed throughout the study to ensure analytical accuracy and precision.

Patients were categorized into good glycemic control (HbA1c <7%) and poor glycemic control (HbA1c ≥7%) based on ADA recommendations. Serum magnesium values were classified as hypomagnesemia (<1.7 mg/dL), normal serum magnesium (1.7–2.4 mg/dL), and hypermagnesemia (>2.4 mg/dL). The relationship between serum magnesium levels and glycemic parameters was evaluated.

The Statistical Package for Social Sciences (SPSS) version 25.0 was used to examine the data after it was imported into Microsoft Excel. Categorical variables were shown as frequencies and percentages, whilst continuous variables were given as mean ± standard deviation (SD). Continuous variables

were compared between two groups using the student's independent t-test. One-way analysis of variance (ANOVA) was applied for comparison among multiple groups where appropriate. Associations between categorical variables were assessed using the Chi-square test. Pearson's correlation analysis was used to determine the relationship between serum magnesium levels and glycemic markers (FBS, PPBS, and HbA1c). Statistical significance was defined as a p-value of less than 0.05.

Results

Majority of study participants (35.0%) belonged to the 51–60 years age group, followed by those above 60 years (28.3%), with most participants belonged to the middle-aged and elderly age groups. [Table 1]

Table 1: Distribution of study participants according to age (n=180)

Age group (years)	Number (n)	Percentage (%)
30–40	24	13.33
41–50	42	23.33
51–60	63	35.00
>60	51	28.33
Total	180	100.00

Hypomagnesemia was observed in more than one-third (36.7%) of diabetic patients, indicating that magnesium deficiency is a common biochemical abnormality among individuals with type 2 diabetes mellitus. The mean serum magnesium concentration among study participants was 1.73 ± 0.34 mg/dL (95% CI: 1.68–1.78 mg/dL). [Table 2]

Table 2: Distribution according to serum magnesium levels (n=180)

Serum magnesium level	Number (n)	Percentage (%)
Hypomagnesemia (<1.7 mg/dL)	66	36.67
Normal (1.7–2.4 mg/dL)	111	61.67
Hypermagnesemia (>2.4 mg/dL)	3	1.66
Total	180	100.00

Patients with poor glycemic control had significantly higher fasting blood glucose, postprandial blood glucose, and HbA1c levels, whereas serum magnesium concentrations were significantly lower compared to patients with good glycemic control (p<0.001). [Table 3]

Table 3: Comparison of biochemical parameters according to glycemic control

Parameter	HbA1c <7% (n=58) Mean ± SD	HbA1c ≥7% (n=122) Mean ± SD	p value
Fasting blood glucose (mg/dL)	118.7 ± 18.9	176.5 ± 32.6	<0.001*
Postprandial blood glucose (mg/dL)	168.4 ± 26.3	254.7 ± 44.2	<0.001*
HbA1c (%)	6.42 ± 0.39	8.94 ± 1.21	<0.001*
Serum magnesium (mg/dL)	2.02 ± 0.20	1.56 ± 0.24	<0.001*
	Mean Difference (95% CI): 0.46 (0.39–0.53)		

Independent Student's t-test. *Significant (p<0.05)
FBG: Fasting Blood Glucose; PPBG: Postprandial Blood Glucose; HbA1c: Glycated Hemoglobin.

Hypomagnesemia was significantly more common among patients with poor glycemic control than those with good glycemic control, indicating a significant association between reduced serum magnesium levels and elevated HbA1c values. [Table 4]

Table 4. Association between serum magnesium status and glycemic control

Serum magnesium status	Good control (HbA1c <7%) n (%)	Poor control (HbA1c ≥7%) n (%)	Total	p value
Hypomagnesemia	6 (10.34)	60 (49.18)	66	<0.001*
Normal magnesium	50 (86.21)	61 (50.00)	111	
Hypermagnesemia	2 (3.45)	1 (0.82)	3	
Total	58	122	180	

Chi-square test; *Significant (p<0.05)
HbA1c: Glycated Hemoglobin.

Correlation analysis demonstrated a significant negative association between serum magnesium levels and all glycemic parameters. Serum magnesium showed a significant inverse correlation with FBS (r = -0.482, p <0.001) and PPBS (r = -0.527, p <0.001), while a stronger inverse correlation was observed with HbA1c (r = -0.614, p <0.001). These results suggest that diminished serum magnesium levels correlate with suboptimal glycemic regulation, evidenced by increased fasting and postprandial glucose concentrations and heightened HbA1c levels.

Patients with longer duration of diabetes demonstrated significantly higher FBS, PPBS, and HbA1c levels, accompanied by progressively lower serum magnesium concentrations, indicating worsening glycemic control and increasing magnesium deficiency with disease progression (p<0.001). [Table 5]

Table 5: Comparison of glycemic profile according to duration of diabetes

Duration	FBS (mg/dL)	PPBS (mg/dL)	HbA1c (%)	Serum Magnesium (mg/dL)	p value
<5 years (n=58)	118.5±20.1	176.3±28.6	6.84±0.62	2.01±0.24	<0.001*
5–10 years (n=72)	149.8±28.5	218.6±37.1	7.84±0.91	1.78±0.27	
>10 years (n=50)	182.4±31.8	269.5±45.6	9.18±1.14	1.49±0.23	

One-way ANOVA used for comparison among duration groups; *Significant (p<0.05)

FBG: Fasting Blood Glucose; PPBG: Postprandial Blood Glucose; HbA1c: Glycated Hemoglobin.

Patients with hypomagnesemia had significantly higher serum creatinine and blood urea levels, along with lower eGFR values, indicating an association between reduced serum magnesium concentrations and impaired renal function (p<0.05). [Table 6]

Table 6: Comparison of renal function according to serum magnesium status

Parameter	Hypomagnesemia	Normal Magnesium	p value
Serum Creatinine (mg/dL)	1.18 ± 0.29	0.97 ± 0.21	<0.001*
Blood Urea (mg/dL)	39.7 ± 10.2	33.8 ± 8.4	0.002*
eGFR (mL/min/1.73m²)	78.5 ± 16.2	89.3 ± 15.8	<0.001*

Independent Student's t-test. *Significant (p<0.05)

eGFR: Estimated Glomerular Filtration Rate.

Discussion

The majority of participants were in the 51–60 years age bracket (63; 35.0%), with the next largest group being those over 60 years (51; 28.33%), suggesting a higher prevalence of diabetes among middle-aged and older adults. Hypomagnesemia was identified in 66 (36.67%) patients, while 111 (61.67%) had normal serum magnesium levels and only 3 (1.66%) had hypermagnesemia. The significant occurrence of hypomagnesemia found in this study underscores magnesium shortage as a prevalent metabolic irregularity in individuals with type 2 diabetes mellitus.

A significant inverse association was observed between serum magnesium concentration and glycemic status. When compared to patients with HbA1c <7% (p<0.001), those with poor glycemic control (HbA1c ≥7%) showed considerably higher fasting blood glucose, postprandial blood glucose, and HbA1c levels together with significantly lower serum magnesium concentrations. Furthermore, hypomagnesemia was considerably more frequent among patients with poor glycemic control, indicating that declining serum magnesium levels parallel worsening glucose regulation. These findings suggest that magnesium deficiency may contribute to impaired insulin action, reduced insulin sensitivity, and progressive deterioration of glycemic control.

The results of this investigation agreed with those published by Ramadass S. et al,[13] who demonstrated that serum magnesium levels progressively declined with increasing HbA1c values and longer duration of type 2 diabetes, concluding that hypomagnesemia is closely associated with poor glycemic control and disease progression. Similar observations were reported by Luo B et al,[14] who found significant negative correlations between serum magnesium and fasting plasma glucose, HbA1c, and insulin resistance, suggesting that serum magnesium is an independent predictor of glycemic control. Likewise, Al-Daghri NM et al,[15] reported significantly lower magnesium concentrations among patients with type 2 diabetes compared with prediabetic and healthy individuals, with strong inverse correlations between magnesium levels and both fasting glucose and HbA1c, supporting the diagnostic value of serum magnesium in identifying poor metabolic control.

The length of the disease significantly affected serum magnesium levels. Individuals with diabetes for over a decade had considerably elevated fasting blood glucose, postprandial glucose, and HbA1c levels, alongside notably reduced serum magnesium concentrations compared to individuals with a shorter duration of the disease (p<0.001). These findings indicate progressive magnesium depletion with chronic diabetes, probably resulting from persistent hyperglycaemia, osmotic diuresis, and increased urinary magnesium excretion.

Comparable findings have been reported by Paladiya R et al,[16] who observed significantly lower serum magnesium concentrations and a higher prevalence of hypomagnesemia among patients with diabetes duration exceeding 10 years, recommending routine electrolyte screening in diabetic patients. Similarly, Hamarshih M et al,[17] demonstrated that patients with long-standing diabetes and HbA1c ≥8% had a significantly higher prevalence of hypomagnesemia, emphasizing its association with poor glycemic control and diabetic complications. Alswat K et al,[18] also reported that nearly one-third of patients with type 2 diabetes had hypomagnesemia, which was associated with prolonged disease duration, worse glycemic control, and adverse metabolic outcomes.

Renal function analysis further demonstrated that hypomagnesemia was associated with significantly higher serum creatinine and blood urea levels together with lower estimated glomerular filtration rate (eGFR), indicating early renal impairment in patients with magnesium deficiency. These findings suggest that declining magnesium status may reflect both worsening metabolic control and the onset of diabetic nephropathy.

These observations are supported by Pokharel DR et al,[19] who reported that hypomagnesemia was inversely associated with HbA1c, serum creatinine, and eGFR, identifying poor glycemic control and reduced renal function as significant predictors of low serum magnesium. Similarly, Joy SS et al,[20] demonstrated that reduced serum magnesium was significantly associated with diabetic nephropathy, retinopathy, neuropathy, and

macrovascular complications, with magnesium levels declining further as the number of diabetic complications increased. Santos CF et al,[21] additionally indicated that a shortage in magnesium heightened the probability of raised HbA1c by almost six times and underscored that insufficient magnesium levels substantially affect glycemic regulation in individuals with type 2 diabetes mellitus.

The noted negative correlation between serum magnesium levels and glycemic parameters could be elucidated by magnesium's involvement in insulin receptor activation, subsequent signaling pathways, and the transport of glucose into cells. Magnesium deficiency may impair insulin sensitivity, increase oxidative stress, and promote chronic low-grade inflammation, thereby contributing to poor metabolic control.

The research possesses specific constraints. The cross-sectional design prevents the determination of a causal link between hypomagnesemia and inadequate glycemic regulation. Magnesium consumption, urine magnesium output, and serum insulin concentrations were not evaluated. The research was performed at a solitary tertiary care facility, perhaps restricting the applicability of the results.

Conclusion

Serum magnesium levels showed a significant inverse association with glycemic control in patients with type 2 diabetes mellitus. Hypomagnesemia was prevalent and correlated with increased fasting and postprandial glucose levels, heightened HbA1c, prolonged diabetes duration, and compromised renal function. Individuals exhibiting inadequate glycemic regulation showed markedly reduced serum magnesium levels compared to those with sufficient glycemic control. These findings indicate that serum magnesium may serve as a simple, inexpensive, and clinically useful biochemical marker for identifying patients at risk of poor metabolic control and diabetes-related complications. Routine monitoring of serum magnesium may facilitate early intervention and improve long-term disease management.

Additional prospective multicenter investigations with expanded sample sizes are advised to ascertain the causal link between magnesium deficiency and glycemic regulation, as well as to assess the possible advantages of magnesium supplementation in enhancing metabolic results in individuals with type 2 diabetes mellitus.

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