

Evaluation of Hypomagnesemia and Its Relationship with Glycemic Control in Patients with Type 2 Diabetes Mellitus

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Abstract:

Background: Magnesium, the second most abundant intracellular cation, plays a critical role in numerous enzymatic reactions, including glucose metabolism, insulin secretion, and insulin signaling. In recent years, hypomagnesemia has been increasingly reported among patients with Type 2 Diabetes Mellitus (T2DM), with emerging evidence suggesting a strong link between magnesium deficiency and poor glycemic control. Magnesium acts as a cofactor in carbohydrate metabolism, and its deficiency may exacerbate insulin resistance and impair glucose tolerance. Despite this, serum magnesium levels are often overlooked in routine diabetes care. This study was conducted to evaluate serum magnesium levels in T2DM patients and examine its correlation with glycemic status.

Objectives:

- To estimate serum magnesium levels in adult patients with Type 2 Diabetes Mellitus.
- To assess the correlation between serum magnesium levels and glycemic indicators such as fasting plasma glucose, postprandial blood glucose, and HbA1c.

Materials and Methods: This cross-sectional observational study was conducted at Shri Ramkrishna Institute of Medical Sciences and Sanaka Hospital, Durgapur, over a duration of 12 months. A total of 120 T2DM patients aged 30–65 years were enrolled after fulfilling inclusion criteria. Venous blood samples were collected after an overnight fast and analyzed for serum magnesium using the colorimetric calmagite method. Glycemic parameters including fasting blood glucose (FPG), postprandial glucose (PPG), and HbA1c were also measured. Statistical correlation analysis was performed using Pearson's correlation coefficient.

Results: The mean serum magnesium level among the study population was 1.62 ± 0.18 mg/dL. Hypomagnesemia (serum Mg < 1.7 mg/dL) was observed in 67.5% of patients. A significant inverse correlation was noted between serum magnesium and both HbA1c ($r = -0.446$, $p < 0.001$) and postprandial blood glucose ($r = -0.389$, $p = 0.002$), while a moderate negative correlation existed with fasting plasma glucose ($r = -0.298$, $p = 0.005$). Patients with lower magnesium levels consistently showed higher glycemic indices, suggesting a contributory role of magnesium deficiency in glycemic dysregulation.

Conclusion: This study demonstrates a high prevalence of hypomagnesemia in T2DM patients and a significant inverse relationship between serum magnesium levels and glycemic control. The findings highlight the potential value of including serum magnesium assessment in the routine biochemical evaluation of diabetic individuals to enhance metabolic management and possibly reduce the burden of complications.

Keywords: Serum Magnesium, Hypomagnesemia, Type 2 Diabetes Mellitus, Glycemic Status, HbA1c, Insulin Resistance.

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Introduction

Type 2 diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia due to insulin resistance and progressive β -cell dysfunction. The global burden of type 2 diabetes is increasing at an alarming rate, particularly in developing countries like India. Uncontrolled diabetes leads to a wide spectrum of microvascular and macrovascular complications, contributing

significantly to morbidity, mortality, and economic burden. Despite advancements in therapeutic options, glycemic control remains suboptimal in a significant proportion of patients, indicating the need to explore additional modifiable factors influencing glucose metabolism [1,2].

Magnesium is the second most abundant intracellular cation and serves as a cofactor for over 300 enzymatic reactions, many of which are involved in glucose homeostasis. It plays a crucial role in insulin secretion, insulin receptor function, and glucose uptake at the cellular level [3]. Magnesium deficiency has been increasingly recognized as a common biochemical abnormality in individuals with type 2 diabetes mellitus. This deficiency may result from increased urinary loss due to osmotic diuresis, impaired intestinal absorption, dietary insufficiency, or insulin resistance itself, which alters renal magnesium handling [4].

Several clinical studies have demonstrated that hypomagnesemia is associated with poor glycemic control, increased insulin resistance, and a higher risk of diabetic complications. Low serum magnesium levels have also been linked with higher levels of HbA1c, suggesting that magnesium deficiency may directly contribute to inadequate metabolic control in diabetic patients [5]. Furthermore, magnesium plays an essential role in cardiovascular stability, endothelial function, and oxidative stress regulation, all of which are of particular concern in the diabetic population [6].

Despite the well-established physiological significance of magnesium, it is not routinely measured in diabetic patients during clinical follow-up. This often leads to an underdiagnosis of hypomagnesemia and missed opportunities for timely intervention. Considering the affordability, accessibility, and utility of serum magnesium testing, it holds potential as a valuable adjunct in the metabolic profiling of diabetic patients [7].

In the Indian clinical context, limited data exist regarding the prevalence of hypomagnesemia and its association with glycemic parameters such as fasting glucose, postprandial glucose, and HbA1c in patients with type 2 diabetes. Therefore, the present study was undertaken to estimate serum magnesium levels in patients with type 2 diabetes mellitus and to assess their correlation with markers of glycemic status. The study aims to re-establish the importance of serum magnesium as an often overlooked but clinically significant parameter in diabetes care.

Objectives

The present study was designed to evaluate the relationship between serum magnesium levels and glycemic status in patients diagnosed with type 2 diabetes mellitus. The objective was to explore whether hypomagnesemia is associated with poor glycemic control and to determine the utility of serum magnesium as a supporting biochemical indicator in routine diabetes management.

Primary objectives:

1. To estimate serum magnesium levels in adult patients with type 2 diabetes mellitus.
2. To assess glycemic parameters including fasting plasma glucose, postprandial plasma glucose, and HbA1c in the study population.

Secondary objective:

1. To determine the correlation between serum magnesium levels and glycemic indices (fasting glucose, postprandial glucose, and HbA1c), thereby evaluating the role of hypomagnesemia in glycemic dysregulation.

Materials and Methods

Study design and setting: This was a hospital-based, observational cross-sectional study conducted in the Department of Biochemistry at Shri Ramkrishna Institute of Medical Sciences and Sanaka Hospital, Durgapur, West Bengal, India. The study was carried out in association with the Department of General Medicine, from where eligible patients were referred for biochemical evaluation.

Study duration: The study was conducted over a period of 12 months

Study population: Adult patients diagnosed with type 2 diabetes mellitus attending the outpatient and inpatient departments during the study period were considered for inclusion in the study.

Inclusion criteria:

- Patients aged between 30 and 65 years.
- Diagnosed with type 2 diabetes mellitus for at least one year.
- Willing to provide informed written consent for participation.

Exclusion criteria:

- Patients on magnesium supplementation or diuretics.
- Individuals with chronic kidney disease, hepatic dysfunction, malabsorption syndromes, or acute infections.
- Pregnant and lactating women.
- Patients with type 1 diabetes mellitus or secondary causes of diabetes.

Sample size: A total of 120 patients meeting the inclusion criteria were enrolled in the study using a purposive sampling technique.

Data collection procedure: After obtaining informed consent, relevant clinical history including age, gender, duration of diabetes, medication history, and presence of complications was recorded. Venous blood samples were collected from each patient after an overnight fast. Two aliquots were processed—one for the estimation of serum magnesium, and the other for measurement of fasting plasma glucose and HbA1c. Postprandial

blood samples were collected two hours after the patient's regular breakfast.

Biochemical parameters and assay methods:

Serum magnesium levels were estimated using the colorimetric calmagite method on a semi-automated analyzer. Fasting and postprandial plasma glucose were measured using the glucose oxidase-peroxidase (GOD-POD) method. Glycated hemoglobin (HbA1c) was analyzed using high-performance liquid chromatography (HPLC), standardized according to the National Glycohemoglobin Standardization Program (NGSP) guidelines.

Statistical analysis: Data were compiled using Microsoft Excel and analyzed with SPSS version 26.0. Continuous variables were expressed as mean \pm standard deviation, while categorical variables were summarized as frequencies and percentages.

Pearson's correlation coefficient was used to evaluate the association between serum magnesium and glycemic parameters. A p-value less than 0.05 was considered statistically significant.

Results

A total of 120 patients with type 2 diabetes mellitus were included in the study. Demographic and clinical variables including age, sex, duration of diabetes, and medication history were recorded. Serum magnesium levels and glycemic parameters such as fasting plasma glucose, postprandial plasma glucose, and HbA1c were measured and analyzed. The majority of the study population had subnormal magnesium levels, and significant correlations were observed between serum magnesium and various glycemic indices.

Table 1: Age-wise distribution of study participants

Age group (years)	Frequency	Percentage (%)
30-40	18	15.0
41-50	36	30.0
51-60	52	43.3
>60	14	11.7

Table 2: Gender distribution among study population

Gender	Frequency	Percentage (%)
Male	66	55.0
Female	54	45.0

Table 3: Duration of diabetes in study subjects

Duration of diabetes (years)	Frequency	Percentage (%)
<5	22	18.3
5-10	64	53.3
>10	34	28.4

Table 4: Distribution of serum magnesium levels

Serum magnesium (mg/dL)	Frequency	Percentage (%)
<1.7	81	67.5
\geq 1.7	39	32.5

Table 5: Distribution of HbA1c levels in the study population

HbA1c (%)	Frequency	Percentage (%)
6.5-7.5	18	15.0
7.6-9.0	54	45.0
>9.0	48	40.0

Table 6: Distribution of fasting plasma glucose levels

Fasting plasma glucose (mg/dL)	Frequency	Percentage (%)
<126	22	18.3
126-140	28	23.3
>140	70	58.4

Table 7: Distribution of postprandial plasma glucose levels

Postprandial glucose (mg/dL)	Frequency	Percentage (%)
<180	21	17.5
180–200	32	26.7
>200	67	55.8

Table 8: Mean and standard deviation of biochemical parameters

Parameter	Mean \pm SD
Serum magnesium (mg/dL)	1.62 \pm 0.18
Fasting plasma glucose (mg/dL)	148.6 \pm 28.4
Postprandial glucose (mg/dL)	216.7 \pm 35.2
HbA1c (%)	8.4 \pm 1.2

Table 9: Correlation of serum magnesium with fasting and postprandial glucose

Parameter	Correlation coefficient (r)	p-value
Magnesium vs Fasting glucose	-0.298	0.005
Magnesium vs Postprandial glucose	-0.389	0.002

Table 10: Correlation of serum magnesium with HbA1c

Parameter	Correlation coefficient (r)	p-value
Magnesium vs HbA1c	-0.446	<0.001

Table 1 showed that the highest proportion of patients were in the 51–60 years age group. Table 2 reflected a mild male predominance among the diabetic population. Table 3 revealed that over half the study participants had diabetes duration between 5 and 10 years. Table 4 demonstrated that 67.5% of patients had serum magnesium levels below 1.7 mg/dL, indicating a high prevalence of hypomagnesemia. Table 5 showed that most patients had HbA1c levels exceeding 8%, suggesting poor glycemic control. Table 6 and Table 7 presented elevated fasting and postprandial glucose levels respectively, further supporting inadequate metabolic control. Table 8 provided the mean values for all major biochemical parameters, highlighting elevated glucose and subnormal magnesium levels. Table 9 showed a statistically significant inverse correlation between serum magnesium and both fasting and postprandial glucose, while Table 10 confirmed a stronger negative correlation between magnesium and HbA1c, reinforcing the association between hypomagnesemia and worsening glycemic status.

Discussion

Type 2 diabetes mellitus is a complex disorder marked by persistent hyperglycemia due to insulin resistance and progressive β -cell dysfunction. While the primary focus in diabetic management has traditionally centered around glucose control and insulin dynamics, recent attention has turned toward the role of micronutrients in modulating metabolic pathways [8]. Among these, magnesium has emerged as a critical element in glucose homeostasis, insulin signaling, and vascular function. This study sought to evaluate the serum magnesium status of type 2 diabetic patients and

explore its relationship with glycemic indicators such as fasting and postprandial plasma glucose and HbA1c [9].

The study found a notably high prevalence of hypomagnesemia, with over two-thirds of the patients exhibiting serum magnesium levels below the normal reference range. This observation is consistent with findings from earlier research conducted which reported hypomagnesemia in 30–65% of diabetic individuals [10]. The underlying mechanisms are multifactorial—ranging from increased renal loss of magnesium due to hyperglycemia-induced osmotic diuresis, to reduced intestinal absorption and altered cellular magnesium transport in the setting of insulin resistance [11].

Glycemic control, as evaluated by fasting glucose, postprandial glucose, and HbA1c, was suboptimal in the majority of the study subjects. The statistical analysis revealed a significant inverse correlation between serum magnesium and all three glycemic indicators, with the strongest negative correlation observed between serum magnesium and HbA1c [12]. This suggests that individuals with lower magnesium levels tend to have poorer long-term glycemic control. These findings are supported by the previous researchers who proposed that magnesium plays a role in insulin receptor function and post-receptor signaling. A deficiency in magnesium may impair insulin's ability to facilitate glucose transport into cells, thereby contributing to hyperglycemia and insulin resistance [13].

The stronger association between serum magnesium and postprandial glucose, as compared to fasting glucose, may be explained by the role of magnesium in modulating the acute phase of insulin release and glucose uptake following a meal [14]. Postprandial

hyperglycemia is often an early indicator of impaired glucose tolerance and a major contributor to elevated HbA1c levels. The present study's findings reinforce the potential impact of magnesium deficiency not only on fasting glucose homeostasis but also on dynamic glucose handling after food intake [15].

Another important implication of this study is that serum magnesium testing remains largely neglected in routine diabetic evaluation, despite its affordability and relevance [16]. The absence of overt clinical symptoms in mild to moderate hypomagnesemia may lead to underdiagnosis and missed intervention opportunities. Considering the role of magnesium in cardiac conduction, neuromuscular function, and endothelial integrity, its deficiency could also exacerbate the risk of diabetes-related complications such as arrhythmias, neuropathy, and nephropathy [17,18].

This study, in identifying a high burden of magnesium deficiency and its clear link to poor glycemic control, adds to the growing body of evidence supporting the inclusion of serum magnesium in routine biochemical profiles of diabetic patients. Early identification and correction of hypomagnesemia may contribute not only to improved glycemic outcomes but also to broader metabolic stability and complication prevention.

Limitations of the study include its cross-sectional nature, which restricts causal inference. The sample size, though adequate for preliminary conclusions, warrants larger, multicentric validation. Furthermore, dietary intake of magnesium and renal magnesium excretion were not assessed, which could have provided a more comprehensive understanding of magnesium status in this population.

Despite these limitations, the study emphasizes the need to look beyond glucose-centric parameters in diabetes and recognize the broader nutritional and metabolic imbalances that influence disease progression and therapeutic outcomes.

Conclusion

This study establishes a significant inverse relationship between serum magnesium levels and glycemic status in patients with type 2 diabetes mellitus. The high prevalence of hypomagnesemia observed reinforces the notion that magnesium deficiency is a common but underrecognized metabolic abnormality in diabetics. Lower magnesium levels were consistently associated with elevated fasting glucose, postprandial glucose, and HbA1c, indicating that inadequate magnesium status may contribute to poor glycemic control. These findings highlight the potential role of serum magnesium estimation as a valuable adjunct in the routine biochemical assessment of diabetic patients.

Early detection and correction of hypomagnesemia could offer a simple, low-cost strategy to enhance glycemic management and reduce the risk of diabetes-related complications.

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