MedNova: International Journal of Medical Research
ISSN: XXXX
DOI: XXXX
Volume 01 Issue 01 September 2026

Association Between Serum Magnesium Levels and Glycaemic Control in Patients With Type 2 Diabetes Mellitus: A Hospital-Based Cross-Sectional Study

Chitamandeep Kaur[1], Sahil Kadian[2], Dr Rajat Gupta[3]

[1,2]MBBS Student, World College of Medical Sciences and Research, Jhajjar, Haryana, India.
[3]Assistant Professor, Department of Community Medicine, World College of Medical Sciences and Research, Jhajjar, Haryana, India.

***Corresponding Author:** Chitamandeep Kaur & Email: chitmandeepkaur@gmail.com*

How to Cite: Chitamandeep Kaur, Sahil Kadian, Dr Rajat Gupta. Association Between Serum Magnesium Levels and Glycaemic Control in Patients With Type 2 Diabetes Mellitus: A Hospital-Based Cross-Sectional Study. MedNova: International Journal of Medical Research, 01(01), 01-12, Doi

Received Date: **04/07/2026** | Revised Date: **16/08/2026** | Accepted Date: **08/09/2026** | Published Date: **21/09/2026**

ABSTRACT

Background: Magnesium is an essential mineral involved in insulin signalling, glucose transport, and carbohydrate metabolism. Altered magnesium status has been reported in patients with type 2 diabetes mellitus (T2DM), but the strength of its association with glycaemic control varies across populations. Evidence from Indian tertiary-care populations remains limited and heterogeneous.

Objective: To assess the association between serum magnesium concentration and glycaemic control among patients with T2DM attending a tertiary care hospital in North India.

Methods: This hospital-based cross-sectional study included 245 adult patients with established T2DM and was conducted from January 2026 to June 2026. Demographic and clinical characteristics were recorded, and venous blood samples were analyzed for serum magnesium, fasting plasma glucose (FPG), and glycated haemoglobin (HbA1c). Serum magnesium was assessed as a continuous variable and categorized using a predefined threshold of <1.70 mg/dL. Glycaemic control was assessed primarily using HbA1c, with FPG considered a secondary measure. Continuous and categorical variables were summarized using appropriate descriptive statistics. Correlation between serum magnesium and glycaemic indices was assessed using an appropriate correlation coefficient according to data distribution.

Results: The mean age of participants was 52.18 ± 10.35 years, and 138 (56.3%) were male. The mean duration of diabetes was 8.22 ± 4.71 years. Mean serum magnesium, FPG, and HbA1c were 1.92 ± 0.17 mg/dL, 135.29 ± 43.54 mg/dL, and 7.92 ± 0.81%, respectively. Low serum magnesium (<1.70 mg/dL) was observed in 29 (11.8%) participants. Mean HbA1c was higher among participants with low magnesium than among those with magnesium ≥1.70 mg/dL (8.43 ± 0.87% vs 7.85 ± 0.78%). Serum magnesium demonstrated an inverse correlation with HbA1c (r = −0.41) and FPG (r = −0.28).

Conclusion: Lower serum magnesium concentrations were associated with poorer glycaemic indices among patients with T2DM. These findings support an association between magnesium status and glycaemic control but do not establish causality. Prospective and interventional studies are required to clarify the clinical significance of this relationship.

Keyword: Type 2 diabetes mellitus; serum magnesium; hypomagnesemia; glycated haemoglobin; HbA1c; fasting plasma glucose; glycaemic control

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from varying degrees of insulin resistance, impaired insulin secretion, and progressive pancreatic β-cell dysfunction. It represents a major and increasing global health burden because prolonged hyperglycaemia is associated with vascular and metabolic complications affecting the eyes, kidneys, nerves, cardiovascular system, and other organs. The World Health Organization reported that the number of adults living with diabetes increased substantially from 200 million in 1990 to 830 million in 2022, with the burden rising particularly rapidly in low- and middle-income countries.[1] Diabetes therefore represents not only an individual clinical problem but also an important public-health challenge requiring sustained attention to prevention, early detection, and effective long-term metabolic control.

The burden of diabetes is particularly substantial in India. According to the International Diabetes Federation Diabetes Atlas, approximately 90 million adults aged 20–79 years in India were living with diabetes in 2024, corresponding to an age-standardized prevalence of approximately 10.5%. India consequently remains one of the countries with the largest absolute numbers of adults living with diabetes worldwide.[2] In this setting, improving glycaemic control is an important component of diabetes management because sustained exposure to elevated glucose is associated with the development and progression of diabetes-related complications. Glycated haemoglobin (HbA1c) is the principal laboratory measure used to assess longer-term glycaemic status and reflects average glycaemia over approximately the preceding 2–3 months. Current clinical recommendations consider an HbA1c target of <7% appropriate for many nonpregnant adults, although individualized targets are required according to patient characteristics and treatment-related risks.[3]

Although glycaemic control is determined by multiple interacting factors, including insulin resistance, β-cell function, dietary intake, physical activity, obesity, medication adherence, and pharmacological treatment, increasing attention has been directed toward the potential role of micronutrients in glucose metabolism. Magnesium is an essential mineral and intracellular cation that functions as a cofactor in numerous enzymatic reactions involved in energy metabolism and carbohydrate utilization. It also has an important role in insulin secretion and insulin action. At the cellular level, magnesium is involved in insulin-receptor phosphorylation and downstream signalling, and reduced intracellular magnesium availability has been associated with impaired

insulin-mediated glucose uptake and increased insulin resistance.[4,5]

Patients with T2DM may be particularly susceptible to disturbances in magnesium homeostasis. Hyperglycaemia can increase urinary magnesium loss, while inadequate dietary intake and other metabolic or renal factors may contribute to magnesium depletion. Conversely, reduced magnesium availability may adversely influence insulin signalling and glucose utilization, raising the possibility of a bidirectional relationship between magnesium status and abnormal glucose metabolism.[4-6] This proposed relationship has biological plausibility, although serum magnesium represents only the circulating component of magnesium homeostasis and may not completely reflect total-body or intracellular magnesium stores.

Observational studies have reported an association between lower serum magnesium concentrations and poorer glycaemic control, although the magnitude of the relationship and the prevalence of hypomagnesemia have varied substantially between populations. A 2019 cross-sectional study from North India involving 250 adults with T2DM reported hypomagnesemia in 44% of participants using a serum magnesium threshold of ≤1.7 mg/dL and found poorer fasting glucose, postprandial glucose, and HbA1c values among participants with lower magnesium concentrations.[7] An Indian study published in 2012 similarly reported hypomagnesemia in 11.3% of patients with T2DM and found significantly higher mean HbA1c among participants with hypomagnesemia.[8] More recently, a 2024 Indian study reported significant inverse correlations between serum magnesium and both HbA1c and fasting blood glucose, while finding no significant relationship between magnesium concentration and duration of diabetes.[9] These findings suggest that magnesium status may be associated with glycaemic control, but differences in study populations, definitions of hypomagnesemia, disease severity, and laboratory methods make direct comparison between studies difficult.

The broader evidence also demonstrates substantial heterogeneity. A systematic review and meta-analysis of observational studies published in 2024, including 19 studies and 4192 participants with T2DM, estimated a pooled prevalence of hypomagnesemia of approximately 32%, with considerable variation between geographical populations.[10] Evidence from interventional studies has also been mixed. Earlier randomized-trial meta-analysis found an improvement in some measures of insulin sensitivity but did not demonstrate a significant overall effect on HbA1c, whereas a subsequent dose-response meta-analysis reported modest improvements in HbA1c and fasting glucose with magnesium supplementation but concluded that the evidence remained

insufficient to establish routine clinical recommendations.[11,12] These findings underscore the distinction between an observed association between magnesium status and glycaemic control and evidence that magnesium supplementation itself improves diabetes outcomes.

Despite the biological plausibility and accumulating observational evidence, serum magnesium assessment is not routinely incorporated into the evaluation of all patients with T2DM. Furthermore, available Indian studies have produced heterogeneous estimates of hypomagnesemia and varying strengths of association with glycaemic indices. A recent tertiary-care cross-sectional study from India reported an inverse correlation between serum magnesium and HbA1c and fasting glucose, further supporting the potential relevance of magnesium status in patients with T2DM.[13] However, additional data from tertiary-care populations in North India remain valuable, particularly because differences in dietary patterns, disease characteristics, medication exposure, renal function, and laboratory thresholds may influence the observed relationship.

Accordingly, the present study was undertaken to evaluate the relationship between serum magnesium concentration and glycaemic control among patients with T2DM attending a tertiary care hospital in North India. Because the study uses a cross-sectional design, the objective is to determine the presence and strength of association rather than establish a temporal or causal relationship between magnesium status and glycaemic control.

Methodology

This hospital-based cross-sectional study was conducted in the Department of Medicine at a tertiary care hospital in North India from January 2026 to June 2026. A total of 245 adult patients with an established diagnosis of type 2 diabetes mellitus were enrolled by consecutive sampling. Patients with type 1 diabetes mellitus, gestational diabetes, known chronic kidney disease or significant renal dysfunction, acute systemic illness, conditions likely to substantially alter serum magnesium concentrations, current magnesium supplementation, or incomplete clinical or biochemical data were excluded. After obtaining written informed consent, demographic and clinical information including age, sex, duration of diabetes, body mass index, hypertension, dyslipidemia, antidiabetic treatment, and relevant diabetes-related complications was recorded using a structured data collection form. Venous blood samples were collected under standardized laboratory conditions for estimation of serum magnesium, fasting plasma glucose, and glycated haemoglobin (HbA1c) using the routine

standardized methods of the institutional laboratory. Serum magnesium was analyzed as a continuous variable and was additionally categorized as low (<1.70 mg/dL) or non-low (≥1.70 mg/dL) according to the predefined study criterion. Glycaemic control was assessed primarily using HbA1c, with fasting plasma glucose considered a secondary glycaemic measure. Data were entered into a computerized database and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, as appropriate, while categorical variables were summarized as frequencies and percentages. Distributional normality was assessed before selection of statistical tests. Continuous variables were compared using the independent-samples t-test or Mann–Whitney U test, and categorical variables using the chi-square test or Fisher's exact test, as appropriate. The correlation between serum magnesium and HbA1c was assessed using Pearson's correlation coefficient or Spearman's rank correlation according to data distribution. All statistical tests were two-sided, and $p<0.05$ was considered statistically significant. The study was conducted following approval from the Institutional Ethics Committee, with written informed consent obtained from all participants, maintenance of confidentiality, and voluntary participation with the right to withdraw. The study adhered to the ethical principles of the Declaration of Helsinki and applicable institutional requirements.

Results

A total of 245 patients with type 2 diabetes mellitus were included in the study. The demographic and clinical characteristics of the study population are presented in Table 1. The mean age was 52.18 ± 10.35 years, and 138 (56.3%) participants were male. The mean duration of diabetes was 8.22 ± 4.71 years, while the mean body mass index (BMI) was 26.31 ± 2.93 kg/m². Hypertension was present in 120 (49.0%) participants and dyslipidemia in 114 (46.5%). Most participants (180, 73.5%) were receiving oral antidiabetic agents alone, whereas 65 (26.5%) were receiving an insulin-containing regimen.

Table 1. Demographic and clinical characteristics of the study participants

Characteristic	Overall (n=245)
Age, years	52.18 ± 10.35
Male sex, n (%)	138 (56.3)
Female sex, n (%)	107 (43.7)
Duration of diabetes, years	8.22 ± 4.71

BMI, kg/m²	26.31 ± 2.93
Hypertension, n (%)	120 (49.0)
Dyslipidemia, n (%)	114 (46.5)
Oral antidiabetic agents only, n (%)	180 (73.5)
Insulin-containing regimen, n (%)	65 (26.5)

Values are presented as mean ± standard deviation or number (percentage). BMI, body mass index.

The biochemical characteristics of the participants are shown in Table 2. The overall mean serum magnesium concentration was 1.92 ± 0.17 mg/dL. The mean fasting plasma glucose (FPG) was 135.29 ± 43.54 mg/dL, and the mean HbA1c was 7.92 ± 0.81%. Using the predefined study threshold of <1.70 mg/dL, 29 (11.8%) participants were classified as having low serum magnesium, while 216 (88.2%) had serum magnesium concentrations ≥1.70 mg/dL.

Table 2. Biochemical characteristics and serum magnesium status

Variable	**Overall (n=245)**
Serum magnesium, mg/dL	1.92 ± 0.17
FPG, mg/dL	135.29 ± 43.54
HbA1c, %	7.92 ± 0.81
Serum magnesium <1.70 mg/dL, n (%)	29 (11.8)
Serum magnesium ≥1.70 mg/dL, n (%)	216 (88.2)
HbA1c <7%, n (%)	35 (14.3)
HbA1c ≥7%, n (%)	210 (85.7)

Values are presented as mean ± standard deviation or number (percentage). FPG, fasting plasma glucose; HbA1c, glycated haemoglobin.

Comparison according to serum magnesium status is presented in Table 3. Participants with serum magnesium <1.70 mg/dL had a lower mean magnesium concentration of 1.62 ± 0.08 mg/dL compared with 1.96 ± 0.14 mg/dL among those with concentrations ≥1.70 mg/dL. The low-magnesium group also had higher mean FPG and HbA1c concentrations. Mean HbA1c was 8.43 ± 0.87% among participants with serum magnesium <1.70 mg/dL compared with 7.85 ± 0.78% among those with serum magnesium ≥1.70 mg/dL.

Table 3. Comparison of clinical and biochemical characteristics according to serum magnesium status

Variable	Magnesium ≥1.70 mg/dL (n=216)	Magnesium <1.70 mg/dL (n=29)
Age, years	51.68 ± 10.31	55.91 ± 10.09
Male sex, n (%)	123 (56.9)	15 (51.7)
Duration of diabetes, years	8.43 ± 4.75	6.62 ± 4.12
BMI, kg/m²	26.17 ± 2.95	27.31 ± 2.65
Hypertension, n (%)	109 (50.5)	11 (37.9)
Dyslipidemia, n (%)	98 (45.4)	16 (55.2)
Oral antidiabetic agents only, n (%)	162 (75.0)	18 (62.1)
Insulin-containing regimen, n (%)	54 (25.0)	11 (37.9)
Serum magnesium, mg/dL	1.96 ± 0.14	1.62 ± 0.08
FPG, mg/dL	132.54 ± 41.96	155.75 ± 50.09
HbA1c, %	7.85 ± 0.78	8.43 ± 0.87

Values are presented as mean ± standard deviation or number (percentage). BMI, body mass index; FPG, fasting plasma glucose; HbA1c, glycated haemoglobin.

With respect to categorical glycaemic control, 210 (85.7%) participants had HbA1c ≥7%. Among those with serum magnesium <1.70 mg/dL, 27 (93.1%) had HbA1c ≥7%, compared with 183 (84.7%) among those with serum magnesium ≥1.70 mg/dL (Table 4). Thus, although poor glycaemic control was numerically more frequent among participants with lower magnesium concentrations, the principal relationship was better represented by analysis of serum magnesium and HbA1c as continuous variables.

Table 4. Glycaemic control according to serum magnesium status

Glycaemic control	Magnesium ≥1.70 mg/dL (n=216)	Magnesium <1.70 mg/dL (n=29)	Total (n=245)
HbA1c <7%, n (%)	33 (15.3)	2 (6.9)	35 (14.3)
HbA1c ≥7%, n (%)	183 (84.7)	27 (93.1)	210 (85.7)

Values are presented as number (percentage).

Correlation analysis demonstrated an inverse relationship between serum magnesium concentration and glycaemic indices. Serum magnesium was negatively correlated with HbA1c, indicating that lower magnesium concentrations were associated with higher HbA1c values. Serum magnesium also showed an inverse relationship with FPG. The association between serum magnesium and duration of diabetes was weak, and the relationship between magnesium and BMI was similarly weak (Table 5).

Table 5. Correlation of serum magnesium with selected clinical and glycaemic variables

Variable	Correlation with serum magnesium
HbA1c	r = −0.41
FPG	r = −0.28
Age	r = −0.17
Duration of diabetes	r = −0.08
BMI	r = −0.08

r, correlation coefficient; BMI, body mass index; FPG, fasting plasma glucose; HbA1c, glycated haemoglobin.

Diabetes-related complications were also recorded as secondary clinical characteristics. Retinopathy was present in 48 (19.6%) participants, nephropathy in 38 (15.5%), and neuropathy in 59 (24.1%). As these complications were not the primary outcomes of the study, their distribution was considered secondary to the principal analysis of serum magnesium and glycaemic control (Table 6).

Table 6. Diabetes-related complications according to serum magnesium status

Complication	Magnesium ≥1.70 mg/dL (n=216)	Magnesium <1.70 mg/dL (n=29)	Total (n=245)
Retinopathy, n (%)	44 (20.4)	4 (13.8)	48 (19.6)
Nephropathy, n (%)	32 (14.8)	6 (20.7)	38 (15.5)
Neuropathy, n (%)	49 (22.7)	10 (34.5)	59 (24.1)

Values are presented as number (percentage).

Discussion

The present study evaluated the association between serum magnesium concentration and glycaemic control among patients with type 2 diabetes mellitus attending a tertiary care hospital in North India. The principal finding was

an inverse relationship between serum magnesium and glycaemic indices, with lower serum magnesium concentrations being associated with higher HbA1c and fasting plasma glucose levels. Serum magnesium showed a moderate inverse correlation with HbA1c ($r = -0.41$) and a weaker inverse correlation with fasting plasma glucose ($r = -0.28$). Participants with serum magnesium <1.70 mg/dL also had higher mean HbA1c and fasting plasma glucose concentrations than those with higher magnesium concentrations. These findings support an association between magnesium status and glycaemic control, although the cross-sectional design does not permit determination of temporal or causal relationships.

The observed relationship is biologically plausible. Magnesium is involved in several processes relevant to glucose metabolism, including insulin receptor signalling, phosphorylation reactions, glucose transport, and intracellular energy metabolism. Magnesium deficiency may impair insulin-mediated glucose uptake and contribute to insulin resistance, while chronic hyperglycaemia may increase urinary magnesium losses and thereby worsen magnesium depletion.[4,5] Thus, the relationship between magnesium and glycaemic control may be bidirectional. However, serum magnesium represents only the circulating component of magnesium homeostasis and may not fully reflect intracellular or total-body magnesium stores. Consequently, the findings should be interpreted as an association with circulating magnesium concentration rather than definitive evidence of tissue magnesium deficiency.

The inverse correlation between serum magnesium and HbA1c observed in the present study is consistent with findings from previous Indian studies. Kumar et al., in a North Indian cross-sectional study of 250 patients with type 2 diabetes, reported significantly poorer fasting glucose, postprandial glucose, and HbA1c values among participants with lower serum magnesium concentrations.[7] Similarly, an earlier Indian study reported higher HbA1c among patients with hypomagnesemia.[8] A 2024 Indian study also demonstrated significant inverse correlations between serum magnesium and HbA1c as well as fasting blood glucose, supporting the direction of association observed in the present study.[9] The correlation coefficient of -0.41 for HbA1c in the present study represents a moderate inverse association and is therefore compatible with the range reported in recent observational research rather than representing an unusually strong relationship.

The observed inverse correlation between magnesium and fasting plasma glucose ($r = -0.28$) is also consistent with previous findings.

The recent tertiary-care study by Sri et al. reported inverse correlations of similar magnitude between serum magnesium and HbA1c and fasting glucose.[13] However, differences in study populations, sample size, dietary patterns, diabetes duration, medication use, renal function, laboratory methods, and definitions of hypomagnesemia may account for variation in the strength of associations between studies. The prevalence of low serum magnesium in the present cohort was 11.8%, which is lower than the 44% reported by Kumar et al. in North India.[7] Such differences highlight the importance of using a consistent biochemical threshold and standardized laboratory methods when comparing magnesium status across populations.

The categorical analysis showed that poor glycaemic control was more frequent among participants with serum magnesium <1.70 mg/dL, although the continuous correlation analysis provided a more informative assessment of the relationship between magnesium and HbA1c. This distinction is important because dichotomizing a continuous biochemical variable may reduce statistical information and obscure relationships occurring across the full range of measurements. The findings therefore suggest that serum magnesium may have a graded relationship with glycaemic status rather than simply distinguishing patients into normal and deficient categories.

Evidence concerning whether magnesium supplementation improves glycaemic control is less consistent than the observational evidence linking magnesium status with diabetes. A systematic review and meta-analysis of randomized controlled trials reported potential benefits of magnesium supplementation for insulin sensitivity but did not demonstrate a consistent overall improvement in HbA1c.[11] A subsequent dose-response meta-analysis reported modest improvements in glycaemic parameters with magnesium supplementation but emphasized the need for further evidence before routine clinical recommendations can be made.[12] Therefore, the present findings should not be interpreted as evidence that magnesium supplementation will necessarily improve glycaemic control.

The study has several strengths, including assessment of serum magnesium alongside objective measures of glycaemic control, evaluation of magnesium both as a continuous variable and according to a predefined threshold, and inclusion of a relatively substantial number of patients from a tertiary-care setting. Nevertheless, several limitations should be considered. The cross-sectional design prevents assessment of temporality and causality. Serum

magnesium was measured at a single time point and may be influenced by dietary intake, renal handling, medications, and other unmeasured factors. Residual confounding cannot therefore be excluded. In addition, the findings from a single tertiary-care hospital may not be directly generalizable to all patients with type 2 diabetes in India. Prospective studies with repeated magnesium measurements, detailed assessment of dietary and renal factors, and adequately powered intervention trials are warranted to clarify the clinical significance of magnesium status in diabetes.

Conclusion

In this cross-sectional study of patients with type 2 diabetes mellitus, lower serum magnesium concentrations were associated with poorer glycaemic control. Serum magnesium demonstrated an inverse relationship with HbA1c and fasting plasma glucose, indicating that participants with lower circulating magnesium tended to have higher glycaemic indices. The findings are consistent with previous observational evidence suggesting a relationship between magnesium status and glucose metabolism. However, the cross-sectional design precludes establishing temporality or causality, and the observed association should not be interpreted as evidence that magnesium deficiency directly causes poor glycaemic control or that supplementation will necessarily improve diabetes outcomes. Assessment of magnesium status may have potential clinical relevance in selected patients with suboptimal glycaemic control, particularly when other risk factors for magnesium depletion are present. Larger prospective and interventional studies with repeated magnesium measurements and careful assessment of potential confounding factors are warranted to clarify the clinical significance of this association and determine whether correction of magnesium deficiency can improve metabolic outcomes.

Declarations

Funding: None.

Conflict of Interest: None declared.

Ethical Approval: The study was conducted after approval from the Institutional Ethics Committee of the study institution. The approval number and date should be reported as per the official ethics documentation.

Informed Consent: Written informed consent was obtained from all participants before enrolment.

Acknowledgements: None.

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