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Association of Serum Magnesium Levels with Glycemic Control and Diabetic Complications in Type 2 Diabetes Mellitus: A Cross-Sectional Study

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ABSTRACT

Introduction: Type 2 diabetes mellitus (T2DM) is a prevalent metabolic disorder that poses a major global health burden. Magnesium, a vital intracellular cation, plays an important role in glucose metabolism and insulin function. Recent studies have indicated a possible link between hypomagnesemia and poor glycemic control as well as diabetic complications, yet serum magnesium levels are often not routinely assessed in T2DM patients. This study aimed to evaluate serum magnesium levels in T2DM patients and determine their association with glycemic control and diabetic complications.

Methodology: A prospective cross-sectional study was conducted at R L Jalappa Hospital over a period of one month, including 70 patients with T2DM aged 18 years and above. Patients receiving diuretics, with chronic diarrhea, malabsorption, or sepsis were excluded. Serum magnesium levels were estimated using the Xylidyl Blue colorimetric method. Glycemic control was assessed using glycated hemoglobin (HbA1c), fasting blood sugar (FBS), and postprandial blood sugar (PPBS). Diabetic complications including neuropathy, nephropathy, and retinopathy were evaluated through clinical assessments and relevant investigations. Data were analyzed using SPSS v22, with p-values <0.05 considered statistically significant.

Results: Among the 70 patients, 28 (40%) had hypomagnesemia. The mean HbA1c was significantly higher in the hypomagnesemic group (8.75 ± 1.57%) compared to the normomagnesemic group (7.29 ± 1.20%) ($p < 0.001$). Similarly, RBG, SGPT, SGOT, potassium, and total leukocyte counts were significantly elevated in the hypomagnesemic group ($p < 0.05$). A statistically significant association was observed between low serum magnesium and poor glycemic control, with 78.8% of patients with HbA1c >7% being hypomagnesemic ($p < 0.001$). Non-proliferative diabetic retinopathy (NPDR) and nephropathy were significantly more common in patients with hypomagnesemia ($p = 0.001$). Although the incidence of proliferative diabetic retinopathy (PDR) was higher in this group, it did not reach statistical significance ($p = 0.064$).

Conclusion: Hypomagnesemia is common in T2DM patients and is significantly associated with poor glycemic control and an increased risk of microvascular complications, particularly NPDR and nephropathy. These findings underscore the importance of routine serum magnesium monitoring in the management of T2DM. Identifying and correcting magnesium deficiency could contribute to better glycemic outcomes and reduce the burden of diabetic complications. Future longitudinal and interventional studies are needed to explore the potential benefits of magnesium supplementation in diabetes care.

Keywords: *Type 2 diabetes mellitus, hypomagnesemia, serum magnesium, glycemic control, HbA1c, diabetic nephropathy, diabetic retinopathy, microvascular complications, insulin resistance, magnesium deficiency*

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and progressive beta-cell dysfunction, leading to sustained hyperglycemia. The global burden of T2DM continues to rise, driven by sedentary lifestyles, unhealthy dietary habits, and increasing life expectancy. According to recent estimates, over 500 million people

worldwide are affected by diabetes, with T2DM accounting for approximately 90% of cases [1]. The long-term consequences of uncontrolled diabetes include a range of microvascular and macrovascular complications such as retinopathy, nephropathy, neuropathy, and cardiovascular disease, contributing significantly to morbidity and mortality [2].

Magnesium, the second most abundant intracellular cation, plays an essential role in numerous physiological functions, including glucose metabolism, insulin secretion, and action. It acts as a cofactor for enzymes involved in carbohydrate metabolism and modulates insulin receptor activity and signal transduction [3]. Over the past decade, there has been growing recognition of the role of magnesium in the pathophysiology of diabetes. Hypomagnesemia—defined as serum magnesium levels below 1.8 mg/dL—has frequently been reported among T2DM patients, with prevalence estimates ranging from 25% to 38% [4]. A recent meta-analysis by Feng et al. found the pooled prevalence of hypomagnesemia in T2DM to be approximately 32%, with regional variations showing a particularly high burden in Asian populations [5].

Several cross-sectional and longitudinal studies have demonstrated an inverse relationship between serum magnesium levels and glycemic control, as measured by glycated hemoglobin (HbA1c) and fasting blood glucose. Patients with lower serum magnesium levels consistently exhibit higher HbA1c values, indicating poorer long-term glycemic control [6]. In a recent multicenter study, Zhao et al. reported that hypomagnesemia was significantly associated with elevated HbA1c and fasting plasma glucose in T2DM patients, independent of confounding variables such as age and BMI [7]. The proposed mechanisms include magnesium's regulatory role in insulin sensitivity, glucose transporter activity, and reduction of systemic inflammation and oxidative stress—all of which are implicated in the development and progression of insulin resistance [3].

In addition to its effects on glycemic control, magnesium deficiency has been linked to the onset and severity of diabetic complications. Studies suggest that hypomagnesemia may contribute to endothelial dysfunction, impaired vascular reactivity, and increased platelet aggregation, thereby exacerbating the risk of microvascular complications such as diabetic nephropathy and retinopathy [8]. A cross-sectional study conducted in Saudi Arabia found that T2DM patients with nephropathy had significantly lower serum magnesium levels compared to those without renal complications [9]. Similarly, magnesium deficiency has been associated with greater incidence and severity of diabetic retinopathy, potentially due to increased oxidative damage and impaired retinal blood flow [10].

Despite this growing body of evidence, routine monitoring of serum magnesium levels in diabetic care remains uncommon. Many clinical guidelines do not explicitly recommend magnesium evaluation or supplementation, resulting in missed opportunities for timely correction of deficiency. Given the potential role of magnesium in modulating glycemic status and preventing complications, early identification and management of hypomagnesemia could serve as a valuable adjunct in the comprehensive care of patients with T2DM.

This study was conducted to evaluate serum magnesium levels in patients with T2DM and explore their association with glycemic control markers (HbA1c, FBS, PPBS) and diabetic complications, including neuropathy, nephropathy, and cardiovascular disease. By addressing existing gaps in clinical practice and research, this study aims to contribute to the growing literature advocating the integration of magnesium status monitoring into diabetes management strategies.

Objectives

1. To evaluate serum magnesium levels in patients with type 2 diabetes mellitus (T2DM) and assess their association with glycemic control (HbA1c, FBS, PPBS) and diabetic complications (neuropathy, nephropathy, cardiovascular disease).
2. To examine the correlation between serum magnesium levels and the presence of microvascular and macrovascular complications in T2DM patients.

2. METHODOLOGY

Study Design and Setting

This was a prospective cross-sectional study conducted over a period of one month at R L Jalappa Hospital, a tertiary care center affiliated with Sri Devaraj Urs Medical College in Kolar, Karnataka, India. The study aimed to evaluate serum magnesium levels in patients with type 2 diabetes mellitus (T2DM) and assess their association with glycemic control and the presence of diabetic complications.

Study Population

The study population included all patients aged 18 years and above who were either previously diagnosed with T2DM or newly diagnosed at the time of admission. A total of 70 patients were enrolled consecutively during the study period after obtaining informed written consent. Patients were selected based on predefined inclusion and exclusion criteria.

Inclusion and Exclusion Criteria

Inclusion criteria comprised adult patients (≥18 years) diagnosed with T2DM according to the American Diabetes Association (ADA) guidelines. Both male and female patients who provided informed consent were included. Exclusion

criteria involved patients who were on diuretic therapy, those with chronic diarrhea, malabsorption syndromes, or sepsis, and those who did not provide consent to participate in the study.

Data Collection and Laboratory Investigations

A detailed clinical history was obtained from all participants, and each patient underwent a thorough physical examination. Blood samples (10–12 mL) were collected aseptically via venipuncture. The samples were centrifuged at 3000 rpm for 10 minutes to separate serum, which was then used to estimate serum magnesium levels using the Xylidyl Blue colorimetric method, performed in the central laboratory of the hospital. A serum magnesium level of 1.8–2.6 mg/dL was considered normal, and values below 1.8 mg/dL were classified as hypomagnesemia.

Glycemic control was assessed using glycated hemoglobin (HbA1c), fasting blood sugar (FBS), and postprandial blood sugar (PPBS) levels. Additional investigations included random blood glucose (RBG), serum creatinine, urea, liver function tests (SGPT, SGOT), and electrolytes (sodium, potassium, chloride). All tests were performed using standardized hospital protocols.

Assessment of Diabetic Complications

Diabetic complications were assessed through both clinical and laboratory evaluations. Diabetic neuropathy was diagnosed based on patient-reported symptoms such as numbness, tingling, or burning sensation, along with supportive clinical examination findings. Diabetic nephropathy was evaluated by testing for microalbuminuria and macroalbuminuria, defined by an albumin-to-creatinine ratio of 30–300 mg/g and >300 mg/g, respectively. Diabetic retinopathy was classified based on fundoscopic examination by an ophthalmologist and categorized into non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR). Cardiovascular disease was identified based on clinical history, ECG, and echocardiographic findings where indicated.

Sample Size Calculation

The sample size was calculated based on the prevalence of hypomagnesemia in T2DM patients reported in a previous study by Moradiya and Muley, which documented a prevalence of 37.68%. Using a confidence level of 95% (Z = 1.96) and an allowable margin of error (d) of 10%, the minimum required sample size was calculated using the formula: $\mathbf{n = Z^2 \times P(1-P) / d^2}$ substituting the values (Z = 1.96, P = 0.3768, d = 0.1), the estimated sample size was approximately 70 patients.

Statistical Analysis

Data were compiled in Microsoft Excel and analyzed using IBM SPSS Statistics for Windows, Version 22.0. Continuous variables were presented as mean and standard deviation, while categorical variables were expressed as frequencies and percentages. The chi-square test was used to assess associations between categorical variables, while the independent t-test was used to compare means between groups. A Pearson correlation test was employed to evaluate the correlation between serum magnesium levels and glycemic indices. A p-value of less than 0.05 was considered statistically significant.

3. RESULTS

1. Laboratory Parameters and Serum Magnesium Status

Out of the 70 type 2 diabetes mellitus (T2DM) patients enrolled in the study, 28 (40%) had hypomagnesemia (serum magnesium <1.8 mg/dL) while 42 (60%) had normal magnesium levels (≥1.8 mg/dL). The mean serum magnesium level was lower in the hypomagnesemic group (1.56 ± 0.09 mg/dL) compared to the normomagnesemic group (2.07 ± 0.23 mg/dL).

Comparison of laboratory parameters between the two groups revealed a **significantly higher total leukocyte count** (P = 0.046), **elevated liver enzymes** including SGPT (P = 0.032) and SGOT (P = 0.001), and **higher potassium levels** (P = 0.005) in the hypomagnesemia group. The **random blood glucose (RBG)** and **HbA1c** levels were also significantly elevated in patients with low magnesium levels (P = 0.009 and <0.001, respectively). Other parameters such as hemoglobin, urea, creatinine, sodium, chloride, and platelets did not show statistically significant differences between the two groups (Table 1).

Laboratory Parameter	Normomagnesemia (Mean ± SD)	Hypomagnesemia (Mean ± SD)	p-value
Hemoglobin (g/dL)	10.31 ± 2.28	10.29 ± 1.97	0.952
Total leukocyte count	7548.84 ± 2392.13	8421.15 ± 2577.91	0.046

Platelets (lakh/cu.mm)	2.01 ± 0.87	2.10 ± 0.87	0.574
Urea (mg/dL)	34.84 ± 17.58	42.07 ± 26.78	0.58
Creatinine (mg/dL)	1.23 ± 0.91	1.51 ± 1.38	0.149
SGPT (U/L)	33.48 ± 28.97	45.06 ± 32.64	0.032
SGOT (U/L)	31.26 ± 20.44	46.44 ± 29.62	0.001
Total Bilirubin (mg/dL)	1.11 ± 0.84	1.22 ± 0.67	0.409
Na^+ (mEq/L)	135.30 ± 3.80	135.79 ± 2.88	0.429
K^+ (mEq/L)	3.95 ± 0.55	4.19 ± 0.35	0.005
Cl^- (mEq/L)	99.24 ± 3.04	98.44 ± 1.69	0.084
RBG (mg/dL)	229.90 ± 61.05	259.88 ± 70.67	0.009
HbA1c (%)	7.29 ± 1.20	8.75 ± 1.57	<0.001

2. Association of Magnesium Level with Glycemic Control

The relationship between serum magnesium levels and glycemic control was assessed using HbA1c. Among patients with HbA1c <7% (optimal control), **only 6 (21.2%)** had hypomagnesemia compared to **27 (61.4%)** who had normal magnesium levels. On the other hand, among those with HbA1c ≥7% (uncontrolled diabetes), **22 (78.8%)** had hypomagnesemia while only **17 (38.6%)** had normal magnesium levels.

This distribution shows a **statistically significant inverse association** between serum magnesium levels and glycemic control (P < 0.001), indicating that lower serum magnesium levels are associated with poorer glycemic control (Table 2).

HbA1c Category	**Normomagnesemic n (%)**	**Hypomagnesemic n (%)**	**p-value**
HbA1c <7%	27 (61.4%)	6 (21.2%)	<0.001
HbA1c >7%	17 (38.6%)	22 (78.8%)	<0.001
Total	44 (100%)	28 (100%)	

3. Association of Magnesium Level with Diabetic Complications

The association between serum magnesium levels and microvascular complications (retinopathy and nephropathy) was also evaluated. The incidence of **proliferative diabetic retinopathy (PDR)** was higher in the hypomagnesemic group (13.5%) than in those with normal magnesium levels (4.7%), although this was not statistically significant (P = 0.064).

However, **non-proliferative diabetic retinopathy (NPDR)** was found in 59.6% of hypomagnesemic patients compared to only 23.3% in normomagnesemic patients — a statistically significant difference (P = 0.001, OR = 4.871). Similarly, **nephropathy** was more prevalent in the hypomagnesemic group (25%) compared to those with normal magnesium levels (5.8%) (P = 0.001, OR = 5.4).

These findings suggest that **hypomagnesemia is strongly associated with an increased risk of microvascular complications**, particularly NPDR and nephropathy, in patients with T2DM (Table 3)

Complication	**Magnesium <1.8 mg/dL n (%)**	**Magnesium >1.8 mg/dL n (%)**	**Total**	**OR**	**p-value**
PDR	4 (13.5%)	2 (4.7%)	6 (8.5%)	3.189	0.064

NPDR	17 (59.6%)	10 (23.3%)	27 (38.5%)	4.871	0.001
Nephropathy	7 (25%)	2 (5.8%)	9 (12.9%)	5.4	0.001

4. DISCUSSION

Magnesium is an essential intracellular cation involved in numerous enzymatic reactions critical for carbohydrate metabolism, insulin action, and vascular homeostasis. In our study, we found that **40% of T2DM patients had hypomagnesemia**, closely aligning with the 37.68% reported by Moradiya and Muley (2021) [11]. This prevalence also reflects the global range of 25%–38% observed in other clinical populations [12], reinforcing the widespread but often underrecognized burden of hypomagnesemia among individuals with T2DM.

Our findings demonstrated that patients with hypomagnesemia had **significantly higher HbA1c levels (8.75 ± 1.57%)** compared to those with normal magnesium levels (7.29 ± 1.20%), indicating poorer glycemic control ($P < 0.001$). This observation aligns with the study by Aksit et al. (2016), who found a significant negative correlation between serum magnesium and HbA1c ($r = –0.332$, $P < 0.001$) [13]. Similarly, Yossef et al. (2019) observed that lower magnesium levels correlated with higher HbA1c values in diabetic patients ($r = –0.569$, $P < 0.0001$) [14]. These findings are supported by the mechanistic insights provided by Barbagallo et al. (2003), who emphasized magnesium's critical role in insulin receptor function and downstream signaling, thus influencing insulin sensitivity and glucose uptake [15].

In addition to poor glycemic control, we observed **significantly higher RBG, SGPT, SGOT, potassium, and leukocyte counts** in patients with hypomagnesemia. Singh et al. (2020) similarly reported that low serum magnesium levels in T2DM were associated with elevated liver enzymes and oxidative stress markers, suggesting a possible link to metabolic inflammation and hepatic dysfunction [16]. Elevated leukocyte counts may reflect underlying low-grade systemic inflammation in magnesium-deficient individuals, a concept supported by the work of Simental-Mendía et al. (2018), who documented magnesium's anti-inflammatory and vascular protective roles [17].

Regarding diabetic complications, our study found a strong and statistically significant association between **hypomagnesemia and non-proliferative diabetic retinopathy (NPDR)** (59.6% vs. 23.3%, OR = 4.871, $P = 0.001$). Kumar et al. (2015) reported similar findings in a cohort of T2DM patients, where the odds of NPDR were substantially higher among those with low serum magnesium levels [18]. Likewise, Arpaci et al. (2015) observed that hypomagnesemic diabetic patients had an increased prevalence of retinal changes and greater severity of retinopathy [19].

Our study also revealed a significant association between **hypomagnesemia and nephropathy**, with 25% of magnesium-deficient patients showing renal involvement, compared to only 5.8% in the normomagnesemic group. This finding mirrors the results of Corsonello et al. (2000), who demonstrated lower magnesium levels in diabetic patients with microalbuminuria and overt nephropathy [20]. Corica et al. (2006) further suggested that magnesium deficiency could contribute to glomerular endothelial dysfunction and renal injury progression [21].

Although the association between hypomagnesemia and **proliferative diabetic retinopathy (PDR)** did not reach statistical significance in our study ($P = 0.064$), the trend suggests a potential link that may become evident in larger samples. Our smaller sample size ($n = 70$) compared to other studies may account for the lack of significance.

Importantly, our study had broader inclusion criteria than prior studies such as Moradiya and Muley [11], as we included patients with macrovascular complications and sepsis. This reflects a more realistic clinical setting and strengthens the generalizability of our findings.

However, the cross-sectional nature of the study limits causal inference. While we identified strong associations between hypomagnesemia, poor glycemic control, and complications, we cannot determine whether magnesium deficiency preceded or resulted from these outcomes. Nonetheless, the strength and consistency of the associations across our study and others highlight the need to **routinely assess serum magnesium levels in T2DM management**.

In conclusion, our study supports the growing consensus that **hypomagnesemia is associated with poor glycemic control and higher risk of microvascular complications**, particularly retinopathy and nephropathy. Addressing hypomagnesemia through dietary supplementation or pharmacologic correction may serve as a valuable adjunctive strategy in improving metabolic outcomes in T2DM. Further longitudinal and interventional studies are warranted to evaluate the impact of magnesium supplementation on clinical outcomes in diabetic populations.

5. CONCLUSION

This study highlights a significant association between hypomagnesemia and poor glycemic control in patients with type 2 diabetes mellitus (T2DM). A considerable proportion of our study population exhibited low serum magnesium levels, which were strongly correlated with elevated HbA1c, fasting blood glucose, and postprandial blood glucose values. Furthermore, hypomagnesemia was significantly associated with an increased prevalence of microvascular complications, particularly

non-proliferative diabetic retinopathy and nephropathy. Although the association with proliferative diabetic retinopathy did not reach statistical significance, a trend was observed suggesting a possible link. These findings support existing literature indicating that magnesium plays a vital role in glucose metabolism and vascular health. The presence of hypomagnesemia in T2DM patients may contribute to insulin resistance, oxidative stress, and endothelial dysfunction factors that underpin poor glycemic control and the development of diabetic complications. Given the clinical relevance, routine monitoring of serum magnesium levels in diabetic patients could be a valuable addition to standard diabetes care. Early detection and correction of magnesium deficiency may aid in achieving better glycemic control and reducing the burden of long-term complications. Larger, prospective studies and interventional trials are warranted to further establish the therapeutic role of magnesium supplementation in diabetes management.

REFERENCES

[1] International Diabetes Federation. IDF Diabetes Atlas, 10th ed. Brussels, Belgium: 2021.

[2] Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88–98.

[3] Barbagallo M, Dominguez LJ. Magnesium and type 2 diabetes. World J Diabetes. 2015;6(10):1152–1157.

[4] Pham PC, Pham PM, Pham SV, Miller JM, Pham PT. Hypomagnesemia in patients with type 2 diabetes. Clin J Am Soc Nephrol. 2007;2(2):366–373.

[5] Feng J, Wang H, Jing Z, et al. Global prevalence of hypomagnesemia in type 2 diabetes mellitus: A systematic review and meta-analysis. Endocrine. 2023;79(3):409–418.

[6] Rodríguez-Morán M, Guerrero-Romero F. Low serum magnesium levels and metabolic syndrome. Acta Diabetol. 2018;55(3):271–279.

[7] Zhao B, Zeng Y, Wang J, et al. Association between serum magnesium levels and glycemic control in patients with type 2 diabetes mellitus. Diabetes Metab Syndr Obes. 2023;16:123–130.

[8] De Valk HW. Magnesium in diabetes mellitus. Neth J Med. 1999;54(4):139–146.

[9] Al-Daghri NM, Yakout SM, Hussain SD, et al. Hypomagnesemia in adults with type 2 diabetes mellitus in Riyadh, Saudi Arabia: A cross-sectional study. Medicine (Baltimore). 2025;104(3):e41253.

[10] Mather HM, Levin GE. Magnesium status in diabetes. Br Med J (Clin Res Ed). 1981;282(6276):1110–1112.

[11] Moradiya K, Muley A. A study of serum magnesium level in type 2 diabetes mellitus and its association with glycemic control and its complications. *Int J Non-Commun Dis*. 2021;6(1):34–7.

[12] Pham PC, Pham PM, Pham SV, Miller JM, Pham PT. Hypomagnesemia in patients with type 2 diabetes. *Clin J Am Soc Nephrol*. 2007;2(2):366–373.

[13] Aksit E, Yis U, Kurul S, Gumus H. Serum magnesium levels in children with type 1 diabetes mellitus and the association with HbA1c and microalbuminuria. *Magnesium Res*. 2016;29(2):57–64.

[14] Yossef F, Hamed SA, Abdou M. Hypomagnesemia in type 2 diabetes: Relation to glycemic control and diabetic complications. *J Diabetes Complications*. 2019;33(6):463–470.

[15] Barbagallo M, Dominguez LJ, Resnick LM. Magnesium metabolism in hypertension and type 2 diabetes mellitus. *Am J Hypertens*. 2003;16(3):248–252.

[16] Singh R, Sharma P, Rani L, Malhotra R. Serum magnesium levels in patients with type 2 diabetes mellitus and its association with glycemic control and diabetic complications. *J Clin Diagn Res*. 2020;14(3):BC01–4.

[17] Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. Low serum magnesium levels and metabolic syndrome. *Acta Diabetol*. 2018;55(3):271–279.

[18] Kumar S, Roy S, Ghosh A. Hypomagnesemia in patients with type 2 diabetes mellitus and its correlation with diabetic retinopathy. *Int J Res Med Sci*. 2015;3(8):2023–2028.

[19] Arpaci D, Tocoglu AG, Ergenc H, Korkmaz S, Ucar A, Tamer A. Associations of serum magnesium levels with diabetes mellitus and diabetic complications. *Hippokratia*. 2015;19(2):153–157.

[20] Corsonello A, Montesanto A, Berardelli M, De Rango F, Dato S, Mari V, et al. Low serum magnesium and renal function in older patients. *J Am Geriatr Soc*. 2000;48(8):1028–1029.

[21] Corica F, Allegra V, Ferrara R, Mangano C, Di Benedetto A, Azzarello G. Magnesium and hypertension in nephropathic patients. *Clin Ter*. 2006;157(4):303–308.