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STUDY OF SERUM MAGNESIUM LEVELS IN TYPE 2 DIABETES MELLITUS

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ABSTRACT:

Background: Magnesium plays a crucial role in glucose metabolism and insulin action. This study aimed to evaluate serum magnesium levels in patients with type 2 diabetes mellitus (T2DM) and explore its relationship with glycemic control and treatment modalities.

Methods: This case-control study included 50 T2DM patients and 50 non-diabetic controls. Serum magnesium levels were measured and compared between groups. The association between magnesium levels and glycemic control, as well as the impact of different treatment types, was analyzed.

Results: T2DM patients had significantly lower mean serum magnesium levels compared to controls (1.72 ± 0.28 mg/dL vs 2.14 ± 0.22 mg/dL, $p<0.001$). Hypomagnesemia (<1.5 mg/dL) was more prevalent in T2DM patients (40%) than in controls (2%). T2DM patients were 32.066 times more likely to have hypomagnesemia ($p<0.001$). Uncontrolled diabetics had lower magnesium levels than controlled diabetics (1.14 ± 0.12 mg/dL vs 1.91 ± 0.32 mg/dL, $p<0.001$). Insulin-treated patients showed lower magnesium levels compared to those on oral hypoglycemic agents (1.35 ± 0.21 mg/dL vs 2.06 ± 0.26 mg/dL, $p<0.001$).

Conclusion: This study demonstrates a significant association between T2DM and lower serum magnesium levels. Poor glycemic control and insulin treatment were associated with lower magnesium levels. These findings suggest the potential importance of monitoring serum magnesium in T2DM management and warrant further investigation into the role of magnesium in diabetes care.

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Keywords: Type 2 diabetes mellitus, serum magnesium, hypomagnesemia, glycemic control, insulin treatment

INTRODUCTION:

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by hyperglycemia resulting from insulin resistance and relative insulin deficiency.[1] As a global health concern, its prevalence continues to rise, with projections estimating that 642 million adults will be affected by 2040.[2] While the pathogenesis of T2DM is complex and multifactorial, increasing evidence suggests that alterations in mineral metabolism, particularly magnesium homeostasis, may play a significant role in the development and progression of the disease.[3]

Magnesium, the fourth most abundant cation in the human body, is crucial for numerous physiological processes, including glucose metabolism and insulin action.[4] It serves as a cofactor for over 300 enzymatic reactions and is essential for the proper functioning of the insulin receptor and glucose transporter protein activity.[5] Recent studies have indicated that magnesium deficiency may contribute to insulin resistance and impaired glucose tolerance, key features of T2DM.[6]

Several epidemiological studies have reported an inverse relationship between serum magnesium levels and the risk of developing T2DM.[7,8] Moreover, lower serum magnesium concentrations have been observed in individuals with established T2DM compared to healthy controls.[9] This association has led researchers to investigate the potential role of magnesium in the pathophysiology of T2DM and its complications.

The present study aims to evaluate serum magnesium levels in patients with T2DM and compare them to those of healthy individuals. Additionally, we seek to explore the relationship between serum magnesium concentrations and various clinical and biochemical parameters associated with diabetes, such as glycated hemoglobin (HbA1c), fasting blood glucose, and lipid profile. By elucidating these associations, we hope to contribute to a better understanding of the role of magnesium in T2DM and potentially identify new avenues for therapeutic interventions or preventive strategies.

METHODS:

This case-control study was conducted at Dr. Balasaheb Vikhe Patil Rural Medical College, Loni, between February 2023 and July 2023. The study protocol

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was approved by the Institutional Ethics Committee, and informed consent was obtained from all participants prior to their enrollment.

The study population consisted of two groups: a case group of 50 patients with type 2 diabetes mellitus (T2DM) and a control group of 50 non-diabetic individuals. Patients in the case group were diagnosed with T2DM according to the American Diabetes Association criteria. The control group comprised individuals without a history of diabetes and with normal fasting blood glucose levels. Both groups were matched for age and gender to minimize potential confounding factors.

Inclusion criteria for the case group were: age 18 years or older, confirmed diagnosis of T2DM, and admission to the hospital during the study period. Exclusion criteria included: type 1 diabetes mellitus, gestational diabetes, use of magnesium supplements, chronic kidney disease (estimated glomerular filtration rate < 60 mL/min/1.73 m^2), and use of medications known to affect magnesium levels (e.g., diuretics, proton pump inhibitors).

Upon enrollment, a detailed medical history was obtained from each participant, including duration of diabetes, current medications, and presence of comorbidities. Anthropometric measurements, including height, weight, and waist circumference, were recorded. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared.

Blood samples were collected from all participants after an overnight fast of at least 8 hours. Serum was separated by centrifugation and analyzed for magnesium levels using the colorimetric method with xylidyl blue. Other laboratory parameters measured included fasting blood glucose, glycated hemoglobin (HbA1c), lipid profile (total cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol), and serum creatinine.

Blood pressure was measured in a seated position after 5 minutes of rest, using a standard mercury sphygmomanometer. Two readings were taken at an interval of 5 minutes, and the average was recorded.

Statistical analysis was performed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The Student's t-test was used to compare continuous variables between the case and control groups, while the chi-square test was employed for categorical variables. Pearson's correlation coefficient

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was calculated to assess the relationship between serum magnesium levels and other continuous variables. Multiple linear regression analysis was conducted to identify independent predictors of serum magnesium levels. A p-value < 0.05 was considered statistically significant.

RESULTS:

Present study included 50 type 2 diabetic patients and 50 control subjects. We had diabetic patients ranging from 40 to 80 years. In both the diabetic & control group males and females were 62% and 38% respectively. 50% of patients were on insulin, 37.5% on OHAs, 12.5% on both as therapy for diabetes. 68% of patients had fair control of diabetes, 34% has poorly controlled diabetes. We assessed serum magnesium levels in all these subjects.

Table 1 compares serum magnesium levels between cases (T2DM patients) and controls. The results show T2DM patients have significantly lower mean serum magnesium levels (1.72 ± 0.28 mg/dL) compared to controls (2.14 ± 0.22 mg/dL). This difference is statistically significant ($p<0.001$), indicating a strong association between T2DM and lower serum magnesium levels.The range of magnesium levels is wider in T2DM patients (1-2.5 mg/dL) compared to controls (1.5-2.6 mg/dL), suggesting more variability in the diabetic group.

Table 1: Effect of DM on serum magnesium

Serum Magenesium	**Cases**	**Controls**
Range (Min-Max)	1-2.5	1.5-2.6
Mean ±S.D	1.72±0.28	2.14±0.22
95% CI	0.052-1.56	0.04-1.96
Significance	Student t=5.649, **p<0.001**	

Table 2 categorizes serum magnesium levels and compares the distribution between cases and controls: 40% of T2DM patients have serum magnesium levels below 1.5 mg/dL, compared to only 2% of controls. The majority of controls (62%) have magnesium levels between 1.5-2 mg/dL, while T2DM patients are more evenly distributed across lower ranges. T2DM patients are 32.066 times more likely to have low serum magnesium (<1.5 mg/dL) compared to controls, a statistically significant finding ($p<0.001$).

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Table 2: Comparison of serum magnesium levels between cases and controls

Serum Magenesium	**Cases**	**Controls**
≤1	1 (2%)	0
1-1.5	19 (38%)	1 (2%)
1.5-2	21 (42%)	31 (62%)
2-2.5	9 (18%)	16 (32%)
>2.5	0	2 (4%)
Inferences	Cases are 32.066 times significantly more likely to have less serum magnesium (<1.5 mg/dl) when compared to controls with p<0.001	

Table 3 compares magnesium levels between controlled and uncontrolled diabetics: Controlled diabetics have significantly higher mean serum magnesium levels (1.91 ± 0.32 mg/dL) compared to uncontrolled diabetics (1.14 ± 0.12 mg/dL). The difference is statistically significant (p<0.001), suggesting a strong association between glycemic control and magnesium levels. The range of magnesium levels is wider in controlled diabetics (1.2-2.5 mg/dL) compared to uncontrolled diabetics (1-1.6 mg/dL).

Table 3: Effect of level of control of DM on serum magnesium

Serum Magenesium	**Controlled (n=34)**	**Not controlled (n=16)**
Range (Min-Max)	1.2-2.5	1-1.6
Mean ±S.D	1.91±0.32	1.14±0.12
95% CI	1.64-1.85	1.09-1.4
Significance	Student t=3.956, **p<0.001**	

Table 4 compares magnesium levels between insulin-treated and oral hypoglycemic agent (OHA) treated diabetics Insulin-treated patients have significantly lower mean serum magnesium levels (1.35 ± 0.21 mg/dL) compared to OHA-treated patients (2.06 ± 0.26 mg/dL).The difference is statistically significant (p<0.001), indicating a strong association between treatment type and magnesium levels.The range of magnesium levels is lower in insulin-treated

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patients (1-2.2 mg/dL) compared to OHA-treated patients (1.6-2.5 mg/dL).

Table 4: Effect of type of treatment on serum magnesium

Serum Magenesium	**Insulin (n=34)**	**OHA (n=16)**
Range (Min-Max)	1-2.2	1.6-2.5
Mean ±S.D	1.35±0.21	2.06±0.26
95% CI	1.41-1.6	1.86-2.18
Significance	Student t=5.988, **p<0.001**	

DISCUSSION:

This study aimed to evaluate serum magnesium levels in patients with type 2 diabetes mellitus (T2DM) compared to non-diabetic controls. Our findings demonstrate significantly lower serum magnesium levels in T2DM patients compared to controls (1.72 ± 0.28 mg/dL vs 2.14 ± 0.22 mg/dL, p<0.001). This result aligns with several previous studies that have reported hypomagnesemia in T2DM patients.[10]

The prevalence of hypomagnesemia (serum magnesium <1.5 mg/dL) was notably higher in the T2DM group (40%) compared to the control group (2%). This observation is consistent with a study by Ramadass et al., which reported hypomagnesemia in 41.8% of T2DM patients.[11] The increased likelihood of hypomagnesemia in T2DM patients (OR: 32.066, p<0.001) underscores the potential role of magnesium deficiency in the pathophysiology of T2DM.

Our study revealed a significant difference in serum magnesium levels between controlled and uncontrolled diabetics (1.91 ± 0.32 mg/dL vs 1.14 ± 0.12 mg/dL, p<0.001). This finding supports the hypothesis that poor glycemic control is associated with lower magnesium levels, as previously reported by Kurstjens et al.[12] The inverse relationship between serum magnesium and glycemic control suggests that magnesium deficiency may contribute to or result from poor glucose regulation.

Interestingly, we observed significantly lower serum magnesium levels in insulin-treated patients compared to those on oral hypoglycemic agents (1.35 ± 0.21 mg/dL vs 2.06 ± 0.26 mg/dL, p<0.001). This finding contrasts with some previous studies that found no significant difference in magnesium levels based on

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treatment type.[13] The lower magnesium levels in insulin-treated patients might be attributed to more severe insulin resistance or a longer duration of diabetes, factors that were not explicitly analyzed in our study.

The mechanism underlying hypomagnesemia in T2DM is multifactorial. Increased urinary magnesium excretion due to osmotic diuresis and tubular dysfunction in diabetic patients has been proposed as a primary cause.[14] Additionally, insulin resistance may impair magnesium uptake and retention in cells, further contributing to lower serum levels.

Our findings have important clinical implications. Given the high prevalence of hypomagnesemia in T2DM patients and its potential role in insulin resistance and glucose metabolism, routine screening of serum magnesium levels in T2DM patients may be warranted. Furthermore, magnesium supplementation could be considered as an adjunct therapy in T2DM management, particularly for patients with documented hypomagnesemia.[15]

This study has several limitations. The cross-sectional design precludes the establishment of causal relationships. Additionally, we did not assess dietary magnesium intake or measure intracellular magnesium levels, which might provide a more accurate representation of total body magnesium status. Future longitudinal studies with larger sample sizes are needed to elucidate the temporal relationship between magnesium status and T2DM progression.

CONCLUSION:

In conclusion, our study confirms the high prevalence of hypomagnesemia in T2DM patients and its association with poor glycemic control. These findings highlight the potential importance of magnesium in T2DM pathophysiology and management, warranting further investigation into the role of magnesium supplementation in diabetes care.

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