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Original Research Article

Association of serum magnesium levels with microvascular complications in type 2 diabetes mellitus, with significant correlation to diabetic retinopathy: a cross-sectional study

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ABSTRACT

Background: Magnesium is essential for numerous biological processes, including glucose metabolism, insulin responsiveness and vascular integrity. A deficiency in magnesium is commonly reported in individuals with type 2 diabetes mellitus (T2DM) and is suspected to contribute to the development of microvascular complications such as nephropathy, retinopathy and neuropathy. This study aimed to evaluate the prevalence of hypomagnesemia in T2DM patients and explore its association with microvascular complications.
Methods: A cross-sectional, observational study was conducted at SRN Hospital, Prayagraj, involving 143 patients diagnosed with T2DM. Serum magnesium levels were estimated, with values below 1.7 mg/dl considered indicative of hypomagnesemia. Statistical analysis was used to examine relationships between magnesium levels and the occurrence of microvascular complications as nephropathy, retinopathy and neuropathy.
Results: Among the 143 participants (average age 57 years), hypomagnesemia was identified in 17.5% of cases. The overall mean serum magnesium concentration was 1.93±0.29 mg/dl. A significant inverse relationship was observed between serum magnesium levels and the presence of diabetic retinopathy (p=0.008); retinopathy was found in 60% of patients with hypomagnesemia compared to 29.66% of those with normal magnesium levels. Although nephropathy and neuropathy were more prevalent in the hypomagnesemia group (40% and 28%, respectively), these associations were not statistically significant.
Conclusions: The study found a significant link between magnesium deficiency and diabetic retinopathy, highlighting a potential role of hypomagnesemia in its pathogenesis. Regular monitoring of magnesium status in T2DM patients may help identify those at higher risk for microvascular complications.

Keywords: Hypomagnesemia, Nephropathy, Neuropathy, Retinopathy, Type 2 diabetes mellitus

INTRODUCTION

Diabetes mellitus encompasses a range of metabolic disorders, all of which are marked by elevated blood glucose levels due to insufficient insulin production, impaired insulin action or a combination of both.[1] In individuals with type 2 diabetes mellitus (T2DM), disturbances in magnesium (Mg) metabolism are frequently encountered. This often manifests as magnesium deficiency, largely driven by inadequate intake and increased excretion through urine. The issue becomes more prominent in those with poorly managed blood sugar levels, extended disease duration and those who have developed chronic microvascular or macrovascular complications.[2-6]

Magnesium, the fourth most abundant mineral in the human body, is involved in more than 300 enzymatic reactions, including those crucial for glucose homeostasis and insulin signaling. Its role extends beyond metabolic

regulation to influence vascular tone, endothelial function and inflammatory pathways. In patients with T2DM, persistent hyperglycemia and insulin resistance can disrupt magnesium balance, leading to excessive renal excretion and reduced intracellular stores. This disruption not only affects glycemic control but may also contribute to the pathophysiology of long-term diabetic complications. Despite its importance, magnesium status is often overlooked in routine diabetes management, underscoring the need for greater awareness of its clinical significance. Magnesium is predominantly obtained through dietary intake, with rich sources including green leafy vegetables (such as spinach), legumes, nuts (like almonds and cashews), seeds, whole grains and certain fruits like bananas and avocados. Seafood, dairy products and dark chocolate also contribute to daily magnesium intake.

Despite the wide availability of magnesium-rich foods, dietary insufficiency remains common, particularly in individuals with poor nutritional habits or chronic illnesses such as diabetes. The frequent reliance on processed or refined foods typically low in magnesium further exacerbates the risk of deficiency.

METHODS

This cross-sectional observational study was carried out at Swaroop Rani Nehru Hospital which is a Tertiary care centre attached to Moti Lal Nehru Medical College Prayagraj, Uttar Pradesh involving 143 individuals previously diagnosed with Type 2 diabetes mellitus (T2DM). Ethical clearance was taken from Institutional Ethical committee.

The diagnosis of T2DM was based on the American Diabetes Association's established criteria. The study was conducted over a 12-month period from June 2023 to May 2024. Diabetic nephropathy was diagnosed based on persistent microalbuminuria (>30 mg/day). Retinopathy was diagnosed by fundus examination and confirmed by an ophthalmologist. Neuropathy was determined by clinical tests (monofilament, vibration perception) and, if necessary, nerve conduction studies.

Inclusion criteria

All those subjects who were more than 18 years of age and suffering from Type 2 diabetes mellitus were included in the study.

Exclusion criteria

Participants were excluded if they were taking medications known to interfere with magnesium levels such as diuretics, aminoglycosides, amphotericin B or digoxin or if they were receiving magnesium supplementation. Additionally, patients with chronic diarrhea, malabsorption syndromes, alcohol dependence or those who were pregnant or breastfeeding were not included in the study.

Serum magnesium concentration was evaluated in all enrolled participants, with hypomagnesemia defined as levels below 1.7 mg/dl. A thorough clinical history was documented for each patient. Laboratory assessments included evaluations for diabetic nephropathy, retinopathy and neuropathy.

The strength of association between two scale parameters was assessed using the Spearman correlation coefficient. A p value of <0.05 was considered statistically significant. Data were entered into an MS Excel spreadsheet and analysed using Statistical Package for Social Sciences (SPSS) version 23.0.

RESULTS

A total of 143 patients with T2DM participated in the study. Their ages ranged from 28 to 85 years, with an average age of 57 years. Among them, 65.73% were male and 34.27% were female. The average duration of diabetes was 7.26±4.71 years, with a median of 6 years (Table 1).

Hypomagnesemia, defined as serum magnesium levels below 1.7 mg/dl, was observed in 25 individuals (17.5%), while the remaining 118 participants (82.5%) exhibited normal magnesium levels. The overall mean serum magnesium level across the entire cohort was 1.93±0.29 mg/dl. Specifically, those in the hypomagnesemia group had a mean level of 1.55±0.12 mg/dl, while those with normal magnesium levels had a mean of 2.01±0.24 mg/dl.

Nephropathy was present in 40% of patients with hypomagnesemia and 44.92% of those with normal magnesium levels. It was less frequent in hypomagnesemia group, but the difference was not statistically significant (p=0.820). Retinopathy was observed in 60% of patients with hypomagnesemia and in 29.66% of those with normal magnesium levels. This difference was statistically significant (p=0.008).

Table 1: Baseline characteristics of study population (n=143).

Parameters	Value
Mean age (in years)	57
Age range (in years)	28–85
Gender (male/female)	94 (65.73%)/49 (34.27%)
Duration of diabetes (in years)	7.26±4.71
Median duration (in years)	6

Continued.

Parameters	Value
Mean serum magnesium (mg/dl)	1.93±0.29
Hypomagnesemia (<1.7 mg/dl)	25 (17.5%)
Normal magnesium (≥1.7 mg/dl)	118 (82.5%)

Table 2: Comparison of microvascular complications between hypomagnesemia and normal magnesium groups.

Complication	Hypomagnesemia (n=25)	Normal Mg (n=118)	P value
Nephropathy	10 (40.0%)	53 (44.92%)	0.820
Retinopathy	15 (60.0%)	35 (29.66%)	0.008*
Neuropathy	7 (28.0%)	23 (19.49%)	0.497

*Statistically significant (p<0.05), NS=Not Significant.

Table 3: Mean serum magnesium levels in relation to microvascular complications.

Complication	Present (N, %)	Mean Mg (mg/dl)	Absent (N, %)	Mean Mg (mg/dl)	P value
Nephropathy	63 (44.09)	1.95±0.30	80 (55.95)	1.90±0.26	0.296
Retinopathy	50 (34.9)	1.84±0.29	93 (65.1)	1.97±0.28	0.09*
Neuropathy	30 (20.97)	1.87±0.25	113 (79.03)	1.94±0.29	0.229

*Statistically significant (p<0.05).

Neuropathy was found in 28% of patients with hypomagnesemia and in 19.49% of patients with normal magnesium levels. Although neuropathy was more frequent in the hypomagnesemia group, the difference was not statistically significant (p=0.497) (Table 2).

DISCUSSION

In this study involving 143 individuals diagnosed with T2DM, the mean serum magnesium level was found to be 1.93±0.29 mg/dl. These results are in close agreement with those reported by Naqvi et al who documented a mean of 1.97±0.52 mg/dl.[7] Hypomagnesemia was present in 17.48% of patients, comparable to Hamarshih et al, Kumar et al, Prabhu et al and Alwsat et al who reported 11%, 19%,25% and 28% prevalence, respectively.[8-11] These figures differ from studies by Yossef et al and Roychowdhury et al which reported higher prevalence (80% and 83.8%) of hypomagnesemia.[12,13] These differences may be due to variations in study design, population characteristics, diagnostic criteria for hypomagnesemia, duration of diabetes, age distribution and treatment modalities.

In the subgroup with hypomagnesemia, the average serum magnesium concentration was 1.55±0.12 mg/dl, whereas it was 2.01±0.24 mg/dl among individuals with normal levels. These values mirror those reported by Moradiya et al who observed means of 1.56±0.09 mg/dl and 2.07±0.23 mg/dl in the respective groups.[14] Magnesium plays a pivotal role in carbohydrate metabolism and insulin signaling. It functions as a cofactor for numerous enzymatic reactions, including those involved in glucose utilization and insulin receptor activation. A deficiency in magnesium has been linked to diminished insulin sensitivity and compromised insulin secretion, both of which may accelerate the development of diabetic complications.[15,16] Persistent hyperglycemia in T2DM leads to osmotic diuresis, contributing to excessive urinary magnesium loss. Over time, this magnesium depletion may promote oxidative stress, endothelial dysfunction and inflammation, which are critical pathways in the pathogenesis of microvascular complications such as nephropathy, retinopathy and neuropathy. Furthermore, hypomagnesemia is associated with increased platelet aggregation and vascular calcification, both of which can aggravate microvascular injury. Nephropathy was present in 63 patients (44.09% of the study population), slightly higher than the 29.9% with microalbuminuria reported by Alswat et al but is lower than that of Shushma et al where 60% of participants exhibited microalbuminuria.[11,17] The mean serum magnesium level in patients with nephropathy was 1.95 mg/dl while it was 1.90 mg/dl in those without nephropathy. These results differ from those of Shushma et al who reported mean serum magnesium levels of 1.7 mg/dl and 1.85 mg/dl in nephropathy and non-nephropathy patients, respectively.[17]

Similarly, a study by Nayyar et al reported lower serum magnesium levels in nephropathy patients (1.68 mg/dl) compared to those without nephropathy (1.82 mg/dl).[18] These results also differ from those of Kumar et al, Alwsat et al who found a significant positive association between hypomagnesemia and diabetic nephropathy.[9,11] The variation in the findings could be due to the relatively small sample size and the reliance on urine microalbumin levels, which may not be the most robust marker for defining nephropathy (Table 3). Retinopathy was observed in 50 patients (34.9%), with a mean serum magnesium level of 1.84 mg/dl. While in those 93 patients without retinopathy (65.1%), the mean serum magnesium level was 1.97 mg/dl (p=0.09). This aligns with the study by Rajpoot et al which reported serum magnesium levels of 1.89 mg/dl in patients with retinopathy and 2.23 mg/dl in those without.[19] A study by Suma et al found an even greater disparity, with magnesium levels of 1.14 mg/dl in

patients with retinopathy and 1.42 mg/dl in those without.[20] Despite differences in absolute values, both studies, including ours, demonstrated a significant inverse association between serum magnesium levels and diabetic retinopathy. Magnesium may protect retinal microvasculature by modulating vascular tone, reducing oxidative damage and improving endothelial function. Deficiency in magnesium could impair retinal blood flow autoregulation and potentiate ischemic and inflammatory damage that leads to retinopathy.[21]

Neuropathy was present in 30 patients (20.97%) in our study. This prevalence is similar to that reported by Naqvi et al and Mukkar et al who found neuropathy in 16% and 15.3% of diabetic patients, respectively.[7,22] The mean serum magnesium level in patients with neuropathy was 1.87 mg/dl compared to 1.94 mg/dl in those without. Mukkar et al reported substantially lower magnesium levels in neuropathy patients (0.8 mg/dl) compared to non-neuropathy patients (1.7 mg/dl).[22] While the difference in serum magnesium levels in our study was less pronounced and not statistically significant (p=0.229), both studies suggest lower magnesium levels may be associated with diabetic neuropathy. In diabetic neuropathy, magnesium deficiency may play a role through neuronal hyperexcitability, impaired neuromuscular conduction and increased susceptibility to oxidative stress. It is hypothesized that low magnesium levels exacerbate neuronal ischemia and metabolic dysfunction, promoting the development of neuropathic symptoms.[23]

The findings of this study suggest that monitoring and managing serum magnesium levels could have significant clinical benefits in patients with T2DM, particularly in those with or at risk of developing microvascular complications. Despite the lack of statistically significant associations with nephropathy and neuropathy in our cohort, the trends observed align with broader evidence suggesting magnesium's protective role. Routine assessment of magnesium status, particularly in patients with poor glycemic control or longstanding diabetes, could facilitate early intervention. Magnesium supplementation in deficient individuals might help slow the progression of complications, improve insulin sensitivity and potentially enhance overall metabolic control.

Limitations

Larger prospective studies are needed to confirm these associations and evaluate the clinical benefits of magnesium supplementation.

CONCLUSION

This cross-sectional study of 143 individuals with T2DM found that 17.5% of participants had hypomagnesemia. A statistically significant inverse relationship was observed between low serum magnesium levels and the presence of diabetic retinopathy (p=0.008), indicating that magnesium deficiency may be a contributing factor in its development. While nephropathy and neuropathy also showed a trend toward association with lower magnesium levels, these associations did not achieve statistical significance. Magnesium's established involvement in insulin action, vascular integrity and oxidative stress response highlights its potential role in the pathogenesis and progression of diabetes-related complications.

These findings underscore the clinical value of routinely monitoring magnesium levels in individuals with T2DM particularly in those with long-standing disease or signs of poor glycemic control. Incorporating magnesium assessment into standard diabetic care may aid in early identification of individuals at increased risk for microvascular complications. Routine magnesium monitoring at primary care level may help in early risk stratification of diabetic retinopathy in resource-limited settings Timely correction of magnesium deficiency, whether through dietary modifications or supplementation, could improve metabolic outcomes and reduce long-term complications. Nonetheless, further large-scale, prospective and interventional studies are warranted to validate these findings and clarify the therapeutic potential of magnesium in the management of T2DM.

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