

HYPOMAGNESEMIA AS AN IMPORTANT ELECTROLYTE IMBALANCE IN PATIENTS WITH DIABETIC NEPHROPATHY

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ABSTRACT

Background & Objective: Diabetes Mellitus (T2DM) leads to disturbances in the homeostasis of different inorganic minerals, including Magnesium, particularly in diabetic nephropathy (DN) patients. The study aimed to find the frequency of hypomagnesemia in patients with T2DM with early DN. It also compares glycemic control (HbA1c levels) and renal function between patients with and without hypomagnesemia.

Methodology: This cross-sectional study was conducted in internal medicine department, Hameed Latif Hospital, Lahore during June 2025 and September 2024. Non-probability consecutive sampling was used. The T2DM patients aged 20-65 years with venous fasting blood sugar (FBS) ≥ 6.5 mmol/L with early DN were included. Thus, comparison of groups (hypomagnesaemia as yes/no) was done with age, BMI, duration of diabetes, HbA1c, eGFR, UACR, and serum magnesium levels using non-parametric Mann-Whitney U test. The multivariate logistic regression model was performed with dependent binary variable (hypomagnesemia) taking $p < 0.05$ to confirm statistical significance at 95% CI.

Results: The 15 (10%) T2DM patients reported hypomagnesemia, with mean age of 49.07 ± 6.46 years, including 7 males and 8 females. In bivariate analysis, hypomagnesemia is significantly associated to middle adult age, duration of DM, glycemic control (HbA1c levels), UACR levels and renal function (eGFR) ($p < 0.05$). While doing logistic regression (multivariate) analysis, only age was significant with odds ratio of (OR = 0.91, 95% CI: 0.83–0.98, $p = 0.02$).

Conclusion: The study highlights the importance of monitoring hypomagnesemia among T2DM patients, both with or without early DN. Hypomagnesemia is found to be prevalent in T2DM and associated with glycemic control (HbA1c), renal function (eGFR), and UACR levels. However, no association was found between BMI, gender, and retinopathy. Interestingly, magnesium imbalance was observed even among patients with relatively preserved renal function and normal to mildly increased UACR levels, suggesting that hypomagnesemia may occur during the early stages of DN. No significant association was found between hypomagnesemia and BMI, gender, or retinopathy. In multivariate analysis, only age is significantly associated to hypomagnesemia among T2DM patients with or without early DN.

KEYWORDS: Diabetes Mellitus, Diabetic Nephropathy, Hypomagnesemia, Electrolyte Imbalance.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is the most common metabolic disorder that is caused due to insulin resistance and decreased insulin responsiveness of the pancreatic beta cells, thus, leading to hyperglycemia (1). In Pakistan, Diabetes Mellitus (DM) affects approximately 10-26% of the adult population (2) and is related to significant morbidity due to its complications. Among these complications, diabetic nephropathy (DN) among DM can cause microvascular and macrovascular complications such as end-stage renal disease (ESRD)(3) affecting approximately 30-40% T2DM in Pakistan. The risk factors of DN include poor glycemic control, long duration of DM, hypertension, and presence of microalbuminuria (4).

Magnesium plays a vital role in maintaining various metabolic physiological functions (5). T2DM disrupts magnesium homeostasis. T2DM patients often have chronic latent magnesium deficiency or clinical

hypomagnesaemia, especially those with poor glycaemic control (6). Magnesium concentration is mostly controlled by insulin (7). Increased urine loss and decreased food intake are the main causes of magnesium depletion in T2DM patients (8). Hypomagnesaemia affects 32% of Pakistani T2DM patients (9). Low magnesium levels in DM can cause neuropathy, retinopathy and foot ulcers (10).

In patients with DM and hypomagnesaemia, magnesium treatment lowers plasma glucose and improves glycemic status (11). The role of serum magnesium in DN is unclear in Pakistan. Some research suggests that lower levels increase the likelihood of development and progression of DM (12). Sudha.S et al. (2018) also found association between hypomagnesemia, T2DM and DN. Out of the 100 patients in total, 40 individuals, aged 55-65 years, were diagnosed with hypomagnesemia. Low serum magnesium is linked to higher HbA1c in T2DM patients, indicating poorer glycemic control (13). Another study also indicate that hypomagnesemia is a common finding in patients having poor glycemic control. The prevalence of hypomagnesemia in patients with HbA1c <7% was 21.2% compared to patients with HbA1c >7% which was markedly higher at 78.8%. So, the difference in serum magnesium levels in relation to HbA1c was statistically significant ($P \leq 0.001$) (14).

Owing to the uncertainty and lack of evidence, there is need for original research to address and provide the initial cross-sectional evidence. However, serum magnesium levels are not routinely done in patients with DN, which needs to be addressed. Furthermore, clinical symptoms associated with hypomagnesemia manifest late, which also emphasize the need for checking magnesium levels. Therefore, we aimed to find the proportion of hypomagnesemia in T2DM patients with DN and compare glycemic control (HbA1c levels) in patients either with or without hypomagnesemia.

METHODOLOGY

This cross-sectional study was conducted in Hameed Latif Hospital's internal medicine department from 1st June 2025 to 12th September, 2025. No-probability consecutive sampling was used. Using open-epi web-based calculator, 150 patients were calculated with $Z\% = 95\%$, $P\% = 11\%$ (16), and absolute precision required (D) = 5% (15). The patients included in this study were; patients with T2DM diagnosis, confirmed by venous fasting blood sugar (FBS) ≥ 6.5 mmol/L, both male and female, aged 20-65 years, patients having normal to early microalbuminuria UACR (<300 mg/g) and eGFR > 15 mL/min/1.73m², suggesting early diabetic kidney involvement, and willing to provide written informed consent. However, patients with eGFR < 15 mL/min/1.73m² (to exclude ESRD), patients with UACR > 300 mg/g (to exclude advance or macroalbuminuria), type-1 diabetics (autoimmune insulin dependent diabetes called juvenile-onset diabetes), patients on hemodialysis, on drugs known to affect magnesium levels i.e., Aminoglycosides, Diuretics, Digoxin, laxatives containing magnesium, multivitamins, etc., suffering from hypertension, with thyroid or adrenal dysfunction, acute or chronic malabsorption states, pregnancy or lactating women were excluded.

Data was collected from Hameed Latif Hospital Lahore outpatient clinics of nephrology and medicine department. All participants fulfilling the inclusion criteria were given a written informed consent form to sign. Implementation steps of the study were as followed; Recruitment was done by identifying and enrolling eligible patients from outpatient clinics. The selected participants' age, gender, FBS or RBS (Venous sample), HbA1c (immuno-inhibition method), UACR, eGFR (Cockcroft-Gault Equation), and serum magnesium levels (photometric xylidyl blue method) were collected and entered after pathologist and classified medical specialist verification. Ethical approval from Hameed Latif Hospital Lahore Research Committee was taken before commencement of the research. All participants were ensured for their confidentiality and anonymity.

Data was entered and analyzed using SPSS version 27.0. Quantitative variables such as age, BMI, duration of diabetes, HbA1c, eGFR, UACR and serum magnesium were reported as mean \pm SD to demonstrate clinical and biochemical characteristics. Qualitative variables such as gender, hypomagnesaemia (Yes/No), and retinopathy (Yes/No) were reported as frequency and percentages. Kolmogorov Smirnov test was used to test the normality of the data of the variables under study. The findings showed that data were not normally distributed. Thus, comparison of groups (hypomagnesaemia as yes/no) was done with age, BMI, duration of diabetes, HbA1c, eGFR, UACR, and serum magnesium levels using non-parametric Mann-Whitney U test. Furthermore, association of hypomagnesemia with gender and retinopathy was also determined using Chi-Square test. The logistic (binary) regression model assumptions were checked and it was found to be met. The dependent variable in the analysis, hypomagnesemia is binary (Yes/No). The standardized residues scatterplot was almost random distribution and it showed that the homoscedasticity had been observed. The multicollinearity was not concerned in cases of VIF less than 10. The p-value < 0.05 was taken to determine statistical significance at 95% confidence interval. To conduct the bivariate test, the continuous variables of BMI, UACR (mg/g), age, disease duration, eGFR (mL/min/1.73 m²), and HbA1c (%) were organized into clinically meaningful categories to evaluate their relationship with hypomagnesemia using Chi-Square tests.

RESULTS

The cross-sectional study included 150 T2DM patients with normal to early stages of diabetic kidney disease, having mean age of 49.07 ± 6.46 years. The majority of patients were males 84(56%) (Table 1). The 15(10%) patients were reported with hypomagnesemia, including 7 males and 8 females (Fig 1).

Table 1: Descriptive Statistics of Clinical and Biochemical Characteristics of Study Participants.

Variables	Mean ± SD	Minimum	Maximum
Age (years)	49.07 ± 6.46	27.00	62.00
Body Mass Index (kg/m ²)	31.10 ± 2.27	24.00	35.00
Duration of Diabetes (years)	8.67 ± 4.48	1.00	15.00
HbA1c (%)	8.46 ± 1.51	6.50	11.60
eGFR (mL/min/1.73 m ²)	62.70 ± 46.01	15.01	129.98
Urine Albumin–Creatinine Ratio (mg/g)	100.19 ± 94.32	0.49	298.25
Serum Magnesium (mmol/L)	0.82 ± 0.10	0.59	0.99

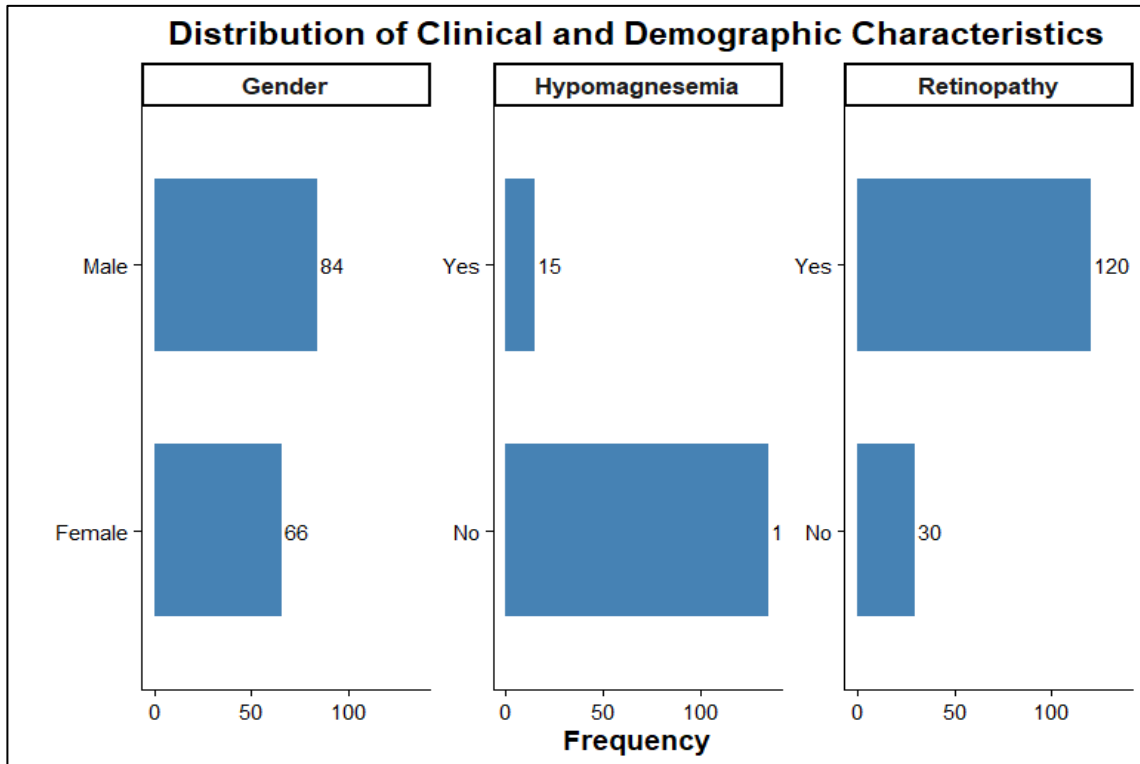


Figure 1: Distribution of Gender, Retinopathy, and Hypomagnesemia among Study Participants

Comparative (Bivariate) analysis of clinical and biochemical parameters using Mann Whitney U test between patients with and without hypomagnesemia showed statistically significant differences in terms of age ($p = 0.034$), duration of diabetes ($p = 0.007$), HbA1c ($p = 0.008$), eGFR ($p = 0.010$) and UACR ($p = 0.039$). Hypomagnesemia patients were considerably younger and had shorter duration of diabetes than those patients without hypomagnesemia. They also exhibited much lower HbA1c as well as lesser UACR levels. On the contrary, mean rank of eGFR among hypomagnesemia patients are also higher than those without hypomagnesemia. There was no significant difference was observed in BMI ($p = 0.380$) (Table 2).

Table 2: Comparison of Clinical and Biochemical Parameters According to Hypomagnesemia

Variables	Hypomagnesemia	N	Mean Rank	Sum of Ranks	p-value
Age	Yes	15	52.93	794.00	0.034
	No	135	78.01	10531.00	
BMI	Yes	15	84.83	1272.50	0.380
	No	135	74.46	10052.50	
Duration of DM	Yes	15	46.90	703.50	0.007
	No	135	78.68	10621.50	
HbA1c (%)	Yes	15	47.13	707.00	0.008
	No	135	78.65	10618.00	
eGFR (mL/min/1.73m ²)	Yes	15	102.93	1544.00	0.010
	No	135	72.45	9781.00	
Urine Albumin to Creatinine Ratio (UACR) mg/g	Yes	15	53.53	803.00	0.039
	No	135	77.94	10522.00	

The UACR levels, duration of DM, renal function (eGFR), and glycemic control (HbA1c levels) were significantly associated with hypomagnesemia. The findings explain patients with hypomagnesemia exhibited relatively

preserved renal function and lower UACR levels, which suggest that magnesium imbalance may be possible during early stages of diabetic kidney involvement. However, hypomagnesemia was not associated with gender or retinopathy ($p > 0.05$) (Table 3).

Table 3: Association of Demographic and Clinical Variables with Hypomagnesemia Using Chi-Square Test.

Variables	Category	Hypomagnesemia Yes	Hypomagnesemia No	Total	p-value
Gender	Male	7	77	84	0.443
	Female	8	58	66	
Retinopathy	Yes	10	110	120	0.174
	No	5	25	30	
BMI	Normal (18.5-24.9)	0	1	1	0.910
	Overweight (25-29.9)	3	31	34	
	Obese (≥ 30)	12	103	115	
UACR (mg/g)	Normal (<100)	12	72	84	0.048
	Early Microalbuminuria (100-300)	3	63	66	
Age (years)	Young Adults (18–40 years)	2	9	11	0.500
	Middle-Aged Adults (41–60 years)	13	121	134	
	Older Adults (>60 years)	0	5	5	
Disease Duration (years)	<5 years	8	37	45	0.038
	>5 years	7	98	105	
eGFR mL/min/1.73 m ²)	Normal Kidney Function (≥ 90)	12	54	66	0.003
	Moderate to Severe Kidney Dysfunction (15-89)	3	81	84	
HbA1c (%)	Well controlled (<7.0%)	4	26	30	0.016
	Moderate control (7.0–8.5%)	10	49	59	
	Poor control (>8.5%)	1	60	61	

In multivariate logistic regression analysis, the only variable that was significantly related with hypomagnesemia in the logistic regression analysis was age (OR = 0.91, 95% CI: 0.83–0.98, $p = 0.02$) (Table 4).

Table 4: Logistic Regression Analysis of Factors Associated with Hypomagnesemia

Variable	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for Exp(B)
Age	-0.09	0.04	5.64	1	0.02	0.91	0.83 – 0.98
Gender	-0.02	0.61	0.00	1	0.97	0.98	0.30 – 3.21
BMI	-0.23	0.15	2.21	1	0.14	0.80	0.59 – 1.08
Duration of DM	-0.02	0.10	0.03	1	0.85	0.98	0.80 – 1.20
HbA1c (%)	-0.34	0.62	0.30	1	0.59	0.71	0.21 – 2.42
Retinopathy	-1.17	0.74	2.53	1	0.11	0.31	0.07 – 1.31
eGFR (mL/min/1.73m ²)	-0.02	0.02	1.31	1	0.25	0.98	0.95 – 1.01
Urine Albumin to Creatinine Ratio (UACR) mg/g	0.00	0.01	0.09	1	0.76	1.00	0.99 – 1.02
Constant	5.54	6.27	0.78	1	0.38	254.75	–

DISCUSSION

The cross-sectional study included 150 T2DM patients, out of which 15(10%) reported hypomagnesemia (7 males and 8 females) with a mean age of 49.07 ± 6.46 years. The significant associations were observed between hypomagnesemia and duration of DM, glycemic control (HbA1c), renal function (eGFR) and UACR levels ($p < 0.05$). Notably, the majority of 12 out of 15 hypomagnesemia patients had reported normal renal function (eGFR) and normal UACR levels, which suggests that hypomagnesemia may occur even in the early stages of diabetic kidney involvement. In multivariate logistic regression analysis, only age was significantly associated with odds ratio of (OR = 0.91, 95% CI: 0.83–0.98, $p = 0.02$).

The study found 15(10%) hypomagnesemia among T2DM patients with normal to early diabetic kidney involvement, which is aligned with the systematic review and meta-analysis conducted by Pitliya A. et al., (2024) who also found that hypomagnesemia is common in DM, and the estimates are between 10% and 40% (6). In another comparable cross-sectional study of 100 T2DM patients conducted by Saproo, N., & Singh, R. (2017), the 30% prevalence of hypomagnesemia was estimated among T2DM with majority of patients were reported with poor glycemic controlled (16). Hence, the hypomagnesemia is prevalent condition among T2DM patients either with or without early kidney involvement.

The study found that hypomagnesemia is significantly associated with duration of DM illness, moderate to poor glycemic control (HbA1c), renal function (eGFR), and UACR levels. The results are also consistent with the study conducted by Rojbi et al., (2025) who also found significant association between higher HbA1c levels and hypomagnesemia. Hence, abnormal renal magnesium reabsorption due to higher levels of HbA1c levels may contribute to hypomagnesemia (17).

Furthermore, it was also observed by Rojbi et al., (2025) that slight renal function decline (eGFR) also deteriorates the reabsorption of magnesium, leading to hypomagnesemia. The similar finding is also found in our study, which also reported that along with T2DM the slight decline in renal function (eGFR) also contribute to hypomagnesemia (3 out of 15 patients). Hence, earlier studies indicate that inadequate glycemic and toxicity are the primary factors causing magnesium deficiency in diabetic patients. Oost LJ et al., (2024) found significant association of serum magnesium levels with renal failure markers such as *MUC1/TRIM46*, *SHROOM3*, and *SLC22A7* (18). However, our cross-sectional study exclude ESRD patients, the prevalence of hypomagnesemia is still evident among T2DM despite normal renal function among 12/15 T2DM with $eGFR > 90 \text{ mL/min/1.73 m}^2$. Furthermore, the 12 out of 15 T2DM patients with hypomagnesemia also reported normal UACR levels. Therefore, hypomagnesemia may happen in T2DM patients in absence renal impairment. The 3 out of 15 T2DM patients with hypomagnesemia reported UACR levels (100–300 mg/g), suggesting early microalbuminuria compromise kidney function is also responsible for hypomagnesemia. As the study also reported that hypomagnesemia is closely related to early or established DN because magnesium is largely excreted by kidneys and its deficiency deteriorates renal function (19).

In this study, age was determined as a significant factor of hypomagnesemia and has odds ratio of (OR = 0.91, 95% CI: 0.83–0.98, $p = 0.02$), which indicates that, adult diabetic patients (30–60 years) with poor glycemic control are more likely to develop magnesium deficiency. This observation is in agreement with study such as Rojbi et al., (2025) who also reported odd ratios of 2.031 in its prospective study (17). The findings of our cross-sectional study are promising, but readers and professional must be cautious while interpreting the results.

There are some limitations of the study such as cross-sectional study design present temporal relation rather than causal. The study is based on single center and specific point of time rather than prospective data collection and observation to establish a causal. Although findings are based on reliable laboratory tests, multicenter and large sample prospective studies are encouraged to validate the findings. The finding that a majority of hypomagnesemia patients preserved renal function is indicative that magnesium deficiency in T2DM may be preceded by hyperglycemia-related excessive urinary magnesium excretion and insulin resistance. Despite the significant values of some of the variables in the bivariate analysis, only age was significant in multivariate logistic regression analysis. This can be attributed to the fact that there are relatively few cases of (15 out of 150) hypomagnesemia and potential overlap among clinical variables such as HbA1c, the duration of diabetes, and the renal function.

CONCLUSION

The study highlights the importance of monitoring hypomagnesemia among T2DM patients, both with or without early DN. Hypomagnesemia is found to be prevalent in T2DM and associated with glycemic control (HbA1c), renal function (eGFR), and UACR levels. However, no association was found between BMI, gender, and retinopathy. Interestingly, magnesium imbalance was observed even among patients with relatively preserved renal function and normal to mildly increased UACR levels, suggesting that hypomagnesemia may occur during the early stages of DN. In multivariate analysis, only age is found to be significant factor related to hypomagnesemia among T2DM patients with or without early DN. As the study design is cross-sectional, more prospective studies are emphasized to monitor magnesium levels over time to further validate the findings. The study is also based on one center of metropolitan city Lahore. The multicenter study is emphasized to generalize the findings.

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AUTHOR'S CONTRIBUTION

MMA: Concept, Data Collection, Statistical Analysis, Results Reporting, Methodology Writing & Planning, Manuscript writing, Final Approval.

RYS: Concept, Methodology Design, Supervision throughout the study, Manuscript revision, Proofreading, Final Approval.

KRR: Critical Review of Manuscript, Designing, Revision, Proofreading.

TS: Critical Review of Manuscript, Designing, Revision, Proofreading.

FK: Review of Manuscript, Revision, Proofreading.

SN: Revision of manuscript, Proofreading, Drafting of Manuscript.

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