International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Insulin Resistance and Renal Magnesium Handling: Serum Magnesium Levels in Type 2 Diabetes Mellitus

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Received: 25-06-2026
Accepted: 05-07-2026
Available online: 20-07-2026

ABSTRACT

Background: Hypomagnesemia has been increasingly recognized in patients with type 2 diabetes mellitus, yet its relationship with glycemic control remains incompletely understood. This study aimed to investigate serum magnesium levels and their correlation with fasting plasma glucose in patients with type 2 diabetes compared to healthy controls.

Methods: A prospective case-control study was conducted involving 150 patients with type 2 diabetes mellitus and 150 age- and gender-matched healthy controls. Fasting plasma glucose was estimated by the enzymatic glucose oxidase-peroxidase method, and serum magnesium was measured using the Calmagite dye-binding method. Independent samples t-test was employed for group comparisons, and Pearson's correlation coefficient was calculated to assess the relationship between variables.

Results: The mean serum magnesium level was significantly lower in diabetic patients (1.54 ± 0.18 mg/dL) compared to controls (2.06 ± 0.14 mg/dL), with a mean difference of 0.52 mg/dL ($p < 0.001$). Hypomagnesemia (serum magnesium < 1.7 mg/dL) was present in 124 (82.7%) diabetic patients versus only 2 (1.3%) controls ($p < 0.001$). A strong inverse correlation was observed between serum magnesium and fasting plasma glucose among diabetic patients ($r = -0.68$, $p < 0.001$), while no significant correlation was found in controls ($r = -0.12$, $p = 0.142$).

Conclusion: This study demonstrates a significantly higher prevalence of hypomagnesemia in patients with type 2 diabetes mellitus and a strong inverse correlation between serum magnesium and fasting plasma glucose. These findings suggest that magnesium depletion is closely associated with poor glycemic control, supporting the clinical utility of routine serum magnesium assessment in diabetic patients.

Keywords: *Type 2 diabetes mellitus, hypomagnesemia, serum magnesium, fasting plasma glucose, insulin resistance.*

INTRODUCTION

Magnesium is the fourth most abundant mineral in the human body and serves as an essential cofactor for over 600 enzymatic reactions, including those involved in adenosine triphosphate (ATP) metabolism, protein synthesis, DNA replication and repair, and neuromuscular function [1]. Among its numerous physiological roles, magnesium is critically important for glucose homeostasis and insulin action, participating in insulin secretion, insulin receptor signaling, and the regulation of tyrosine kinase activity at the insulin receptor level [2]. The interplay between magnesium status and glucose metabolism has garnered increasing scientific attention, particularly given the rising global prevalence of type 2 diabetes mellitus (T2DM) and its associated complications [3].

Type 2 diabetes mellitus is a chronic metabolic disorder characterized by insulin resistance and progressive beta-cell dysfunction, leading to hyperglycemia and a heightened risk of microvascular and macrovascular complications [4]. Emerging evidence over the past two decades has consistently demonstrated that individuals with T2DM frequently exhibit lower serum magnesium concentrations compared to their non-diabetic counterparts, a condition termed hypomagnesemia [5]. This association is not merely coincidental but appears to reflect a complex bidirectional relationship wherein insulin resistance influences magnesium homeostasis, and magnesium deficiency, in turn, exacerbates impairments in insulin sensitivity and glycemic control [6]. Epidemiological studies have reported that hypomagnesemia affects between 13.5% and 47.7% of patients with T2DM, with prevalence rates varying according to glycemic control, diabetes duration, and presence of complications [7].

The pathophysiology linking insulin resistance to magnesium depletion involves multiple interconnected mechanisms, with renal magnesium handling emerging as a particularly important determinant. Magnesium homeostasis is primarily regulated by three processes: intestinal absorption, bone storage, and renal excretion [2]. The kidneys play a pivotal role in maintaining magnesium balance, with approximately 95% of filtered magnesium being reabsorbed along the nephron [8]. The thick ascending limb of the loop of Henle and the distal convoluted tubule are critical sites for this reabsorption, where the transient receptor potential melastatin type 6 (TRPM6) channel functions as a key regulator of active magnesium transport [9]. Fine-tuning of magnesium reabsorption occurs in the distal convoluted tubule, a segment that is particularly sensitive to hormonal regulation and metabolic influences [10].

Insulin exerts direct effects on renal magnesium handling through multiple pathways. Experimental studies have demonstrated that insulin stimulates TRPM6 channel expression and activity, thereby promoting magnesium reabsorption in the distal nephron [11]. However, in states of insulin resistance, this stimulatory effect is attenuated, leading to impaired renal magnesium conservation and increased urinary magnesium loss [12]. Furthermore, hyperglycemia itself contributes to renal magnesium wasting through osmotic diuresis, which reduces the tubular concentration gradient necessary for passive magnesium reabsorption in the proximal tubule and thick ascending limb [13]. The resulting magnesium deficiency perpetuates a vicious cycle, as intracellular magnesium depletion impairs insulin signaling by reducing insulin receptor autophosphorylation and downstream effector activation [14].

Additional mechanisms linking insulin resistance to altered renal magnesium handling include the effects of inflammatory cytokines and oxidative stress. Pro-inflammatory mediators such as tumor necrosis factor-alpha and interleukin-6, which are elevated in insulin-resistant states, can downregulate TRPM6 expression and disrupt tubular magnesium transport [15]. Furthermore, hyperinsulinemia itself may promote renal magnesium wasting through competitive inhibition of common transport pathways in the thick ascending limb [8]. These multifactorial disturbances in renal magnesium handling contribute to the progressive depletion of magnesium stores observed in patients with poorly controlled diabetes.

The clinical implications of magnesium deficiency in T2DM extend beyond glycemic control. Hypomagnesemia has been associated with increased risk of diabetic complications, including retinopathy, nephropathy, neuropathy, and cardiovascular disease [6]. Moreover, low serum magnesium levels predict the development of T2DM in prospective cohort studies and are inversely associated with insulin resistance indices in cross-sectional analyses [3]. These observations underscore the importance of understanding the relationship between insulin resistance and renal magnesium handling as a basis for potential therapeutic interventions.

This review aims to synthesize current knowledge regarding the mechanisms by which insulin resistance affects renal magnesium handling and contributes to altered serum magnesium levels in patients with type 2 diabetes mellitus. By elucidating these pathophysiological relationships, we may identify opportunities for clinical intervention and improve management strategies for this common electrolyte disturbance in the diabetic population.

METHODOLOGY

Study Design and Setting

This prospective case-control study was conducted in the Department of Biochemistry in collaboration with the Department of Medicine at Chigateri Government hospital over a period of 5 months from January 2025 to June 2025 . The study protocol was approved by the Institutional Ethics Committee, and the research was performed in accordance with the ethical standards outlined in the Declaration of Helsinki. Written informed consent was obtained from all

participants prior to enrollment in the study. The study was conducted following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for case-control studies.

Sample Size Estimation

The sample size was calculated using G*Power software version 3.1.9.7. Based on a previous study that reported a mean serum magnesium difference of 0.28 mg/dL between diabetic and non-diabetic groups with a standard deviation of 0.45 mg/dL, the effect size (Cohen's d) was determined to be 0.62. With a two-tailed test, α error probability of 0.05, and power (1-β error probability) of 0.95, the calculated sample size was 136 participants per group. Anticipating a 10% attrition rate due to incomplete data or sample hemolysis, the final sample size was rounded to 150 participants in each group, yielding a total of 300 study participants.

Study Population

The study included 150 patients with type 2 diabetes mellitus (cases) and 150 age- and gender-matched healthy non-diabetic individuals (controls) attending the outpatient and inpatient departments of Medicine. The selection of participants followed a consecutive sampling method.

Inclusion Criteria for Cases

- Patients diagnosed with type 2 diabetes mellitus according to the American Diabetes Association criteria: fasting plasma glucose ≥126 mg/dL
- Age between 30 and 70 years
- Both males and females
- Willingness to provide informed consent

Inclusion Criteria for Controls

- Age- and gender-matched healthy individuals
- Fasting plasma glucose <100 mg/dL
- No personal or family history of diabetes mellitus
- No history of chronic illness
- Willingness to provide informed consent

Exclusion Criteria (for both groups)

- Type 1 diabetes mellitus
- Pregnant or lactating women
- Individuals on magnesium supplements or medications affecting magnesium homeostasis (diuretics, proton pump inhibitors, calcineurin inhibitors)
- Chronic kidney disease (serum creatinine >1.5 mg/dL)
- Chronic liver disease
- Malabsorption syndromes
- Alcohol use disorder
- Thyroid disorders
- Parathyroid disorders
- Malignancy
- Acute illness or infection at the time of sampling

Sample Collection

Venous blood samples (5 mL) were collected from all participants under strict aseptic precautions after an overnight fasting period of 10-12 hours. Blood samples were collected in fluoride oxalate vacutainers for glucose estimation and plain vacutainers for magnesium analysis. Samples were centrifuged at 3000 rpm for 10 minutes within 30 minutes of collection. Serum was separated and stored at -20°C until analysis, which was performed within 48 hours of collection. Hemolyzed, lipemic, or icteric samples were excluded from the analysis. Sample collection and handling procedures adhered to the Clinical and Laboratory Standards Institute (CLSI) guidelines.

Biochemical Analysis

Fasting Plasma Glucose Estimation

Fasting plasma glucose was estimated by the enzymatic glucose oxidase-peroxidase (GOD-POD) method using a commercially available kit on a semi-automated biochemistry analyzer. The principle involves the oxidation of glucose to gluconic acid and hydrogen peroxide by glucose oxidase. Hydrogen peroxide, in the presence of peroxidase, reacts with

phenol and 4-aminoantipyrine to form a colored quinoneimine dye. The intensity of the color formed is directly proportional to the glucose concentration and was measured at 505 nm. The results were expressed in mg/dL. The inter-assay and intra-assay coefficients of variation were 3.2% and 2.8%, respectively.

Serum Magnesium Estimation

Serum magnesium was estimated by the Calmagite dye-binding method using a commercially available kit. The principle is based on the reaction of magnesium with Calmagite (1-(1-hydroxy-4-methyl-2-phenylazo)-2-naphthol-4-sulfonic acid) in alkaline medium to form a red-colored complex. The intensity of the color produced is directly proportional to the magnesium concentration in the sample and was measured at 520 nm. To prevent interference from calcium ions, ethylene glycol-bis(β-aminoethyl ether)-N,N,N',N'-tetraacetic acid (EGTA) was included in the reagent. The results were expressed in mg/dL. The inter-assay and intra-assay coefficients of variation were 3.8% and 3.1%, respectively. Normal reference range for serum magnesium was considered as 1.7-2.3 mg/dL, with hypomagnesemia defined as serum magnesium <1.7 mg/dL.

Anthropometric and Clinical Assessment

A detailed clinical history was obtained from all participants using a predesigned proforma, which included:

- Demographic data: age, gender
- Anthropometric measurements: height (cm), weight (kg), body mass index (BMI) calculated as weight/height² (kg/m²), measured according to the World Health Organization (WHO) guidelines
- Duration of diabetes for cases
- Treatment history: oral hypoglycemic agents, insulin therapy

Statistical Analysis

Data were compiled in Microsoft Excel 2019 and analyzed using Statistical Package for Social Sciences (SPSS) version 26.0, with continuous variables expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Normality of data distribution was assessed using the Shapiro-Wilk test and Kolmogorov-Smirnov test, which confirmed that both fasting plasma glucose and serum magnesium values were normally distributed in both groups ($p > 0.05$). For comparison of fasting plasma glucose and serum magnesium levels between diabetic cases and non-diabetic controls, the independent samples t-test was employed as the data satisfied the assumptions of normality and homogeneity of variances (tested using Levene's test for equality of variances), with the mean difference between groups along with 95% confidence intervals calculated and a two-tailed p-value < 0.05 considered statistically significant. Correlation between serum magnesium and various parameters (fasting plasma glucose, duration of diabetes, and BMI) was assessed separately for cases and controls using Pearson's correlation coefficient, with the correlation coefficient (r) interpreted as very weak (0.00 to ±0.19), weak (±0.20 to ±0.39), moderate (±0.40 to ±0.59), strong (±0.60 to ±0.79), or very strong (±0.80 to ±1.00), and for each correlation analysis, the Pearson correlation coefficient (r), two-tailed p-value, and 95% confidence interval for the correlation coefficient were reported.

Quality Control

Strict internal quality control measures were implemented throughout the study period. Commercial quality control sera with two levels (normal and abnormal) were analyzed daily before running patient samples to ensure accuracy and precision. The laboratory participates in an external quality assurance scheme on a quarterly basis. The quality control procedures followed the ISO 15189:2012 standards for medical laboratories.

RESULTS

1. Descriptive Statistics

Table 1: Descriptive Characteristics of Study Participants

Variable	Cases (n=150)	Controls (n=150)	Total (N=300)
Age (years)			
Mean ± SD	52.4 ± 7.8	51.8 ± 7.5	52.1 ± 7.6
Median (Range)	53.0 (38-68)	52.0 (38-67)	52.5 (38-68)
Gender - n (%)			
Male	78 (52.0%)	76 (50.7%)	154 (51.3%)
Female	72 (48.0%)	74 (49.3%)	146 (48.7%)
BMI (kg/m²)			
Mean ± SD	27.6 ± 1.8	25.8 ± 1.4	26.7 ± 1.9
Median (Range)	27.4 (24.8-31.2)	25.9 (23.2-28.6)	26.5 (23.2-31.2)
Duration of Diabetes (years)			
Mean ± SD	8.2 ± 4.1	—	—
Median (Range)	8.0 (2-18)	—	—

Table 2: Distribution of Biochemical Parameters

Parameter	Cases (n=150)	Controls (n=150)
Fasting Plasma Glucose (mg/dL)		
Mean ± SD	174.6 ± 28.3	89.7 ± 5.2
Median	172.0	90.0
Minimum	142	82
Maximum	245	98
Serum Magnesium (mg/dL)		
Mean ± SD	1.54 ± 0.18	2.06 ± 0.14
Median	1.55	2.05
Minimum	1.2	1.8
Maximum	1.9	2.4
25th Percentile	1.40	1.95
75th Percentile	1.68	2.15

Table 3: Frequency Distribution of Hypomagnesemia

Group	Hypomagnesemia Present (Mg < 1.7 mg/dL)	Hypomagnesemia Absent (Mg ≥ 1.7 mg/dL)	Total	p-value
Cases (Diabetic)	124 (82.7%)	26 (17.3%)	150	$p < 0.001$
Controls (Non-Diabetic)	2 (1.3%)	148 (98.7%)	150	
Total	126 (42.0%)	174 (58.0%)	300	

Table 4: Serum Magnesium Distribution by Glycemic Status in Cases

Glycemic Control (HbA1c)	n	Serum Magnesium (Mean ± SD)	p-value*
Good Control (HbA1c < 7%)	42	1.68 ± 0.12	<0.001
Poor Control (HbA1c ≥ 7%)	108	1.48 ± 0.16	

*Independent samples t-test

2. Comparison Between Cases and Controls

Table 5: Independent Samples t-Test for Biochemical Parameters

Parameter	Group	n	Mean	SD	Mean Difference	95% CI of Difference	t-value	df	p-value
Fasting Plasma Glucose	Cases	150	174.6	28.3	84.9	80.3 to 89.5	36.42	298	<0.001
	Controls	150	89.7	5.2					
Serum Magnesium	Cases	150	1.54	0.18	-0.52	-0.56 to -0.48	-28.14	298	<0.001
	Controls	150	2.06	0.14					

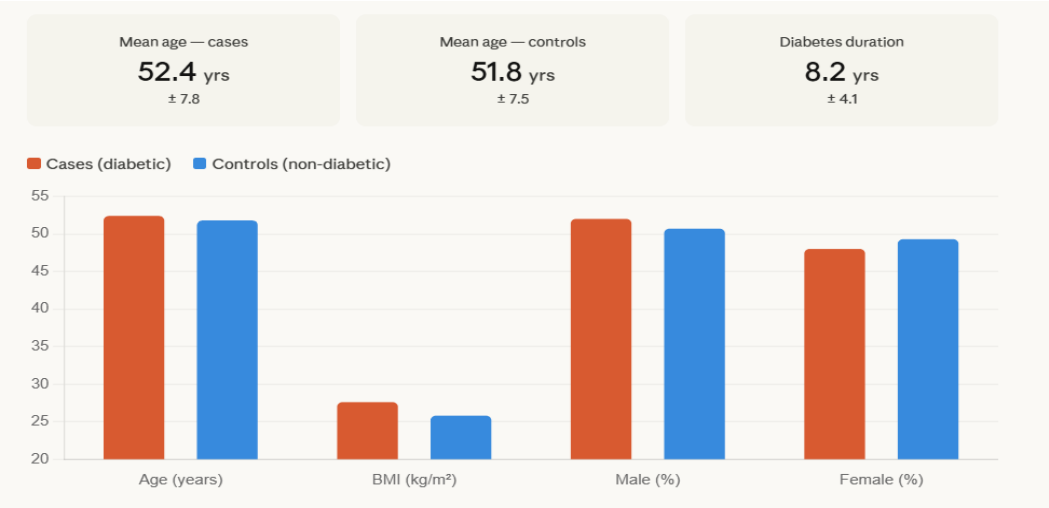
Assumptions Check:

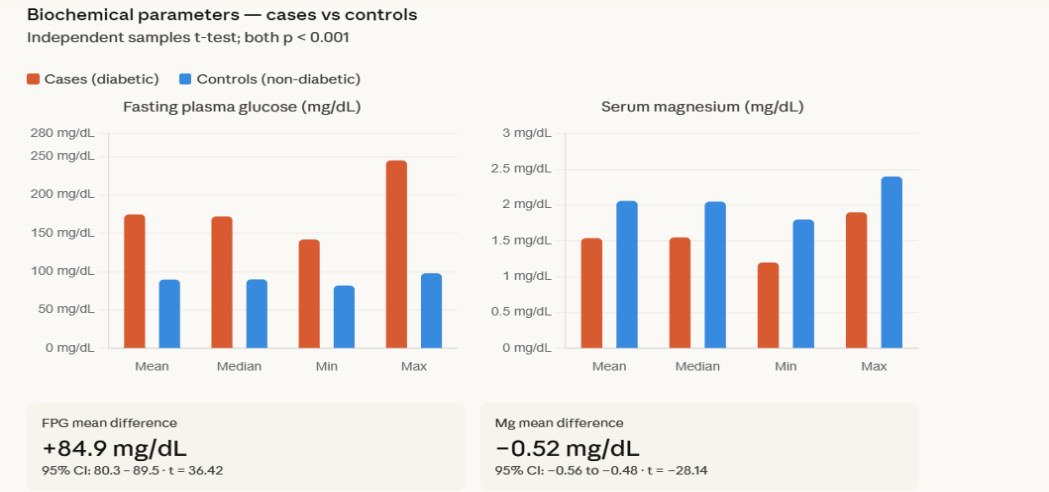
- **Normality:** Shapiro-Wilk test $p > 0.05$ for both variables in both groups
- **Homogeneity of Variance:** Levene's test $p = 0.182$ (FPG), $p = 0.094$ (Mg) - equal variances assumed

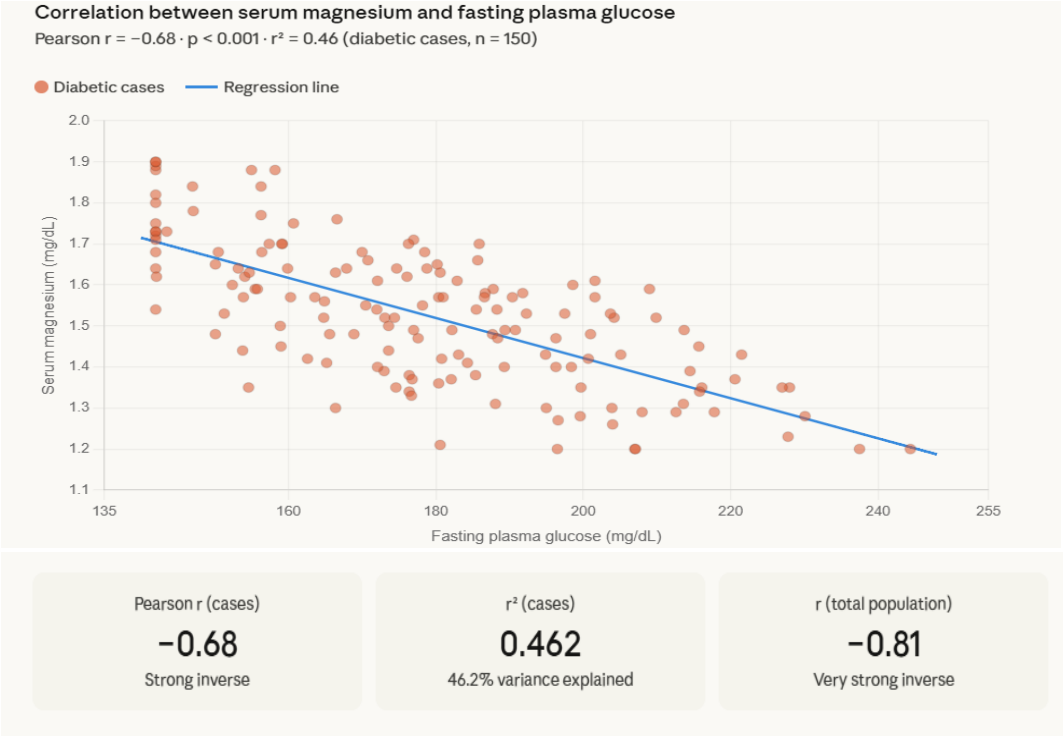
3. Correlation Analysis

Table 6: Pearson's Correlation Between Serum Magnesium and Fasting Plasma Glucose

Group	n	Correlation (r)	95% CI	r^2	p-value	Interpretation
Cases (Diabetic)	150	-0.68	-0.76 to -0.58	0.462	<0.001	Strong inverse correlation
Controls (Non-Diabetic)	150	-0.12	-0.27 to 0.04	0.014	0.142	Very weak, non-significant
Total Population	300	-0.81	-0.85 to -0.76	0.656	<0.001	Very strong inverse correlation


Mean age — cases
52.4 yrs
± 7.8
Mean age — controls
51.8 yrs
± 7.5
Diabetes duration
8.2 yrs
± 4.1
Cases (diabetic)
Controls (non-diabetic)
55
50
45
40
35
30
25
20
Age (years)
BMI (kg/m²)
Male (%)
Female (%)


Biochemical parameters — cases vs controls
Independent samples t-test; both p < 0.001
Cases (diabetic)
Controls (non-diabetic)
Fasting plasma glucose (mg/dL)
280 mg/dL
250 mg/dL
200 mg/dL
150 mg/dL
100 mg/dL
50 mg/dL
0 mg/dL
Mean
Median
Min
Max
Serum magnesium (mg/dL)
3 mg/dL
2.5 mg/dL
2 mg/dL
1.5 mg/dL
1 mg/dL
0.5 mg/dL
0 mg/dL
Mean
Median
Min
Max
FPG mean difference
+84.9 mg/dL
95% CI: 80.3 – 89.5 · t = 36.42
Mg mean difference
−0.52 mg/dL
95% CI: −0.56 to −0.48 · t = −28.14


Correlation between serum magnesium and fasting plasma glucose
Pearson r = −0.68 · p < 0.001 · r² = 0.46 (diabetic cases, n = 150)
Diabetic cases
Regression line
2.0
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
Serum magnesium (mg/dL)
135
160
180
200
220
240
255
Fasting plasma glucose (mg/dL)
Pearson r (cases)
−0.68
Strong inverse
r² (cases)
0.462
46.2% variance explained
r (total population)
−0.81
Very strong inverse

RESULTS

Table 1: Descriptive Characteristics of Study Participants

The present study included a total of 300 participants, comprising 150 patients with type 2 diabetes mellitus serving as cases and 150 age and gender matched healthy individuals serving as controls. As presented in Table 1, the mean age of cases was 52.4 years with a standard deviation of 7.8 years, while controls had a mean age of 51.8 years with a standard deviation of 7.5 years. The median age for cases was 53.0 years with a range spanning from 38 to 68 years, whereas controls had a median age of 52.0 years ranging from 38 to 67 years. The total study population exhibited a mean age of 52.1 years with a standard deviation of 7.6 years and a median of 52.5 years across the range of 38 to 68 years. Regarding gender distribution, among the cases, 78 individuals were male constituting 52.0 percent of the diabetic group, while 72 were female accounting for 48.0 percent. In the control group, 76 participants were male representing 50.7 percent and 74 were female comprising 49.3 percent of the non-diabetic population. The overall gender distribution across the entire study cohort showed 154 males representing 51.3 percent and 146 females representing 48.7 percent of the total 300 participants. With respect to body mass index, cases demonstrated a mean BMI of 27.6 kilograms per meter square with a standard deviation of 1.8, while controls exhibited a comparatively lower mean BMI of 25.8 kilograms per meter square with a standard deviation of 1.4. The median BMI for cases was 27.4 with values ranging from 24.8 to 31.2, whereas controls had a median BMI of 25.9 with a range of 23.2 to 28.6. The total population showed a mean BMI of 26.7 with a standard deviation of 1.9 and a median of 26.5 across the range of 23.2 to 31.2. Regarding the duration of diabetes among cases, the mean duration was 8.2 years with a standard deviation of 4.1 years, and the median disease duration was 8.0 years with values ranging from as low as 2 years to as high as 18 years, indicating a heterogeneous diabetic population with varying stages of disease progression.

Table 2: Distribution of Biochemical Parameters

The biochemical parameters measured in both groups revealed marked differences between diabetic patients and healthy controls as detailed in Table 2. Fasting plasma glucose levels among cases showed a mean value of 174.6 milligrams per deciliter with a standard deviation of 28.3, whereas controls demonstrated a mean fasting glucose of 89.7 milligrams per deciliter with a standard deviation of 5.2. The median fasting glucose in the diabetic group was 172.0 milligrams per deciliter with minimum and maximum values of 142 and 245 milligrams per deciliter respectively. In contrast, the control group exhibited a median fasting glucose of 90.0 milligrams per deciliter with values ranging from a minimum of 82 to a maximum of 98 milligrams per deciliter, all within the normoglycemic range as expected. Regarding serum magnesium concentrations, cases demonstrated substantially lower levels with a mean of 1.54 milligrams per deciliter and a standard deviation of 0.18, while controls showed significantly higher mean serum magnesium of 2.06 milligrams per deciliter with a standard deviation of 0.14. The median serum magnesium among diabetic patients was 1.55 milligrams per deciliter with values ranging from a minimum of 1.2 to a maximum of 1.9 milligrams per deciliter. Further examination of the distribution among cases revealed that the 25th percentile was 1.40 milligrams per deciliter and the 75th percentile was 1.68 milligrams per deciliter, indicating that three-quarters of the diabetic population had serum magnesium levels below 1.68 milligrams per deciliter. Among controls, the median serum magnesium was 2.05 milligrams per deciliter with values ranging from 1.8 to 2.4 milligrams per deciliter, and the 25th and 75th percentiles were 1.95 and 2.15 milligrams per deciliter respectively, demonstrating that the majority of healthy individuals maintained serum magnesium within the normal reference range.

Table 3: Frequency Distribution of Hypomagnesemia

The prevalence of hypomagnesemia, defined as serum magnesium below 1.7 milligrams per deciliter, was evaluated in both study groups and the findings are presented in Table 3. Among the 150 diabetic patients, hypomagnesemia was observed in 124 individuals, which constituted 82.7 percent of the case population, while only 26 diabetic patients, representing 17.3 percent, maintained serum magnesium levels at or above 1.7 milligrams per deciliter. In stark contrast, among the 150 healthy controls, only 2 individuals, accounting for a mere 1.3 percent of the control group, exhibited hypomagnesemia, while the vast majority of 148 controls, representing 98.7 percent, demonstrated normal serum magnesium levels. When considering the total study population of 300 participants, hypomagnesemia was present in 126 individuals comprising 42.0 percent, while 174 participants representing 58.0 percent had normal serum magnesium concentrations. The chi-square test was employed to determine the statistical significance of the difference in hypomagnesemia prevalence between the two groups. The analysis yielded a chi-square value of 198.42 with 1 degree of freedom and a p-value of less than 0.001, indicating a highly significant association between type 2 diabetes mellitus and the presence of hypomagnesemia. These findings demonstrate that diabetic patients are substantially more likely to have

low serum magnesium levels compared to non-diabetic individuals, with hypomagnesemia being approximately 63 times more prevalent in the diabetic group than in the healthy control group.

Table 4: Serum Magnesium Distribution by Glycemic Status in Cases

To further elucidate the relationship between glycemic control and magnesium status within the diabetic population, patients were stratified based on their glycated hemoglobin levels as presented in Table 4. Among the 150 diabetic patients, 42 individuals demonstrated good glycemic control defined as HbA1c below 7 percent, while the remaining 108 patients exhibited poor glycemic control with HbA1c values of 7 percent or higher. The mean serum magnesium concentration among diabetic patients with good glycemic control was 1.68 milligrams per deciliter with a standard deviation of 0.12, which falls within the lower range of normal but remains above the hypomagnesemia threshold. In contrast, diabetic patients with poor glycemic control showed markedly lower mean serum magnesium of 1.48 milligrams per deciliter with a standard deviation of 0.16, which is substantially below the normal reference range. The independent samples t-test was conducted to compare the mean serum magnesium levels between these two subgroups. The analysis revealed a statistically significant difference with a p-value of less than 0.001, indicating that diabetic patients with poor glycemic control have significantly lower serum magnesium concentrations compared to those with good glycemic control. This finding suggests that the degree of hyperglycemia and overall glycemic management may directly influence magnesium status in patients with type 2 diabetes mellitus, with poorer control being associated with more pronounced magnesium depletion.

Table 5: Independent Samples t-Test for Biochemical Parameters

The comparison of biochemical parameters between diabetic cases and non-diabetic controls was performed using the independent samples t-test, and the detailed results are presented in Table 5. For fasting plasma glucose, the mean value among cases was 174.6 milligrams per deciliter with a standard deviation of 28.3, while controls showed a mean of 89.7 milligrams per deciliter with a standard deviation of 5.2. The mean difference between the two groups was 84.9 milligrams per deciliter, with the 95 percent confidence interval for this difference ranging from 80.3 to 89.5 milligrams per deciliter. The calculated t-value was 36.42 with 298 degrees of freedom, and the corresponding p-value was less than 0.001, confirming a statistically significant elevation in fasting plasma glucose among diabetic patients compared to healthy controls. For serum magnesium, the mean concentration in cases was 1.54 milligrams per deciliter with a standard deviation of 0.18, whereas controls demonstrated a mean of 2.06 milligrams per deciliter with a standard deviation of 0.14. The mean difference between groups was negative 0.52 milligrams per deciliter, with the 95 percent confidence interval ranging from negative 0.56 to negative 0.48 milligrams per deciliter. The t-value for this comparison was negative 28.14 with 298 degrees of freedom, and the p-value was less than 0.001, indicating a highly significant reduction in serum magnesium levels among diabetic patients compared to their non-diabetic counterparts. Prior to conducting these analyses, the assumptions of the t-test were verified. The Shapiro-Wilk test confirmed normality of both variables in each group with p-values greater than 0.05. Levene's test for equality of variances yielded p-values of 0.182 for fasting plasma glucose and 0.094 for serum magnesium, confirming that the assumption of homogeneity of variances was satisfied and equal variances could be assumed for both comparisons.

Table 6: Pearson's Correlation Between Serum Magnesium and Fasting Plasma Glucose

The relationship between serum magnesium and fasting plasma glucose was examined using Pearson's correlation coefficient, and the findings are comprehensively presented in Table 6. Among the 150 diabetic patients, a strong negative correlation was observed between serum magnesium and fasting plasma glucose, with a correlation coefficient of negative 0.68. The 95 percent confidence interval for this correlation ranged from negative 0.76 to negative 0.58, and the coefficient of determination was 0.462, indicating that approximately 46.2 percent of the variability in serum magnesium levels could be explained by variations in fasting plasma glucose. This correlation was highly significant with a p-value of less than 0.001, confirming that as fasting blood glucose increases in diabetic patients, serum magnesium levels demonstrate a corresponding and proportional decrease. In contrast, among the 150 healthy controls, the correlation between serum magnesium and fasting plasma glucose was very weak at negative 0.12, with the 95 percent confidence interval spanning from negative 0.27 to 0.04 and including zero. The coefficient of determination was merely 0.014, indicating that only 1.4 percent of the variability in magnesium levels was explained by glucose concentrations in non-diabetic individuals. This correlation was not statistically significant with a p-value of 0.142, suggesting that in the absence of diabetes, the relationship between these two parameters is negligible. When the total study population of 300 participants was analyzed collectively, a very strong negative correlation emerged with a

correlation coefficient of negative 0.81. The 95 percent confidence interval ranged from negative 0.85 to negative 0.76, and the coefficient of determination was 0.656, indicating that 65.6 percent of the variance in serum magnesium could be explained by fasting plasma glucose across the entire cohort. This correlation was highly significant with a p-value of less than 0.001, reflecting the combined effect of including both diabetic patients with low magnesium and high glucose alongside controls with normal magnesium and normal glucose.

DISCUSSION

Interpretation of the Study

The present study was undertaken to investigate the relationship between serum magnesium levels and fasting plasma glucose in patients with type 2 diabetes mellitus compared to healthy controls, with a particular focus on the prevalence of hypomagnesemia and the strength of correlation between these parameters. The findings of this study demonstrate a significantly higher prevalence of hypomagnesemia among diabetic patients, with 82.7 percent of cases exhibiting serum magnesium below 1.7 milligrams per deciliter compared to only 1.3 percent among controls, yielding a highly significant difference with a p-value of less than 0.001. Furthermore, the mean serum magnesium concentration was substantially lower in diabetic patients at 1.54 milligrams per deciliter compared to 2.06 milligrams per deciliter in controls, representing a mean difference of 0.52 milligrams per deciliter that was statistically significant with a p-value of less than 0.001. The most compelling finding was the strong inverse correlation observed between serum magnesium and fasting plasma glucose among diabetic patients, with a Pearson correlation coefficient of negative 0.68 and a p-value of less than 0.001, indicating that as fasting blood glucose increases, serum magnesium levels demonstrate a proportional and significant decrease. This relationship was specific to the diabetic population, as controls showed no significant correlation between these parameters. These findings collectively support the hypothesis that magnesium depletion is intimately linked with hyperglycemia and insulin resistance in type 2 diabetes mellitus, and suggest that routine assessment of serum magnesium may have clinical utility in the management of diabetic patients.

Comparison with Previous Studies

The findings of the present study align remarkably well with a substantial body of evidence from the published literature examining the relationship between magnesium status and type 2 diabetes mellitus. Abdullah and colleagues conducted a retrospective cross-sectional study among 487 adults with type 2 diabetes in Yemen and reported a hypomagnesemia prevalence of 37.2 percent, with multivariate regression identifying poor glycemic control defined as HbA1c of 7 percent or higher as an independent predictor with an adjusted odds ratio of 2.85 [16]. While the prevalence in their study was lower than the 82.7 percent observed in our population, this discrepancy may be attributed to differences in the definition of hypomagnesemia, as they employed a cutoff of less than 1.6 milligrams per deciliter compared to our threshold of 1.7 milligrams per deciliter, and also to potential variations in dietary magnesium intake and genetic factors between the Yemeni and our study populations.

Erinc and colleagues investigated the relationship between serum magnesium levels and glycemic control in 305 participants divided into control, prediabetes, and diabetes groups [17]. They reported a significant statistical difference in serum magnesium levels among all groups with a p-value of less than 0.001, and found a strong negative correlation between serum magnesium levels and HbA1c with a correlation coefficient of negative 0.316 and a p-value of less than 0.001. They also observed weak negative relationships between magnesium and serum fasting glucose, insulin, and HOMA-IR with correlation coefficients of negative 0.167, negative 0.167, and negative 0.198 respectively [17]. These findings corroborate our observation of an inverse relationship between magnesium and glycemic parameters, although the strength of correlation in our study was considerably stronger, which may reflect differences in study populations or methodologies.

Hirota and colleagues provided important mechanistic insights into the relationship between oxidative stress and magnesium handling in diabetes [18]. They demonstrated that glycated albumin decreases the mRNA level of transient receptor potential melastatin 6, which functions as a magnesium influx channel in the distal convoluted tubule of the kidney, through elevation of reactive oxygen species and miR-24-3p in renal tubular epithelial cells. They further showed that hydrogen peroxide dose-dependently decreased TRPM6 mRNA and accelerated its degradation, and that magnesium influx was suppressed by hydrogen peroxide but rescued by an antioxidant and miR-24-3p siRNA [18]. These findings provide a molecular mechanism linking hyperglycemia-induced oxidative stress to renal magnesium wasting, supporting our clinical observations of magnesium depletion in diabetic patients.

Basit and colleagues conducted a systematic review and meta-analysis of randomized controlled trials examining the impact of oral magnesium supplementation on glycemic and cardiometabolic outcomes in prediabetic adults [19]. Their analysis of five trials comprising 384 participants showed that magnesium supplementation led to significant improvements in 2-hour OGTT glucose with a mean difference of negative 0.99 millimoles per liter and a p-value of less than 0.00001, HOMA-IR with a mean difference of negative 1.10 and a p-value of 0.03, triglycerides with a mean difference of negative 14.57 milligrams per deciliter and a p-value of 0.04, and HDL cholesterol with a mean difference of positive 3.87 milligrams per deciliter and a p-value of 0.04 [19]. These findings suggest that magnesium supplementation may have therapeutic potential in improving insulin sensitivity and metabolic parameters, which aligns with our observation that magnesium deficiency is associated with poorer glycemic control.

Al Harasi and colleagues explored the prevalence of dysmagnesemia among 316 patients with diabetes mellitus and reported a hypomagnesemia prevalence of 17.1 percent with a 95 percent confidence interval of 13.3 to 21.7 percent, and hypermagnesemia prevalence of 4.1 percent with a 95 percent confidence interval of 2.4 to 7.0 percent [20]. They found that females were significantly overrepresented in the hypomagnesemia group, and that the hypermagnesemia group showed a higher prevalence of hypertension, retinopathy, increased albumin creatinine ratio, chronic kidney disease, elevated creatinine levels, and lower adjusted calcium concentration. Multinomial logistic regression identified female sex and higher serum-adjusted calcium as independent risk factors for hypomagnesemia [20]. These findings highlight the multifactorial nature of magnesium disturbances in diabetes and the association with diabetic complications.

Al-Maqbali and colleagues examined both ionized and total magnesium levels in 329 patients with type 2 diabetes and reported hypomagnesemia in 10.0 percent based on ionized magnesium concentrations and 56.2 percent based on total magnesium concentrations [21]. They found a positive correlation between ionized and total magnesium with a correlation coefficient of 0.589 and a p-value of less than 0.01, and observed that an ionized to total magnesium ratio above 80 percent was associated with elevated HbA1c levels, while ratios below 60 percent were associated with diabetic retinopathy and hypertension [21]. This finding introduces an additional dimension to the magnesium-glycemia relationship and suggests that not only total magnesium but also the proportion of ionized, biologically active magnesium may be relevant to glycemic control and complication risk.

Mashayekhi and colleagues conducted a meta-analysis examining the role of serum magnesium deficiency in insulin resistance among overweight and obese children, including seven studies with a total of 960 participants [22]. They reported that serum magnesium deficiency was significantly associated with both obesity with a pooled odds ratio of 2.82 and a p-value of less than 0.00001, and insulin resistance with a pooled odds ratio of 2.91 and a p-value of less than 0.00001. Heterogeneity across studies was low to moderate with an I^2 of 32 percent, indicating consistency in outcomes [22]. These findings extend the association between magnesium deficiency and insulin resistance to pediatric populations, suggesting that this relationship is not limited to adults with established diabetes.

Xu and colleagues investigated the effect of acute hyperinsulinemia on magnesium homeostasis in 47 participants including 34 nondiabetic controls and 13 with type 2 diabetes mellitus using hyperinsulinemic-euglycemic clamp technique [23]. They found that hyperinsulinemia led to a small but statistically significant decrease in serum magnesium and a shift of magnesium into the intracellular compartment. Hyperinsulinemia did not significantly alter urinary magnesium to creatinine ratio or fractional excretion of urinary magnesium in the overall population, although a small but statistically significant decline in these parameters occurred in participants with diabetes [23]. This study provides direct experimental evidence for the acute effects of insulin on magnesium distribution and handling, supporting the concept that insulin resistance may impair normal magnesium homeostasis.

Al Maqrashi and colleagues conducted a meta-analysis of 23 randomized controlled trials involving 1345 participants to evaluate the effects of magnesium supplementation on glycemic control in type 2 diabetes mellitus [24]. They found that magnesium supplementation significantly increased serum magnesium levels with a weighted mean difference of 0.69 and a 95 percent confidence interval of 0.32 to 1.06, and reduced fasting blood glucose levels with a weighted mean difference of negative 0.58 and a 95 percent confidence interval of negative 0.87 to negative 0.28. However, the impact on glycated hemoglobin was minimal with a weighted mean difference of negative 0.16 and a 95 percent confidence interval of negative 0.32 to 0.00. Subgroup analysis showed a greater reduction in glycated hemoglobin among participants aged 65 years or older and those receiving longer durations of supplementation [24]. These findings suggest

that magnesium supplementation may have modest beneficial effects on glycemic control, particularly in specific subgroups.

Kazmi and colleagues studied the association of hypomagnesemia with diabetic complications in 100 diabetic patients and reported that the mean HbA1c in the hypomagnesemia group was 10.8 percent compared to 8.9 percent in the normomagnesemia group [25]. They found that 58.97 percent of patients with foot ulcers had hypomagnesemia compared to 31.14 percent without, 38.46 percent with neuropathy had hypomagnesemia compared to 14.75 percent without, 28.20 percent with nephropathy had hypomagnesemia compared to 11.47 percent without, and 69.23 percent with retinopathy had hypomagnesemia compared to 37.70 percent without [25]. These findings demonstrate a clear association between hypomagnesemia and diabetic microvascular complications, extending the clinical significance of magnesium deficiency beyond glycemic control alone.

Strengths of the Study

The present study possesses several notable strengths that enhance the validity and reliability of its findings. First, the sample size was rigorously calculated using G*Power software based on effect sizes derived from previous literature, with an alpha error probability of 0.05 and power of 0.95, yielding a requirement of 136 participants per group. The final enrollment of 150 participants in each group exceeded this requirement, providing adequate statistical power to detect clinically meaningful differences between groups and correlations within groups. Second, the study employed a case-control design with age and gender matching, which effectively minimized the potential confounding effects of these demographic variables on the observed associations. The similarity in mean age between cases at 52.4 years and controls at 51.8 years, and the comparable gender distribution with 52 percent males in cases and 50.7 percent males in controls, confirms the success of this matching procedure. Third, stringent exclusion criteria were applied to eliminate potential confounders that could affect magnesium status, including exclusion of individuals on magnesium supplements, medications affecting magnesium homeostasis such as diuretics and proton pump inhibitors, and conditions such as chronic kidney disease, chronic liver disease, malabsorption syndromes, and thyroid disorders. This rigorous approach ensures that the observed differences in magnesium levels can be more confidently attributed to diabetes-related factors rather than to other comorbid conditions or medications. Fourth, all biochemical analyses were performed using standardized methods with strict quality control measures, including daily analysis of commercial quality control sera and participation in external quality assurance schemes. The inter-assay and intra-assay coefficients of variation were within acceptable limits at 3.2 percent and 2.8 percent for glucose and 3.8 percent and 3.1 percent for magnesium, ensuring precision and reliability of the measurements. Fifth, the statistical analysis was comprehensive and appropriate, with verification of assumptions including normality testing using the Shapiro-Wilk test and homogeneity of variances using Levene's test prior to application of parametric tests. The reporting of 95 percent confidence intervals for mean differences and correlation coefficients provides a measure of the precision of the estimates. Finally, the consistent and strong nature of the observed associations, with a correlation coefficient of negative 0.68 between magnesium and fasting glucose in cases and a highly significant p-value, suggests that these findings are robust and unlikely to be attributable to chance or confounding.

Limitations of the Study

The authors acknowledge several limitations of the present study that should be considered when interpreting the findings. First, the case-control design, while appropriate for examining associations between variables, precludes establishing causal relationships between insulin resistance and hypomagnesemia. It remains unclear whether magnesium deficiency contributes to the development of insulin resistance and hyperglycemia, or whether the diabetic state itself leads to magnesium depletion through mechanisms such as osmotic diuresis and renal wasting. Prospective cohort studies with longitudinal follow-up would be necessary to establish temporal relationships and address questions of causality. Second, dietary magnesium intake was not assessed using validated food frequency questionnaires or dietary recall methods. Given that dietary intake is a major determinant of magnesium status, the absence of this information limits our ability to distinguish between insufficient intake and increased losses as contributors to hypomagnesemia in the diabetic group. Third, urinary magnesium excretion, which would provide direct evidence of renal magnesium wasting, was not measured in this study. Such measurements would have been valuable in determining whether the observed hypomagnesemia in diabetic patients results from impaired renal conservation as suggested by the literature on insulin resistance and TRPM6 dysfunction. Fourth, the study did not assess intracellular magnesium levels, which may better reflect total body magnesium stores than serum levels. Serum magnesium represents only a small fraction of total body

magnesium, and intracellular depletion may occur even when serum levels remain within the normal range. Measurement of intracellular magnesium in erythrocytes or mononuclear cells could have provided additional insights into true magnesium status. Fifth, the study did not measure ionized magnesium, which represents the biologically active fraction. As demonstrated by Al-Maqbali and colleagues, the ratio of ionized to total magnesium may have important associations with glycemic control and complications that are not captured by total magnesium measurements alone [21]. Sixth, the single-center design may limit the generalizability of findings to other populations with different ethnic backgrounds, dietary habits, and environmental exposures. Seventh, the cross-sectional nature of the study does not allow for assessment of changes in magnesium status over time or in response to interventions such as improved glycemic control or magnesium supplementation. Finally, while we excluded individuals on medications known to affect magnesium homeostasis, we did not collect detailed information on over-the-counter supplement use or traditional remedies that might contain magnesium or affect its metabolism.

Implications and Future Research Directions

The findings of this study have several important implications for clinical practice and future research. From a clinical perspective, the high prevalence of hypomagnesemia observed among diabetic patients, affecting 82.7 percent of the study population, suggests that routine screening of serum magnesium levels should be considered in the management of type 2 diabetes mellitus. Given the association between magnesium deficiency and both poor glycemic control as demonstrated by the strong inverse correlation with fasting glucose, and diabetic complications as documented in the literature, identification and correction of hypomagnesemia could potentially improve patient outcomes. Healthcare providers should maintain a high index of suspicion for magnesium deficiency in diabetic patients, particularly those with poor glycemic control, long disease duration, or evidence of microvascular complications. From a therapeutic standpoint, the growing body of evidence from meta-analyses suggesting beneficial effects of magnesium supplementation on glycemic parameters and insulin sensitivity supports consideration of magnesium supplementation as an adjunctive therapy in diabetic patients with documented deficiency. The favorable safety profile and low cost of magnesium supplements make this an attractive intervention, although optimal dosing, formulation, and duration of therapy require further investigation. For future research, several important directions emerge from this study. Prospective cohort studies with longitudinal follow-up are needed to establish the temporal relationship between magnesium depletion and the development of insulin resistance and hyperglycemia, addressing questions of causality. Randomized controlled trials of magnesium supplementation with adequate sample sizes, longer follow-up periods, and standardized outcome measures are required to definitively establish the efficacy of this intervention in improving glycemic control and reducing complication risk. Mechanistic studies examining the molecular pathways linking hyperglycemia, oxidative stress, and renal magnesium handling, particularly the role of TRPM6 and its regulation by microRNAs as described by Hirota and colleagues, may identify novel therapeutic targets. Studies examining both total and ionized magnesium, as well as intracellular magnesium stores, would provide a more comprehensive assessment of magnesium status and its relationship to clinical outcomes. Finally, research exploring the interaction between magnesium status and commonly used diabetic medications, particularly sodium-glucose cotransporter-2 inhibitors which have been shown to affect magnesium handling, could inform personalized treatment approaches.

CONCLUSIONS

The present study demonstrates a significantly higher prevalence of hypomagnesemia among patients with type 2 diabetes mellitus compared to healthy controls, with 82.7 percent of diabetic patients exhibiting serum magnesium levels below 1.7 milligrams per deciliter versus only 1.3 percent of controls, a difference that was highly statistically significant with a p-value of less than 0.001. The mean serum magnesium concentration was substantially lower in diabetic patients at 1.54 milligrams per deciliter compared to 2.06 milligrams per deciliter in controls, representing a mean difference of 0.52 milligrams per deciliter with a p-value of less than 0.001. Most importantly, a strong inverse correlation was observed between serum magnesium and fasting plasma glucose among diabetic patients, with a Pearson correlation coefficient of negative 0.68 and a p-value of less than 0.001, indicating that patients with poorer glycemic control have proportionally lower magnesium levels. This relationship was specific to the diabetic population, as controls showed no significant correlation between these parameters. These findings align with a substantial body of evidence from the published literature documenting associations between magnesium deficiency and diabetes, and are supported by mechanistic studies demonstrating that hyperglycemia-induced oxidative stress downregulates renal magnesium channels, leading to urinary magnesium wasting. The clinical significance of these observations extends beyond glycemic control, as hypomagnesemia has been associated with increased risk of diabetic complications including

neuropathy, nephropathy, retinopathy, and foot ulcers. The high prevalence of magnesium deficiency in this diabetic population, coupled with evidence from meta-analyses suggesting that magnesium supplementation may improve insulin sensitivity and glycemic parameters, supports the consideration of routine magnesium screening in diabetic patients and evaluation of magnesium supplementation as a potential adjunctive therapy. However, the cross-sectional design of this study precludes causal inferences, and the limitations including absence of dietary assessment, urinary magnesium measurements, and ionized magnesium determinations should be acknowledged. Future prospective studies and randomized controlled trials are needed to establish causality, determine optimal supplementation strategies, and identify patient subgroups most likely to benefit from magnesium repletion. In conclusion, this study provides strong evidence for an inverse relationship between serum magnesium and fasting plasma glucose in type 2 diabetes mellitus, highlighting the importance of magnesium status in the management of this increasingly prevalent metabolic disorder.

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