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ORIGINAL ARTICLE

Association between Serum Magnesium Levels and Glycemic Control (HbA1c) in Patients with Type 2 Diabetes Mellitus in Private Clinics in Baghdad City

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ABSTRACT

Magnesium is important for insulin-mediated cellular glucose uptake and for many of the various enzymatic reactions that are involved with glucose metabolism. Hypomagnesemia is increasingly thought to be common among patients with type 2 diabetes mellitus (T2DM) and to affect glycemic control adversely. The aim of this study is to evaluate serum magnesium levels among T2DM patients who were seen in private medical clinics in Baghdad, Iraq, and their relationship with the patients' glycemic control as evaluated using HbA1c. A cross-sectional analytical study was performed from January to December 2024, at six clinics in Baghdad, enrolling 200 patients (type 2 diabetes mellitus). Serum magnesium was measured using an xylidyl blue colourimetric method, while HbA1c was determined using high-performance liquid chromatography (HPLC). Data analysis was performed using Pearson correlation, one-way ANOVA, and binary logistic regression. The mean serum magnesium was 0.68 ± 0.12 mmol/L, with 112 (56%) of the patients having hypomagnesemia (<0.75 mmol/L). The mean HbA1c of the patients was 8.9 ± 1.6%. A significant inverse correlation was found between serum magnesium and HbA1c ($r = -0.52$, $p < 0.001$), with the level of serum magnesium in patients who were poorly controlled (HbA1c ≥ 9%) was 0.59±0.08 compared to those who were well controlled (0.81±0.07 mmol/L, $p < 0.001$). Patients with poorly controlled diabetes, who have had diabetes for >10 years, or who use insulin, are independent predictors of hypomagnesemia. Hypomagnesemia is prevalent in T2DM patients in Baghdad; hypomagnesemia is associated with poor glycemic control. Routine screening for serum magnesium and providing nutritional advice should be included in the overall management of patients with T2DM.

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1- INTRODUCTION

According to the International Diabetes Federation (2021), in 2021, around 537 million adults lived with type 2 diabetes mellitus (T2DM), making it one of the most common chronic diseases in the world. By 2045, it is anticipated that this number will grow to 783 million people. T2DM is principally characterised by progressive insulin resistance and, at the same time, a relative deficiency of endogenous insulin, resulting in ongoing hyperglycemia that, over time, causes progressive injury to the vascular system, kidneys, peripheral nerves, and retina [1]. The greatest public health issue related to T2DM occurs in the Middle East and North Africa (MENA), where the rate of type 2 diabetes mellitus is the highest in the world when adjusted for age [2,3].

Iraq has one of the worst rates of diabetes in the world, and the capital city of Baghdad is especially affected; the national and regional surveys have demonstrated that over 12-15% of adults in Baghdad have diabetes, largely as the result of rapid urbanization, dietary transition to energy-dense foods that are highly refined, increasing obesity, and sedentary lifestyles [4]. Private diabetes and internal medicine clinics provide a major proportion of healthcare within Baghdad. Still, patients attending these clinics have not been adequately evaluated regarding the role of nutrition and metabolic factors in glycemic control. HbA1c is a widely accepted biomarker of long-term glycemic control. Scientists have determined that the level of HbA1c within an individual's bloodstream represents the average plasma glucose concentration over the past 8 to 12 weeks [5]. Additionally, HbA1c is an important diagnostic tool for T2DM and the most reliable predictor of T2DM-associated complications (i.e., diabetic nephropathy, retinopathy, neuropathy, and heart disease) [1]. Even with various diabetes medications, getting and keeping A1C at an optimal level (a reading of 7.0% or less) continues to be difficult in practice. This is particularly true in resource-poor communities, where issues such as medication adherence, access to specialist care, dietary advice, and the ability to monitor blood sugar levels regularly may be limited. Therefore, one of the priorities for improving clinical outcomes in populations with diabetes is identifying modifiable biochemical and nutritional factors that contribute to abnormal blood sugar levels [3].

Magnesium is the body's 4th most abundant mineral and the 2nd most prevalent intracellular cation; it acts as a required co-activator for over 300 enzymatic reactions in the human body, including those that control protein synthesis, nucleic acid metabolism, energy transduction and most importantly carbohydrate metabolism [6]. Magnesium is required for the proper functioning of the insulin receptor (tyrosine kinase) during glucose metabolism. This is the initiator of the insulin signalling pathway necessary for the disposal of glucose in peripheral tissues [7]. Several mechanisms contribute to hypomagnesemia: chronic hyperglycemia leads to osmotic diuresis and renal tubular wasting of magnesium; autonomic neuropathy reduces intestinal absorption of magnesium; and insulin resistance alters renal reabsorption of magnesium. In the setting of insulin resistance, loss of insulin's physiological effect on distal tubular magnesium reabsorption also contributes to diuresis and, thus, hypomagnesemia, creating a self-perpetuating pathophysiological cycle [8].

Several studies have confirmed the association between low magnesium levels and poor glycemic control. Likewise, Guerrero-Romero and Rodríguez-Morán (2011) reported that magnesium supplementation improved pancreatic beta-cell secretory function and insulin sensitivity indices in patients with chronic hypomagnesemia [9]. Multiple meta-analyses that combine results from many prospective cohort studies confirm a statistically significant inverse association between dietary magnesium intake and the incidence of T2DM [10]. Mechanistically, magnesium deficiency can lead to increased systemic inflammation and endothelial dysfunction through the activation of the NF-κB signalling pathway and upregulation of pro-inflammatory cytokines such as interleukin-6 and tumour necrosis factor-alpha, which have both been shown to contribute to insulin resistance and beta cell dysfunction [11].

De Baaij *et al*. (2015) [12], noted that magnesium deficiency can impair the expression of transient receptor potential melastatin 6 (TRPM6) channels in the distal convoluted tubules of the kidney, leading to increased urinary magnesium wasting and contributing to the pathophysiological cycle of hypomagnesemia and poor metabolic health. The findings from in vitro, animal, and human studies support the conclusion that hypomagnesemia is both a consequence and an exacerbator of metabolic dysregulation in T2DM. Despite the growing body of literature on hypomagnesemia and T2DM, there are currently no consistent protocols in place for clinicians to measure serum magnesium levels in T2DM patients in Iraq routinely. Furthermore, there are no data on the prevalence of hypomagnesemia and its relationship with glycemic control in T2DM patients living in Baghdad. The current study will therefore assess serum magnesium levels in T2DM patients receiving care at private clinics in Baghdad and examine the relationship between serum magnesium concentrations and HbA1c, an indicator of glycemic control, to provide a regional evidence base for developing clinical guidelines for diabetes management in Iraq.

2- MATERIALS AND METHODS

2.1 Study Design and Setting

Between January 2024 and December 2024, 6 private diabetes/internal medicine clinics located in Al-Mansour, Al-Karada, Al-Adhamiyya, Al-Zayouna, Al-A'amil, and Bab al-Muadham, representative districts of Baghdad, were included in this cross-sectional analytical study on diabetic patients with type 2 diabetes. These clinics had been selected based on their patient dump sizes, geographic locations, and physicians' willingness to participate. The private clinics providing care for patients diagnosed with DM constitute the bulk of DM care available to the people of Baghdad and serve patients from diverse socio-economic backgrounds and residential areas; therefore, they provide their patients with an accurate representation of the population from which they come.

2.2 Study Population

The selected study population consisted of adults with a known diagnosis of T2DM who attended the selected private clinics throughout the study period. A total population of 200 individuals was enrolled via purposive consecutive sampling. The inclusion criteria consisted of a confirmed diagnosis (to the criteria established by the American Diabetes Association for diagnosing T2DM; American Diabetes Association, 2024) [1], being 30 years old and older, having documented diabetes duration for at least 1 year, having an HbA1c measurement available from within the past 3 months of enrollment, and providing informed consent. Individuals were excluded if they: 1) were diagnosed with type 1 diabetes mellitus or other disease with recognized underlying causes; 2) had chronic kidney disease defined by eGFR < 60mL/min/1.73m²; 3) were currently taking medications that would significantly change serum magnesium levels such as proton pump inhibitors, loop diuretics, aminoglycosides, or calcineurin inhibitors; 4) had gastrointestinal malabsorption disorders or significant liver disease; 5) were pregnant or lactating; or 6) had an acute illness or infection at the time of enrollment.

2.3 Data Collection

Information from verified participants was collected using a standard questionnaire developed by the researchers, administered alongside interviews. The degree and types of sociodemographic information captured included age, gender, number of years of schooling completed, occupation, and municipality of residence. Clinical information that was recorded consists of: number of years diagnosed with diabetes, medications for Diabetes, hypertension, dyslipidemia, smoking status, and self-reported amount of physical activity engaged in. Body weight and body height were measured using calibrated equipment at the respective clinics; body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m²). Blood pressure was measured (using an appropriately sized, calibrated aneroid sphygmomanometer) from subjects while sitting after 5 minutes of rest; the mean of 2 readings was recorded.

2.4 Laboratory Measurements

Prior to the 8-hour blood draw, participants' fasting blood samples were collected via distal venipuncture and analysed using an automated instrument. The manufacturer's protocol was followed for sample processing using a previously developed method. Serum magnesium was measured using a Cobas c 311 automated biochemical analyzer from Roche Diagnostics in Mannheim, Germany using the xylidyl blue colorimetric assay method to quantitate serum magnesium from specimens processed within 2 hours of collection; the laboratory reference (normal) serum magnesium ranges from 0.75 to 0.95 mmol/L; hypomagnesemia is defined as having a serum magnesium concentration ≤0.75 mmol/L per internationally accepted criteria [13]. HbA1c measurement will be performed using high-performance liquid chromatography (HPLC) on Bio-Rad's D-10 Haemoglobin Testing System. The lab procedures utilised to perform HPLC on a Bio-Rad D-10 differ from those of NGSP/IFCC for establishing an HbA1c value. They will therefore be standardised using the NGSP/IFCC reference method. Classification of glycemic control into three clinically significant levels will be based on HbA1c: good control (HbA1c <7.0%), moderate control (HbA1c between 7.0 & 8.9%), and poor control (HbA1c ≥9.0%) per current ADA standards of medical care [1]. Fasting plasma glucose was analysed using the hexokinase enzymatic assay method. Other analytes, such as creatinine, total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol, will be quantified using standard assay methods with the same Cobas c 311 automated biochemical analyser. The eGFR calculated using the CKD-EPI equation will be used to determine whether a participant has significant renal impairment.

2.5 Ethical Considerations

All people who took part in this study signed a consent form to indicate that they agreed to be a part of it before they joined; all participants' identities were kept confidential via coded identifiers; all data collected from participants were kept in an electronic database that was password-protected, and only the research team members had access to the database. The study was conducted in accordance with the ethical standards outlined in the Declaration of Helsinki.

2.6 Statistical Analysis

Data entry and analysis were completed using the IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Mean ± standard deviation (SD) was used to express continuous variable data, while descriptive statistics (frequency and percentage) were used to express categorical variable data. The distribution of continuous variable data was assessed for normality with the Kolmogorov–Smirnov test. Continuous variable data were compared between two independent groups using independent samples t-tests and for three or more groups using analysis of variance (ANOVA) with a Tukey's honest significant difference (HSD) post hoc test. The strength of the linear relationship between the continuous serum magnesium variable and the continuous glycemic and biochemical variables was assessed with a Pearson's Product-Moment Correlation Coefficient (r). The logistic regression analysis was conducted to determine the independent predictors of hypomagnesemia, adjusting for covariates of age, sex, BMI, diabetes duration, glycemic control category, and antidiabetic treatment modality, and the results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). Statistical significance throughout the analyses was determined with a two-tailed p-value of ≤0.05.

3- RESULTS AND DISCUSSION

A total of 200 patients with type 2 diabetes mellitus (T2DM) were included in this study. The demographic information and clinical characteristics of the participants in this study are presented in Table 1. The average age of the participants was 52.4 ± 10.8 years; most were female (54.5%). The average duration of diabetes was 8.3 ± 5.6 years. Of the patients enrolled in the study, 62% had hypertension as the most common comorbidity. An overall mean serum magnesium level of 0.68 ± 0.12 mmol/L was observed, which is below the lower limit of the reference range (0.75 mmol/L), and 112 patients (56%) were hypomagnesemic. The average HbA1c level across all participants was 8.9 ± 1.6%, and 42% had poor glycemic control.

Table (1): Sociodemographic and Clinical Characteristics of the Study Participants (n = 200)

Variable	Value (Mean ± SD or n %)
Age (years)	52.4 ± 10.8
Female sex	109 (54.5%)
BMI (kg/m²)	29.7 ± 4.3
Diabetes duration (years)	8.3 ± 5.6
Hypertension	124 (62.0%)
Dyslipidemia	98 (49.0%)
Current smokers	44 (22.0%)
Oral antidiabetics only	112 (56.0%)
Insulin ± oral agents	88 (44.0%)
HbA1c (%)	8.9 ± 1.6
Fasting plasma glucose (mmol/L)	9.8 ± 2.9
Serum magnesium (mmol/L)	0.68 ± 0.12
Hypomagnesemia (<0.75 mmol/L)	112 (56.0%)

The serum magnesium concentrations for the three glycemic control categories are shown in Table 2. A statistically significant difference was present among the three glycemic control categories ($p < 0.001$, one-way ANOVA with Tukey's HSD). The group with the lowest mean serum magnesium concentration was the poor glycemic control group (0.59 ± 0.08 mmol/L). The group with the next highest mean serum magnesium concentration was the moderate glycemic control group (0.71 ± 0.09 mmol/L). The good glycemic control group had the highest mean serum magnesium concentration (0.81 ± 0.07 mmol/L). There was a progressive increase in the prevalence of hypomagnesemia from 11.8% (good glycemic control category) to 83.3% (poor glycemic control category). This difference was also statistically significant.

Table (2): Serum Magnesium Levels by Glycemic Control Category

Glycemic Control Category	n (%)	Serum Mg (mmol/L) Mean ± SD	Hypomagnesemia n (%)	p-value
Good control (HbA1c <7.0%)	34 (17.0%)	0.81 ± 0.07	4 (11.8%)	<0.001*
Moderate control (HbA1c 7.0–8.9%)	82 (41.0%)	0.71 ± 0.09	56 (68.3%)	
Poor control (HbA1c ≥9.0%)	84 (42.0%)	0.59 ± 0.08	70 (83.3%)	

*** One-way ANOVA with Tukey HSD post-hoc test; all pairwise differences were statistically significant ($p < 0.05$)**

There was a statistically significant negative correlation between serum magnesium levels and HbA1c ($r = -0.52$, $p<0.001$); fasting plasma glucose ($r = -0.44$, $p<0.001$); diabetes duration ($r = -0.31$, $p<0.001$); and BMI ($r = -0.19$, $p=0.007$). In each of these cases, lower serum magnesium levels were associated with higher glycemic indices, longer diabetes duration, and greater adiposity. Age did not have a statistically significant correlation with serum magnesium levels ($r = -0.081$, $p = 0.263$).

Table (3): Pearson Correlation Between Serum Magnesium and Clinical/Biochemical Parameters

Parameter	Pearson r	p-value
HbA1c (%)	−0.52	<0.001
Fasting plasma glucose (mmol/L)	−0.44	<0.001
Diabetes duration (years)	−0.31	<0.001
BMI (kg/m²)	−0.19	0.007
Age (years)	−0.08	0.263

The serum magnesium level data presented in Table 4 indicate a statistically significant difference in mean magnesium levels between those with the longest duration of diabetes (>10 years) and those with a shorter time since diabetes diagnosis (<5 years) ($p = 0.001$). The mean serum magnesium concentration for the 10 years + group was 0.58 mmol/L (± 0.09); the mean concentration for <5 years' duration of diabetes was 0.77 mmol/L (± 0.10). The patients with >10 years of diabetes showed the greatest prevalence of hypomagnesemia (72.4%) compared with the less than 5-year category (38.7%).

Table (4): Serum Magnesium Levels by Duration of Diabetes

Diabetes Duration	n (%)	Serum Mg (mmol/L) Mean ± SD	Hypomagnesemia n (%)	p-value
<5 years	62 (31.0%)	0.77 ± 0.10	24 (38.7%)	<0.001*
5–10 years	80 (40.0%)	0.67 ± 0.11	46 (57.5%)	
>10 years	58 (29.0%)	0.58 ± 0.09	42 (72.4%)	

*** One-way ANOVA, $p < 0.001$; all pairwise comparisons significant by Tukey HSD**

According to the binary logistic regression analysis carried out in this study, we found that poor glycemic control (HbA1c ≥9.0%; OR=3.84, 95% CI: 1.96–7.52; p <0.001), length of time diagnosed with diabetes greater than 10 years (OR=2.93, 95% CI: 1.41–6.09; p=0.004), and use of insulin (OR=2.17, 95% CI: 1.08–4.37; p=0.029) were all found to be statistically significant independent predictors for hypomagnesemia after adjusting for all covariates. In contrast, BMI ≥30 kg/m2, male gender, and hypertension did not reach statistical significance (p > 0.05).

Table (5): Binary Logistic Regression Analysis for Independent Predictors of Hypomagnesemia

Predictor Variable	OR	95% CI	p-value
Poor glycemic control (HbA1c ≥9.0%)	3.84	1.96 – 7.52	<0.001
Diabetes duration >10 years	2.93	1.41 – 6.09	0.004
Insulin use	2.17	1.08 – 4.37	0.029
BMI ≥30 kg/m²	1.58	0.84 – 2.97	0.152
Male sex	1.23	0.65 – 2.33	0.521
Hypertension	1.41	0.72 – 2.75	0.314

OR: Odds Ratio; CI: Confidence Interval. Reference categories: glycemic control HbA1c <9.0%; diabetes duration <10 years; antidiabetic treatment: oral agents only.

The current study was a cross-sectional analysis of serum magnesium concentrations and their association with glycemic control (HbA1c) among T2DM patients treated at private health care clinics in Baghdad City. Three important findings were reported: (1) Hypomagnesemia was found to be highly prevalent (56%) among the enrolled sample; (2) Serum magnesium levels were negatively and significantly correlated with both HbA1c and fasting plasma glucose level; and (3) Independent predictors of magnesium deficiency, following multivariate adjustment, included poor glycemic control, longer duration of diabetes, and insulin use. This study provides valuable epidemiological data to support the growing international literature on the metabolic consequences of magnesium status in T2DM. The prevalence of hypomagnesemia (56%) in the present study is considerably higher than reported in other Western and East Asian countries, where hypomagnesemia among T2DM patients is generally in the range of 25-39% [14]. Osmotic diuresis results in poor reabsorption of magnesium from the renal tubular system, due to the increased volume of urine that needs to be reabsorbed, leading to excessive urination, which ultimately results in the loss of magnesium. In particular, this can occur when patients with diabetes have chronically elevated blood glucose levels, which reduce the expression of TRPM6 channels in the renal tubular system, thereby significantly impeding magnesium transport in the renal tubules [8].

When examining the correlation between serum magnesium and HbA1c levels ($r = -0.52$, $p < 0.001$), the findings of this study are consistent with several recent studies that have reported an inverse relationship between these two markers. The most important mechanistic explanation for the link between magnesium levels, insulin receptor function, and HbA1c levels is that magnesium is an obligatory cofactor in activating the insulin receptor through its tyrosine kinase activity [7]. If a person is magnesium-deficient, the phosphorylation of the insulin receptor beta subunit by insulin will not occur, resulting in decreased activation of insulin receptor substrate-1 (IRS-1) and reduced stimulation of the phosphoinositide-3-kinase (PI3K/Akt) signalling pathway. This signalling pathway is required for GLUT-4 translocation to the plasma membranes of skeletal muscle and adipose tissue for the uptake of glucose from the blood [15]. The present study demonstrated significantly lower serum magnesium concentrations (0.59 ± 0.08 vs 0.81 ± 0.07 mmol/L; $p < 0.001$) in individuals with poor glycemic control (HbA1c ≥9%) compared with those with good glycemic control. Thus, this finding illustrates and supports the bidirectional aspect of the magnesium and hyperglycemia relationship as proposed by Gommers *et al*. (2016) [8]. In this hypothesis, chronic hyperglycemia causes renal magnesium wasting due to osmotic diuresis and TRPM6 downregulation; this mechanism of hypomagnesemia contributes to impaired insulin receptor signalling and diminished insulin secretion, leading to poor glycemic control, which further compounds and perpetuates urinary magnesium loss through a positive feedback cycle [16].

The decrease in serum magnesium concentration is clinically significant and consistent with previous pathophysiological explanations, given the sustained reduction in serum magnesium over the course of diabetes, as demonstrated in this analysis (Table 4). The lowest serum magnesium concentration (0.58 ± 0.09 mmol/L) and the

highest prevalence of hypomagnesemia (72.4%) were observed in individuals with diabetes for more than 10 years; the aforementioned factors of hyperglycemia-induced renal magnesium wasting, autonomic neuropathy, and subclinical renal impairment associated with more advanced disease are likely contributors to this phenomenon [17]. Our logistic regression analysis indicates that diabetes duration is an independent predictor of hypomagnesemia (OR = 2.93, 95% CI [1.41,6.09], p=.004), indicating the importance of routinely monitoring serum magnesium levels in long-standing type 2 diabetes mellitus (T2DM) patients due to their increased risk of clinically significant depletion due to long duration of T2DM.

Insulin-treated patients in our study were typically more severely diseased, had longer duration of diabetes, and had lower baseline glycemic control than those patients without insulin treatment, with each of these variablesThe independent effect of insulin therapy on magnesium levels in our multivariate model (adjusting for HbA1c level and diabetes duration) indicates that factors other than glycemic dysregulation (e.g., alterations in renal tubular magnesium handling due to insulin resistance) may also act independently to cause magnesium depletion. There was a strong relationship between insulin resistance and levels of hypomagnesemia, which illustrates the synergistic impact of the two metabolic variables [18].

Although we did not measure dietary magnesium intake in the present study, the role of dietary magnesium in causing magnesium depletion in Baghdad warrants consideration. Multiple large prospective cohort studies have found that higher dietary magnesium intake is associated with a lower risk of T2DM and better markers of glycemic dysregulation. In addition to the glycemic impact of magnesium supplementation. There was evidence that low levels of magnesium in diet were positively associated with the indeces of systemic inflammation measured by C-reactive protein (CRP), interleukin-6 (IL-6) and markers of endolothelial dysfunction in a large population of multiple ethnic tribes in the USA [19]; this is important because the increased level of systemic inflammation is the basis for poor glycemic control in patients suffering from type 2 diabetes mellitus (T2DM). Since progressive insulin resistance and beta-cell failure are both mainly caused by chronic low-grade inflammation, it follows that the pro-inflammatory effects of low levels of magnesium, as evidenced by Barbagallo and Dominguez (2015), will add to the metabolic deterioration of patients suffering from T2DM with low levels of magnesium, beyond the reduction in insulin receptor function [7].

The current study has many strengths, including multiple sites in geographically diverse areas of Baghdad; utilisation of standardised, validated, and used measures for both serum magnesium content and HbA1c levels; and the application of multivariate logistic regression to identify distinct predictors while controlling for all potential confounding factors. There are, however, some weaknesses in this study that should be noted. The cross-sectional design does not allow for establishing causal relationships or determining the sequence of magnesium and glucose metabolism over time. Magnesium intake as measured by diet was not profiled in this study; therefore, we are not able to differentiate between patients who were magnesium-deficient due to nutritional versus renal depletion mechanisms. This study included participants only from private clinics in the city of Baghdad; therefore, many T2DM patients treated in public and primary care facilities may not have been included. Additionally, total serum magnesium was used to evaluate magnesium status rather than ionised magnesium; however, it has been shown that total serum magnesium may not accurately reflect intracellular magnesium stores in patients with hypoalbuminemia and/or acid–base disturbances [20]. Finally, there may have been bias from purposive sampling. More studies in Iraq, using prospective designs and the collection of new data, including ionised magnesium, 24-hour urinary excretion of magnesium, a dietary assessment of magnesium and inflammatory biomarkers, will provide a better understanding of the factors that will determine magnesium deficiency and how it may affect the health of T2DM patients in Baghdad and all of Iraq.

4- CONCLUSION

The present study demonstrates that hypomagnesemia is highly prevalent among T2DM patients attending private clinics in Baghdad city, affecting more than half of the study cohort. Serum magnesium levels showed a significant, clinically meaningful inverse association with HbA1c and fasting plasma glucose, corroborating the established bidirectional pathophysiological relationship between magnesium deficiency and glycemic dysregulation. Poor glycemic control, longer diabetes duration, and insulin use were identified as statistically significant independent predictors of hypomagnesemia after multivariate adjustment. These findings collectively highlight the importance of integrating routine serum magnesium measurement into the biochemical monitoring protocols for T2DM in Iraqi clinical practice. Nutritional counselling promoting the consumption of magnesium-rich foods including whole grains, legumes, nuts, seeds, and dark green leafy vegetables should be incorporated into

diabetes management. Consideration should be given to therapeutic magnesium supplementation in T2DM patients with documented hypomagnesemia, particularly those with persistently poor glycemic control. Prospective randomised controlled trials evaluating the impact of magnesium supplementation on long-term glycemic outcomes, complication risk, and quality of life in Iraqi patients with T2DM are warranted to establish a comprehensive therapeutic evidence base.

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