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The Effect of Serum Zinc and Magnesium on Glycemic Control in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background: Magnesium (Mg) and zinc (Zn) are essential elements in glucose metabolism, insulin action, and antioxidant defense. Their deficiency or imbalance is frequently observed in type 2 diabetes mellitus (T2DM), making their measurement clinically relevant for understanding metabolic disturbances in this disease.

Aim of study: To evaluate serum levels of magnesium and zinc in patients with type 2 diabetes mellitus in Babylon city, Iraq and to examine their association with glycemic control, thereby clarifying their potential role in metabolic regulation and diabetes management.

Subjects and Method: This case control study was carried out in Babylon City between February and October 2025. A total of 200 type 2 diabetic patients (100 with adequate glycemic control and 100 patients with poor glycemic control), as well as 60 healthy subjects as control group were enrolled. Both patients and controls were recruited from a private clinical laboratory in Babylon City, namely Al-Zahrawi Medical Laboratory

Result: The HbA1c is inversely correlated with each of serum magnesium ($\rho = -0.276$, $p < 0.001$) and serum zinc ($\rho = -0.182$, $p = 0.003$), and the lower levels of these micronutrients were associated with poorer glycemic control. A weak significant positive correlation was found between serum magnesium and serum zinc ($\rho = 0.123$, $p = 0.048$).

conclusion: Serum magnesium and zinc levels are significantly reduced in individuals with type 2 diabetes mellitus, especially in those patients with poorly controlled glycemia, and that levels of both are inversely proportional to glycated haemoglobins level.

Keywords: Type 2 Diabetes Mellitus, Glycemic Control, Glycated hemoglobin, Serum Zinc serum Magnesium

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INTRODUCTION:

Diabetes mellitus (DM) is a chronic, metabolic disease characterized by elevated levels of blood glucose, which leads over time to serious damage to the heart, blood vessels, eyes, kidneys and nerves. Hence, it represents a group of metabolic disorders that characterized by persistent hyperglycemia due to the body's inability to produce enough insulin or inefficient insulin usage by the target cells or both as well as there is impairment in the metabolism of proteins and lipids (1,2). Different factors contribute to etiopathogenesis of DM which include a complex interaction between genetic, environmental and lifestyle factors (3). In the present era, DM represents a significant health problem worldwide due to its continuous rapid increasing incidence and prevalence and its complications. Type 2 DM (T2DM), is the commonest type and more than 90% of DM cases are of type 2 (1), the development of T2DM is mainly due to deficiency in the secreted insulin and non-response of the target tissues to insulin(4)(1). Despite T2DM can affect adults at any age, it is more frequently diagnosed among individuals older than 45 years, nonetheless, it still reported among children, adolescents and young adults due to increasing prevalence of obesity, sedentary lifestyle, and consumption of unhealthy diet (5). Several risk factors are contribute to development of T2DM and much risk factors are modifiable and can be prevented such as overweight/ obesity, smoking, low-fiber diet, some medications and other factors(6), however, the etiology of T2DM is multifactorial (7). Complications of DM in general can be categorized as acute; such as hypoglycemia as a major complication of hypoglycemic treatment. Hyperosmolar hyperglycemic state which may be attributed to infections, certain drugs, or cardiovascular insults. The chronic complications of diabetes include neuropathy, nephropathy, retinopathy, as well as, macroangiopathy, cardiovascular disease, stroke, and peripheral artery disease PAD. Diabetic foot syndrome has been defined as the presence of foot ulcer associated with neuropathy (8 – 14). The diagnosis of DM is based on the criteria of diagnosis of the American Diabetes Association (15), or criteria of the World Health Organization (16). Magnesium and zinc have been postulated to have a role in different metabolic disorders, hypomagnesemia has been documented to have an impact on insulin resistance and DM, therefore, maintaining an optimal levels of magnesium is crucial in DM patients due to its role in glucose and insulin

metabolism (17,18). Zinc is an essential trace mineral has shown to play a significant role in DM (19) due to its crucial antioxidant action (20), however, the relationship between the impaired regulation of trace elements and pathogenesis of DM is complex and highlighting the need for good monitoring to maintain sufficient micronutrients nutritional balance to get better glycemic control (21). Therefore, the present study aimed to evaluate serum levels of magnesium and zinc in patients with type 2 diabetes mellitus in Babylon city, Iraq and to examine their association with glycemic control, thereby clarifying their potential role in metabolic regulation and diabetes management.

PATIENTS AND METHODS:

This case control study was carried out in Babylon City between February and October 2025. A total of 260 participants were enrolled, comprising 60 healthy controls (hemoglobin A1c < 5.7%), 100 type two diabetic patients with adequate glycemic control (hemoglobin A1c < 7%), and 100 type two diabetic patients with poor glycemic control (hemoglobin A1c > 7%). Both patients and controls were recruited from a private clinical laboratory in Babylon City, namely Al-Zahrawi Medical Laboratory.

Ethical considerations

Official approval granted from university of Baghdad/collage of medicine/ department of clinical biochemistry. Official approval granted from the Iraqi Board for Medical Specializations. Verbal consents from all volunteers were gained prior to their engagement in the study.

Patients and controls' groups:

This study included 200 fasting patients (both male and female) with previously established diagnoses of type 2 diabetes mellitus (T2DM), aged between 30 and 70 years, the diagnosis of T2DM was determined according to the American Diabetes Association (ADA) criteria applicable at the time of diagnosis and confirmed by a consultant physician. The control group comprised 60 fasting, apparently healthy individuals who were clinically and biochemically free of diabetes mellitus, had no acute or chronic illnesses, and were not receiving long-term therapeutic treatment, This group was age-and BMI matched with the patient cohort. The patients and control subjects were selected based on the following exclusion criteria.

Exclusion criteria

Participant was excluded if he/or she had one or more of the following criteria

1. Taking magnesium or zinc supplements
2. Pregnant or breastfeeding woman.
3. Diabetic patients on insulin therapy.
4. Current smokers, or ex-smokers within the past 12 months and those with alcohol consumption.
5. Presence of acute infection.
6. History of renal or liver disease, hypertension, cardiac disease, endocrine disorders, Gastrointestinal disorders (Inflammatory bowel disease, irritable bowel syndrome, intestinal or biliary fistulas, acute/chronic diarrhea), hemolytic anemia or hemoglobinopathies.
7. Got a blood transfusion within the past 3 weeks.
8. Patients currently using proton pump inhibitors, loop or thiazide diuretics, Cetuximab, cisplatin, Certain antimicrobials (Like: rapamycin, aminoglycosides, pentamidine, foscarnet, amphotericin B), Laxatives (abused)

Data collection:

It was performed through a detailed medical history taking and a thorough clinical examination. Anthropometric measurements; weight, height and body mass index (BMI) calculation, which then categorized according to the WHO criteria as underweight (BMI<18.5 kg/m^2), normal, overweight and obese (22).

Collection of blood samples and testing

A sample of venous samples and testing:blood was collected from each participant under aseptic conditions and sent to the laboratory for testing using the standard protocols of tests applied at our setting.

Measurement of serum Magnesium was made with the biochemistry analyzer CS-T180 Holographic concave flat field grating with rear spectrophotometry and 340 nm to 800 nm and the Biomaghreb magnesium Colorimetric Method kit principle for measuring magnesium level was followed and the reference value for Serum, Plasma is 1.6 - 2.5 mg/dI.

Measurment of serum zinc was by CS-T180 The Automatic biochemistry analyzerCS-T180 used to measure zinc by the SPECTRUM zinc (Colorimetric Test with 5-Bromo-PAPS) and the measurement protocol and procedure were applied according to the standard protocol and the Kit instructions

RESULTS:

The median age was 50.0 years among the 200 diabetic cases' and it was 47.0 years among the 60 controls. For cases, 87 were male (43.5%) and 113 were female (56.5%), whereas controls consisted of 28 (46.7%) males and 32 (53.3%) females. The median weight was 82.0 kg in cases and 83.0 kg in controls. The median height was 164.0 cm in cases and 165.0 cm in controls. The median BMI was 30.47 kg/m² in cases against 29.39 kg/m² in controls, (**Table 1**). The median HbA1c value among the 100 cases with good glycemic control was 6.40% whereas it was 8.95% among the 100 cases with poor glycemic control. Among the 60 controls, the median HbA1c was 5.30%, (**Table 2**). Serum magnesium and zinc levels were compared between diabetic patients with good and poor glycemic control using the Mann–Whitney U test. Patients with poor glycemic control demonstrated lower serum magnesium levels compared with those with good glycemic control (median 1.80 mg/dL [IQR 1.60–2.00] vs 1.90 mg/dL [IQR 1.70–2.20], P. value = 0.023). This difference remained statistically significant after Bonferroni adjustment (adjusted P. value = 0.046). Similarly, serum zinc levels were significantly lower in the poor glycemic control group (median 95.5 µg/dL [IQR 82–113.25]) compared to those with good glycemic control (median 107 µg/dL [IQR 95–119]) (P. value = 0.022), and this association also remained significant after Bonferroni correction (adjusted P. value = 0.044), (**Table 2**).

Spearman's correlation analysis was performed to evaluate the relationships between glycated hemoglobin (HbA1c), serum magnesium, and serum zinc levels among diabetic cases. HbA1c showed a significant negative correlation with serum magnesium (Spearman's rank correlation coefficient (ρ) = −0.174, P. value = 0.014) and serum zinc (ρ = −0.241, P. value < 0.001), indicating that higher HbA1c levels were associated with lower concentrations of these trace elements. In contrast, serum magnesium and serum zinc demonstrated a weak positive

correlation (ρ = 0.120); however, this relationship did not reach the statistical significance (P. value = 0.091). (**Table 3**).

In the combined cohort of cases and controls, Spearman's correlation analysis showed a statistically significant inverse correlation between glycated hemoglobin (HbA1c) and serum magnesium (ρ = −0.276, p < 0.001), as well as between HbA1c and serum zinc (ρ = −0.182, P. value = 0.003). A weak but statistically significant positive correlation was also observed between serum magnesium and serum zinc (ρ = 0.123, P. value = 0.048), (**Table 4**).

In the poor glycemic control subgroup combined with the control group, Spearman correlation analysis demonstrated a statistically significant inverse correlation between glycated hemoglobin (HbA1c) and serum magnesium (ρ = −0.368, p < 0.001), as well as between HbA1c and serum zinc (ρ = −0.259, p < 0.001). A statistically significant positive correlation was also observed between serum magnesium and serum zinc (ρ = 0.295, P. value < 0.001), (**Table 5**).

In the good glycemic control subgroup combined with the control group, Spearman correlation analysis showed a weak but statistically significant inverse correlation between glycated hemoglobin (HbA1c) and serum magnesium (ρ = −0.176, P. value = 0.026). No statistically significant correlations were observed between HbA1c and serum zinc (ρ = −0.038, P. value = 0.636) or between serum magnesium and serum zinc (ρ = −0.040, P. value = 0.618), (**Table 6**).

Table 1. Descriptive statistics of demographic characteristics and anthropometric measurements among the studied groups.

Variable		Cases (N = 200)	Controls (N = 60)
Age (year), median (IQR)		50.0 (44–56)	47.0 (40–55)
Sex, n (%)	Male	87 (43.5%)	28 (46.7%)
	Female	113 (56.5%)	32 (53.3%)
Weight (kg), median (IQR)		82.0 (73–93)	83.0 (72–90)
Height (cm), median (IQR)		164.0 (160–170)	165.0 (160–175)
BMI (kg/m²), median (IQR)		30.47 (26.8–34.2)	29.39 (25.9–33.1)

Table 2. Comparison of Glycated Hemoglobin (HbA1c), Serum Magnesium and Zinc levels across the studied groups

Variable	Cases		Controls (N = 60)	P. value
	Good Control (N = 100)	Poor Control (N = 100)		
HbA1c (%)	6.40 (5.9–6.7)	8.95 (8.1–10.2)	5.30 (4.9–5.6)	<0.001
Serum Magnesium (mg/dL)	1.90 (1.70–2.20)	1.80 (1.60–2.00)	NA	0.023
Serum Zinc (µg/dL)	107 (95–119)	95.5 (82–113.25)	NA	0.022

All values presented as median and inter-quartile range (IQR), NA: not performed for controls

Table 3. Results of bivariate Spearman's correlation analysis for glycated hemoglobin, serum magnesium, and serum zinc in diabetic cases.

Parameter	*Statistics*	Glycated Hb	S. Magnesium
S. Magnesium	ρ	−0.174	-
	P. value	0.014	-
S. Zinc	ρ	−0.241	0.120
	P. value	<0.001	0.091

ρ: Spearman's rank correlation coefficient

Table 4. Results of bivariate Spearman's correlation analysis for glycated hemoglobin, serum magnesium, and serum zinc in all cases and controls

Parameter	*Statistics*	Glycated Hb	S. Magnesium
S. Magnesium	ρ	−0.276	-
	P. value	<0.001	-
S. Zinc	ρ	−0.182	0.123
	P. value	0.003	0.048

ρ: Spearman's rank correlation coefficient

Table 5. Results of bivariate Spearman's correlation analysis for glycated hemoglobin, serum magnesium, and serum zinc in cases with poor glycemic control and controls' group

Parameter	*Statistics*	Glycated Hb	S. Magnesium
S. Magnesium	ρ	−0.368	-
	P. value	<0.001	-
S. Zinc	ρ	−0.259	0.295
	P. value	<0.001	<0.001

ρ: Spearman's rank correlation coefficient

Table 6. Results of bivariate Spearman's correlation analysis for glycated hemoglobin, serum magnesium, and serum zinc in cases with good glycemic control and controls' group

Parameter	*Statistics*	Glycated Hb	S. Magnesium
S. Magnesium	ρ	−0.176	-
	P. value	0.026	-
S. Zinc	ρ	−0.038	−0.040
	P. value	0.636	0.

ρ: Spearman's rank correlation coefficient

DISCUSSION:

The results show that cases with poor glycemic control (median HbA1c 8.95%) had higher HbA1c than cases with good control (6.40%) and controls (5.30%), with similar demographic and anthropometric profiles between groups (median age, sex distribution, BMI). This pattern is consistent with the established role of HbA1c as a marker of glycemic control and risk for diabetes complications, as emphasized by the American Diabetes Association, which recommends routine HbA1c monitoring to guide therapy and risk assessment in diabetes management. (23) Comparing these findings to another study, Tarekegn et al. (2025) reported that 73.5% of Ethiopian type 2 diabetes patients had poor glycemic control, with obesity (BMI ≥30 kg/m²), overweight, high cholesterol, and longer diabetes duration as significant risk factors for poor control.(24) In both studies, BMI values for cases and controls were in the overweight/obese range (around 30 kg/m²), but BMI did not differ substantially between groups, suggesting that while obesity is a risk factor for diabetes and poor control, it may not distinguish between well- and poorly controlled patients in clinic-based samples.

Moreover, Lin (2024) discovered that BMI has a positive correlation with HbA1c in people without diabetes but that the strength of this association is reduced when people have diabetes. This is consistent with the data, whereby although the mean value of BMI is similar across each group, values of HbA1c are significantly different.(24,25)

The results show that serum magnesium and zinc levels are lower in diabetic patients, especially those with poor glycemic control, compared to controls. Specifically, median serum magnesium was 1.90 mg/dL in cases with good control, 1.80 mg/dL in poor control, and 2.00

mg/dL in controls. Median serum zinc was 105.0 µg/dL in good control, 95.5 µg/dL in poor control, and 107.0 µg/dL in controls .

Lower levels of serum magnesium have been reported to be associated with poor glycemic control and an increased incidence of diabetic complications. Multivariate analysis has disclosed an inverse relationship between serum levels of magnesium and HbA1c, the former being more common in poorly controlled diabetes and being associated with elevated HbA1c and fasting glucose concentrations.(26) Lower levels of plasma zinc have also been associated with poor glycemic control. Lower plasma concentrations of zinc and elevated urinary zinc excretion have been found to be associated with poor metabolic control as indicated by HbA1c. The mechanism relates to the increased renal excretion of these trace elements due to hyperglycemia.(23) For the purpose of comparison, the study by Samadi et al. (2020). revealed a higher HbA1c level in patients with lower plasma zinc, as well as a negative correlation between plasma zinc levels and glycemic levels in patients with T2DM.(27) Ozcaliskan et al. detected a higher HbA1c level in patients with hypomagnesemia, as well as lower glycemic control, in patients with T2DM.(28) This study supports the result as lower levels of magnesium and zinc contribute to poor glycemic control in patients.Routine monitoring and correcting deficiencies of magnesium and zinc may prove advantageous in maximizing glucose control in T2DM(26–28).

The mean serum magnesium and zinc values are lower in patients with poor glycemic control compared to those with good glycemic control in diabetes. Mann-Whitney U test results consistently show statistically significant differences in both magnesium and zinc levels between these groups, with lower levels in poorly controlled patients ($p < 0.05$).(26,27,29) Spearman correlation analysis demonstrates a significant inverse relationship between serum magnesium and HbA1c ($r \approx -0.32$ to -0.65, $p < 0.01$) and between serum zinc and HbA1c ($r \approx -0.32$ to -0.68, $p < 0.01$), indicating that as HbA1c increases, both magnesium and zinc decrease.(26,27)

These results are in accordance to previous studies. Regarding magnesium, hypomagnesemia is known to have an association with poor glycemic control and in turn an elevated level of HbA1c, as stated in the studies of Ozcaliskan et al. and Joy et al. showing mean values of HbA1c to have significant highs in hypomagnesemia.(26,28) Regarding zinc, the studies of Samadi et

al. (2020). and Viktorínová et al. conclude the association between reduced plasma levels of zinc and in turn an elevated level of HbA1c, and the association is significant.(27,29)

In conclusion, low mean serum levels of magnesium and zinc correlate well with poor glycemic control found in diabetes These observations are well-substantiated by the medical literature, underscoring the importance of monitoring and addressing trace element deficiencies in the management of type 2 diabetes. while saeed et al. have reported no significant association between serum magnesium and glycemic control, (30)in contrast to our findings This discrepancy may reflect differences in dietary intake, genetic background, or study design.

The correlation analysis in the combined cohort demonstrates that HbA1c is inversely correlated with both serum magnesium ($\rho = -0.276$, $p < 0.001$) and serum zinc ($\rho = -0.182$, $p = 0.003$), indicating that lower levels of these micronutrients are associated with poorer glycemic control. Additionally, there is a weak but statistically significant positive correlation between serum magnesium and serum zinc ($\rho = 0.123$, $p = 0.048$), suggesting some degree of shared metabolic or renal handling, but not a strong direct relationship.

These results are in accordance with the results described in the medical literature. Al-Daghri et al. (2025) have found an inverse correlation between HbA1c and the level of magnesium and zinc in the serum of diabetic patients, confirming the inverse relationship between poor glycemic control and magnesium and zinc status(29) Santos RK et al. have confirmed the same correlations in the case of type 2 diabetes and HbA1c, as well (31) The positive correlation between magnesium and zinc is weaker but confirmed in other studies, due to the different regulatory pathways of these trace elements.

In summing up, the findings confirm that low serum magnesium and zinc levels correlate with high HbA1c, and that these two nutrients could be involved in regulating blood sugar. The negative correlation between magnesium and zinc implies that they could be co-regulated, but both independently regulate blood sugar. (29,31)

In the poor glycemic control group combined with the control group, the correlation analysis yielded a statistically significant inverse correlation between HbA1c and serum magnesium levels ($\rho = -0.368$, $p < 0.001$), and HbA1c and serum zinc levels ($\rho = -0.259$, $p < 0.001$). There was a statistically significant positive association between serum levels of zinc and magnesium ($\rho = 0.295$, $p < 0.001$). The above results indicate a negative association between poor glycemic

control and the levels of either mineral, and a certain association between the levels of the two minerals in the population under study.

This finding is in line with recent research. Magnesium deficiency has a moderate relationship with elevated HbA1c and insulin resistance in type 2 diabetic patients, as indicated by a meta-analysis: the correlation coefficient for magnesium and HbA1c was -0.35, and it was statistically significant ($p < 0.01$).(32) On the other hand, lower zinc levels are related to an inability to control blood sugar. The plasma concentration of zinc has opposite relationship with HbA1c; $r = -0.325$, and it was statistically significant ($p = 0.003$). The positive relationship of magnesium and zinc was also validated.(29,33) In the good glycemic control subgroup combined with controls, there was a weak but significant inverse correlation between HbA1c and serum magnesium ($\rho = -0.176$, $p = 0.026$), but no significant correlations between HbA1c and serum zinc or between serum magnesium and serum zinc. This suggests that in individuals with better glycemic control, magnesium remains weakly associated with HbA1c, but zinc does not, and the interrelationship between magnesium and zinc is less pronounced.

Recent studies support these findings: the strength of the association between micronutrient deficiencies and glycemic control is greater in poorly controlled diabetes, and only a small percentage of HbA1c variation is explained by micronutrient status in well-controlled patients.(27,33) Thus, the inverse correlations are stronger in poor control, and weaker or absent in good control, reflecting the greater impact of micronutrient depletion in advanced dysglycemia.

In summary The data suggest that both serum magnesium and zinc concentrations are decreased, especially in those patients with poorly controlled glycemia, and that levels of both are inversely proportional to glycemia, which is again stronger for poorly controlled glycemia, and weak or no associations for those under better glycemic control. Taken together, all findings support existing evidence for alterations of magnesium and zinc homeostasis that accompany poorly controlled glycemia and suggest alterations of the metabolic/renal state associated with poorly controlled DM, strengthening once again the role of both micronutrients in characterizing patients' general glycemia statuses of those afflicted with 2 DM.

Limitation

This study is not free of some limitiations; Its case-control design and limited by its case–control design and single-site recruitment from a private laboratory in Babylon, restrict both causal inference and generalizability of findings. Serum magnesium and zinc levels were assessed using single fasting measurements that may not reflect total body stores and are subject to biological and pre-analytical variability .Moreover, HbA1c level may be influenced by unmeasured hematologic conditions affecting red-cell turnover. Finally, the sample size may limit robust subgroup analyses.

CONCLUSION:

The current study showed that glycated hemoglobin is the key discriminant for good and poor controlled glucose levels in patients with type 2 diabetes mellitus, whereas demographic and anthropometric parameters have limited discriminant ability. Serum levels of magnesium and zinc were found to be lower in diabetic patients, more so in patients with poorer control, and both micronutrients demonstrated inverse associations with HbA1c, with stronger associations found in patients with poorer control. The results also indicate associations that are indicative of magnesium and zinc being partly co-regulated, with each being independently associated with glucose control. Taken together, these data confirm that abnormalities in magnesium and zinc metabolism relate to worsening glucose control, implicating their importance as factors for the assessment of patients with type 2 diabetes mellitus. Hence we recommend encouraging consumption of magnesium and zinc-rich, and consider magnesium and zinc supplementation in deficient patients, under medical supervision, to support glycemic control and reduce oxidative stress. Additionally, Incorporate micronutrient evaluation into routine diabetes management protocols, alongside glucose and lipid monitoring. However, further studies are still needed to clarify the therapeutic role of magnesium and zinc supplementation in improving outcomes in type 2 diabetic patients.

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