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Research Article

The impact of magnesium on glycemic regulation

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Abstract

Objectives: Magnesium is a cofactor for the phosphorylation of glucose and many other enzymatic reactions, and a deficiency can cause insulin resistance, carbohydrate intolerance, and dyslipidemia. A low serum magnesium level can lead to the development of diabetic complications, such as retinopathy, abnormal platelet function, cardiovascular disease, or hypertension. The aim of this study was to investigate the relationship between serum magnesium level and glucose regulation.

Methods: The results of serum level tests of glucose, magnesium, glycated hemoglobin (HbA1c), and insulin values, as well as a lipid panel, performed for 256 patients between January 2016 and December 2017 were retrospectively evaluated. The patients were divided into 3 groups: nondiabetic, prediabetic, and diabetic, according to the glucose and HbA1c levels specified in the 2017 American Diabetes Association criteria. Kruskal-Wallis H and Mann-Whitney U tests were used for intergroup comparisons.

Results: The patients who were nondiabetic (n=137), prediabetic (n=85), and diabetic (n=34) were aged (median [range]) 40 years (24-55), 45 years (35-58), and 48 years (30-57), respectively. There were significant differences between the nondiabetic and diabetic groups, as well as the prediabetic and diabetic groups, with respect to magnesium level ($p<0.001$ and $p<0.001$, respectively). No statistically significant difference was detected between the magnesium level of the nondiabetic and prediabetic groups. The magnesium level was significantly lower in the insulin resistant group (Homeostatic Model Assessment of Insulin Resistance [HOMA-IR] ≥2.5) ($p=0.020$). There was a significant negative correlation between magnesium level and glucose, HbA1c, and HOMA-IR values ($r=-0.244$, $p<0.001$; $r=-0.332$, $p<0.001$; and $r=-0.162$, $p=0.010$, respectively).

Conclusion: Magnesium is a cofactor of various enzymes involved in carbohydrate oxidation and plays an important role in the transport of glucose to the cell membrane. In this study, it was observed that a low level of magnesium negatively affected glucose regulation. Magnesium-rich foods and/or oral magnesium supplementation may be beneficial in diabetic patients with reduced serum magnesium by increasing insulin sensitivity.

Keywords: Diabetes mellitus, insulin, magnesium, prediabetes

Magnesium is an integral part of the cellular membrane structure, and aids in stabilization of the membrane [1, 2]. As a cofactor of many enzymes involved in energy metabolism, magnesium has a role in carbohydrate, lipid, and protein metabolism. It also plays a part in the activity of various enzymes related to glucose oxidation and insulin release [3, 4].

Studies have shown that serum magnesium levels are lower in diabetic patients than in nondiabetic patients, and magnesium deficiency has been suggestively associated with hyperglycemia, hyperinsulinemia, and hence, insulin resistance [5-8]. The incidence of magnesium deficiency in diabetic patients has been reported as 11% to 47.7% [9-13]. This wide range of incidence is probably due to differences in the

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techniques of magnesium measurement, the heterogeneity of the selected patient cohort, and differences in regional dietary habits.

Along with glucosuria, magnesium loss, insufficient magnesium intake, glomerular hyperfiltration, impaired insulin metabolism, diuretic use, and recurrent metabolic acidosis may be listed among the factors leading to a higher frequency of hypomagnesemia in diabetics [12, 14]. According to clinical and epidemiological studies, hypomagnesemia is often seen in patients with poor metabolic control or chronic complications of diabetes [15, 16].

As a component of the Mg^{2+}–ATP complex, magnesium affects blood glucose levels as the cofactor of many carbohydrate metabolism enzymes that catalyze the phosphorylation reactions of the insulin signaling pathway [7, 17]. Magnesium, which modulates insulin action and secretion in target tissues by interacting with insulin receptors, acts as an insulin sensitizer by regulating the tyrosine kinase activity of the insulin receptor and the autophosphorylation of the receptor beta subunit [18, 19]. Magnesium also plays a role in the control of L-type calcium channels, blocking the uptake of calcium into adipocytes. When the intracellular magnesium concentration is reduced, the control of calcium channels is impaired and calcium input to adipocytes increases. This condition leads to insulin resistance by increasing the transcription of inflammatory mediators and oxidative stress [7, 20].

Contradictory findings have been reported in the literature demonstrating that serum magnesium levels were higher [21] or lower [13, 22-25], and indicating the lack of any [26-28] or the presence of a negative correlation [29-32] between serum magnesium and HbA1c levels in patients with diabetes. Therefore, the aim of this study was to further investigate the relationship between serum magnesium level and glucose regulation.

Materials and Methods

In this study, the data pertaining to the results of glucose, magnesium, lipid panel, insulin and HbA1c testing, as well as other simultaneously conducted laboratory tests performed for 256 patients between January 2016 and December 2017, and data related to their age, gender, and prediagnosis, were collected retrospectively from the hospital information system. Only the results of patients who presented at this hospital for the first time were included in the study; recurrent admissions were excluded. In addition, patients who were under 18 or over 65 years of age were not included. Patients with a chronic illness or in need of treatment for anything other than diabetes and dyslipidemia were also excluded from the study. This study was approved by the University of Health Sciences Tepecik Training and Research Hospital Ethics Committee (Decision Date/Number: 04.04.2018/3-8).

The magnesium level was determined using a photometric method, while glucose, total cholesterol, triglyceride, and high-density lipoprotein cholesterol (HDL-C) levels were assessed using an enzymatic method in an AU5800 analyzer (Beckman Coulter Inc., Brea, CA, USA). The low-density lipoprotein cholesterol (LDL-C) level was calculated using the Friedewald formula (LDL-C=total cholesterol–[HDL-C+ triglyceride/5]). Insulin levels were determined by chemiluminescence with a DXI 800 system (Beckman Coulter Inc., Brea, CA, USA), and HbA1c level with high performance liquid chromatography using a Premier Hb9210 analyzer (Trinity Biotech, Bray, Ireland).

The patients were divided into 3 groups: nondiabetics, prediabetics, and diabetics, according to the glucose and HbA1c levels specified in the 2017 criteria of the American Diabetes Association [33]. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) value was calculated using the formula of fasting blood glucose (mg/dL) x fasting insulin level (μU/mL)/405. A HOMA-IR value of ≥2.5 was considered insulin resistance.

Statistical analysis of the data was performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). Conformity of variables with normal distribution was evaluated using the Shapiro-Wilk test. Data with a non-normal distribution were expressed as median (interquartile range). The relationship between variables was evaluated with Spearman correlation analysis. The Kruskal-Wallis H and Mann-Whitney U tests were used for the analysis of continuous variables, and a chi-square test was used for categorical variables. P values below 0.05 were considered statistically significant. In intergroup comparisons, the level of significance was accepted as 0.017 with Bonferroni correction.

Results

Nondiabetic (n=137), prediabetic (n=85), and diabetic (n=34) patients were (median [range]) 40 years (24-55), 45 years (35-58), and 48 years (30-57) of age, respectively (Table 1). A trend toward a lower serum magnesium level was observed with decreasing frequency in the nondiabetic, prediabetic, and diabetic groups, respectively. While there was no statistically significant difference between the magnesium level of the nondiabetic and prediabetic groups ($p=0.038$), a significant difference was observed between the nondiabetic and diabetic groups, and the prediabetic and diabetic groups ($p<0.001$ and $p<0.001$, respectively). In addition, the serum magnesium level was significantly lower in the insulin-resistant group ($p=0.020$) (Table 2).

There was a significant negative correlation between the magnesium level and glucose, HbA1c, and HOMA-IR values ($r=-0.244$, $p<0.001$; $r=-0.332$, $p<0.001$; $r=-0.162$, $p=0.010$, respectively) (Figs. 1-3). There was a significant increase in the triglyceride level in the prediabetic and diabetic patients compared with the nondiabetic group ($p<0.001$ and $p<0.001$, respectively) (Table 1). However, there was no correlation between the serum lipid panel and magnesium

Table 1. Comparison of nondiabetic, prediabetic, and diabetic groups

	Nondiabetic (n=137)	Prediabetic (n=85)	Diabetic (n=34)	P value
Female/male (n)	102/35	69/16	25/9	0.467
Age (years)	40 (24-55)	45 (35-58)	48 (30-57)	0.078
Magnesium (mg/dL)	2.1 (2.0-2.2)	2.1 (2.0-2.1)	1.9 (1.8-2.0)	<0.001*
Glucose (mg/dL)	89 (84-93)	101 (95-104)	150 (113-249)	<0.001*
HbA1c (%)	5.3 (5.1-5.4)	5.8 (5.6-6.0)	7.8 (6.5-9.6)	<0.001*
Insulin (µU/mL)	8.0 (5.0-11.1)	10.1 (6.2-15.7)	12.1 (7.8-30.7)	<0.001*
HOMA-IR	1.7 (0.9-2.5)	2.4 (1.5-4.2)	5.0 (2.7-14.7)	<0.001*
Triglyceride (mg/dL)	103 (73-135)	130 (91-189)	140 (102-221)	<0.001*
Total cholesterol (mg/dL)	203 (173-238)	216 (193-258)	203 (173-245)	0.027*
LDL-C (mg/dL)	126 (104-155)	140 (114-166)	120 (103-156)	0.028*
HDL-C (mg/dL)	52 (44-62)	51 (43-59)	51 (40-61)	0.722

*p<0.05 statistically significant P value.
HbA1c: Glycated hemoglobin; HDL-C: High-density lipoprotein; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; LDL-C: Low-density lipoprotein.

Table 2. Comparison of data according to insulin resistance

	HOMA-IR <2.5 (n=153)	HOMA-IR ≥2.5 (n=103)	P value
Female/male (n)	111/42	85/18	0.065
Age (years)	41 (29-56)	46 (27-58)	0.325
Magnesium (mg/dL)	2.1 (2.0-2.2)	2.0 (1.9-2.1)	0.020*
Glucose (mg/dL)	90 (85-97)	100 (93-113)	<0.001*
HbA1c (%)	5.4 (5.2-5.7)	5.7 (5.4-6.3)	<0.001*
Insulin (µU/mL)	6.3 (4.5-8.2)	15.8 (11.9-27.3)	<0.001*
Triglyceride (mg/dL)	107 (75-143)	130 (92-182)	0.001*
Total cholesterol (mg/dL)	213 (180-254)	203 (171-241)	0.096
LDL-C (mg/dL)	133 (112-164)	127 (103-155)	0.042*
HDL-C (mg/dL)	53 (44-63)	50 (41-57)	0.001*

*p<0.05 statistically significant P value.
HbA1c: Glycated hemoglobin; HDL-C: High-density lipoprotein; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; LDL-C: Low-density lipoprotein.

Table 3. Correlation between magnesium and other parameters

	r	P
Age (years)	–0.061	0.334
Glucose (mg/dL)	–0.244	<0.001*
HbA1c (%)	–0.332	<0.001*
Insulin (µU/mL)	–0.087	0.164
HOMA-IR	–0.162	0.010*
Triglyceride (mg/dL)	–0.068	0.279
Total cholesterol (mg/dL)	0.057	0.360
LDL-C (mg/dL)	0.061	0.332
HDL-C (mg/dL)	0.103	0.099

*p<0.05 statistically significant P value.
HbA1c: Glycated hemoglobin; HDL-C: High-density lipoprotein; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; LDL-C: Low-density lipoprotein.

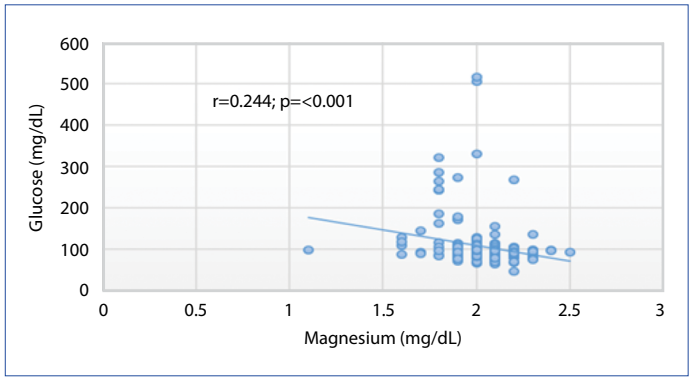

Figure 1. Correlation between serum magnesium and glucose levels.

level (Table 3). When the patients were grouped according to insulin resistance, a significant difference was found in the level of triglycerides, LDL-C, and HDL-C according to the status of the metabolic process (Table 2).

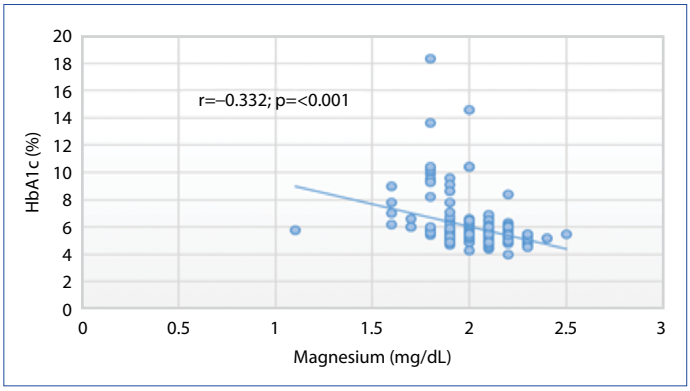

Figure 2. Correlation between serum magnesium and HbA1c levels.

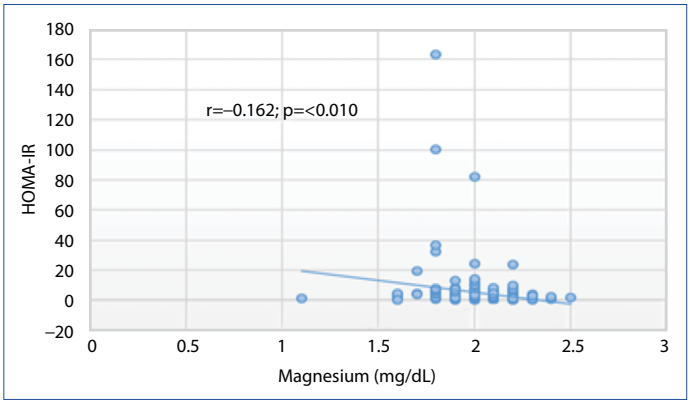

Figure 3. Correlation between serum magnesium and HOMA-IR values.

Discussion

A low level of magnesium is a frequently seen electrolyte disorder in diabetic patients [34]. Magnesium plays an important role in glucose and insulin metabolism, especially its effect on the tyrosine kinase enzyme. Magnesium, which can directly affect the activity of glucose transporter proteins, also regulates entry of glucose into the cell [35]. A study demonstrated that diabetes was an independent risk factor for a low magnesium level in people aged 55 years or older [36]. Such a decrease in magnesium level may occur due to many factors, such as changes in insulin metabolism, poor glycemic control, or osmotic diuresis [12].

As in some studies in the literature, [19, 24] we also found a negative correlation between magnesium and glucose, HbA1c, and HOMA-IR. Chen et al. [37] investigated the relationship between prediabetes and diabetes and magnesium level. Similar to our results, it was reported that the magnesium level decreased consecutively from a normal glucose tolerance group to prediabetes and diabetes groups. These findings suggest that the low level of magnesium seen in diabetic patients has a negative effect on glucose regulation.

In a meta-analysis conducted in 2011, 9 of 13 selected studies reported a statistically significant inverse relationship between magnesium intake and diabetes risk [38]. A daily 100 mg increase in magnesium intake (by diet or supplementation) led to a 15% reduction in the incidence of diabetes [39]. Furthermore, low magnesium levels increase the risk of developing complications of diabetes, while insulin sensitivity and metabolic complications decrease with the addition of magnesium to the diet [13, 40]. Correcting a low magnesium level in diabetic patients may help to prevent diabetes-related complications by eliminating the detrimental effects of oxidative stress [31].

In a randomized double-blind meta-analysis investigating the effects of oral magnesium supplementation on glycemic control in diabetic patients, 370 patients received an average of 15 mmol/day (360 mg/day) magnesium supplementation for 4 to 16 weeks to assess their HbA1c, glucose, and lipid levels. It was concluded that magnesium supplementation significantly decreased the glucose and increased the HDL-C level with long-term beneficial effects [41].

Mahalle et al. [42] reported that diabetes, dyslipidemia, and hypertension were inversely correlated with the serum magnesium level and that patients with a magnesium deficiency had significantly higher total cholesterol, triglyceride, and LDL-C levels, as well as significantly lower HDL-C levels. Corica et al. [43] reported that magnesium supplementation decreased serum total cholesterol and LDL-C levels and increased HDL-C levels. However, some studies have also suggested that there is no relationship between serum lipids and magnesium level [9, 44]. Although a significant increase in the triglyceride level was observed in the prediabetic and diabetic patients in our study, as expected from a metabolic perspective, no significant correlation was found between triglyceride or other lipid panel markers and serum magnesium level.

Our study has some limitations. In order to avoid any effect due to different devices or methodological changes, the patient data evaluated were from a confined time interval. Limited clinical prediagnostic data derived from the hospital information management system were used to group patients into categories of prediabetes and diabetes according to their fasting blood glucose and HbA1c values. Since there were no test data for an oral glucose tolerance test for these patients, only a finding of impaired fasting glucose was used for the definition of prediabetes. Since impaired glucose tolerance could not assessed simultaneously, these patients may have shifted to the nondiabetic group. Therefore, an inevitable selection bias exists in our study, as seen in other retrospective studies.

As a result, though there are contradictory data in the literature, it has been reported in most studies that a low magnesium level has a negative effect on glucose regulation, as observed in our study as well. In diabetic patients, glycemic control should be improved to prevent osmotic diuresis, which is the most important cause of magnesium deficiency. A diet rich in magnesium and/or oral magnesium supplementation may help to increase insulin sensitivity and prevent diabetes-related complications in diabetic patients with a reduced serum magnesium level.

Conclusion

Despite the contradictory data in the literature, it has been reported in the most of the studies that low magnesium levels have a negative effect on glucose regulation, similar to our study. In diabetic patients, glycemic control should be improved to prevent osmotic diuresis which is the most important cause of magnesium deficiency. A diet rich in magnesium and / or oral magnesium supplementation may help to increase insulin sensitivity and prevent diabetes-related complications in diabetic patients with reduced serum magnesium levels.

Conflict of interest: The authors of this paper have no conflicts of interest, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included.

Ethics Committee Approval: The Ethics Committee of the University of Health Sciences Tepecik Training and Research Hospital approved the study (Decision Date/Number: 04.04.2018/3-8).

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