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Original Research Article

The Influence of Magnesium on Blood Glucose Control

Kiran R. Bagale, Dinesh Kumar Sharma*

Department of Biochemistry, Shri Balaji Institute of Medical Science, Raipur, Chhattisgarh

***Correspondence:** Dr. Dinesh Kumar Sharma (dksharma0305@gmail.com)

ABSTRACT

Background: Magnesium acts as a cofactor in glucose phosphorylation and numerous other enzymatic reactions. Its deficiency may contribute to insulin resistance, carbohydrate intolerance, and dyslipidemia. Reduced serum magnesium levels have been linked to the development of diabetic complications, including retinopathy, altered platelet function, cardiovascular disease, and hypertension. The present study aimed to examine the association between serum magnesium levels and glucose regulation.

Materials and methods: Data from 256 patients who underwent serum testing for glucose, magnesium, glycated hemoglobin (HbA1c), insulin, and lipid profile between May 2024 and June 2025 were retrospectively analyzed. Based on glucose and HbA1c values defined by the 2017 American Diabetes Association guidelines, participants were categorized into three groups: non-diabetic, prediabetic, and diabetic. Intergroup comparisons were conducted using the Kruskal–Wallis H test and the Mann–Whitney U test.

Results: Among the participants, 137 were nondiabetic, 85 were prediabetic, and 34 were diabetic, with median ages (range) of 40 years (24–55), 45 years (35–58), and 48 years (30–57), respectively. Magnesium levels differed significantly between the nondiabetic and diabetic groups, as well as between the prediabetic and diabetic groups (both $p<0.001$). However, no significant difference was observed between the nondiabetic and prediabetic groups. Patients with insulin resistance (HOMA-IR ≥2.5) had significantly lower magnesium levels ($p=0.020$). Furthermore, serum magnesium showed a significant negative correlation with glucose ($r=–0.244$, $p<0.001$), HbA1c ($r=–0.332$, $p<0.001$), and HOMA-IR values ($r=–0.162$, $p=0.010$).

Conclusion: Magnesium serves as a cofactor for several enzymes involved in carbohydrate oxidation and is essential for glucose transport across the cell membrane. This study found that reduced magnesium levels adversely influenced glucose regulation. Incorporating magnesium-rich foods or providing oral magnesium supplementation may help improve insulin sensitivity in diabetic patients with low serum magnesium.

Keywords: Diabetes mellitus, Insulin, Magnesium, Pre-diabetes

INTRODUCTION

Magnesium forms an essential component of the cellular membrane structure, contributing to its stability[1,2]. Acting as a cofactor for numerous enzymes involved in energy metabolism, it plays a vital role in carbohydrate, lipid, and protein metabolism. In addition, magnesium is involved in the activity of several enzymes associated with glucose oxidation and insulin secretion[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 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 part of the Mg^{2+}–ATP complex, magnesium influences blood glucose regulation by serving as a cofactor for several carbohydrate metabolism enzymes involved in phosphorylation reactions within the insulin signaling pathway[7,17]. By interacting with insulin receptors, magnesium modulates both insulin action and secretion in target tissues, acting as an insulin sensitizer through regulation of receptor tyrosine kinase activity and autophosphorylation of the receptor β-subunit[18,19]. In addition, magnesium regulates L-type calcium channels by limiting calcium influx into adipocytes. A decline in intracellular magnesium disrupts this control, leading to increased calcium entry, which promotes insulin resistance via enhanced transcription of inflammatory mediators and oxidative stress[7,20]. Previous studies have reported inconsistent findings regarding serum magnesium levels in diabetes, with some showing elevated values[21], others decreased[13,22–25], and some reporting no association[26–28] or a negative correlation[29–32] between serum magnesium and HbA1c. Given these conflicting results, the present study aimed to further evaluate the relationship between serum magnesium levels and glucose regulation.

MATERIALS AND METHODS

In this study, retrospective data were collected from the hospital information system, including results of glucose, magnesium, lipid profile, insulin, and HbA1c tests, along with other concurrent laboratory investigations, for 256 patients evaluated between May 2024 and June 2025.

Demographic information such as age, sex, and preliminary diagnosis was also recorded. Only first-time admissions to the hospital were considered, while repeat visits were excluded. Patients younger than 18 years or older than 65 years, as well as those with chronic diseases or requiring treatment for conditions other than diabetes and dyslipidemia, were not included.

Serum magnesium concentrations were measured using a photometric method. Glucose, total cholesterol, triglycerides, and high-density lipoprotein cholesterol (HDL-C) were analyzed with an enzymatic method on an AU480 analyzer (Beckman Coulter Inc.). Low-density lipoprotein cholesterol (LDL-C) levels were calculated using the Friedewald formula: LDL-C = total cholesterol – (HDL-C + triglyceride/5). Insulin concentrations were determined by chemiluminescence on the DXI 800 system (Beckman Coulter Inc.), while HbA1c was assessed using Lifotronic GH-900 Plus analyser. Patients were categorized into three groups—nondiabetic, prediabetic, and diabetic—based on glucose and HbA1c levels in accordance with the 2017 American Diabetes Association criteria[33]. Insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), calculated as: fasting blood glucose (mg/dL) × fasting insulin (μU/mL) / 405. A HOMA-IR value ≥2.5 was taken to indicate insulin resistance.

Statistical analyses were conducted using IBM SPSS Statistics for Windows, Version 21.0. The Shapiro–Wilk test was applied to assess the normality of variable distributions. Non-normally distributed data were presented as median (interquartile range). Associations between variables were examined using Spearman's correlation analysis. For continuous variables, intergroup comparisons were performed using the Kruskal–Wallis H test and the Mann–Whitney U test, while categorical variables were evaluated with the chi-square test. A p-value of <0.05 was considered statistically significant; however, for pairwise intergroup comparisons, the significance threshold was adjusted to 0.017 using the 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 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).

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)	85/18	111/42	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.

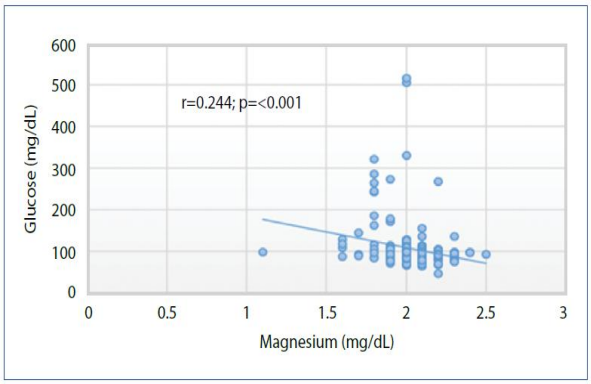

Figure-1: Correlation between serum magnesium and glucose levels.

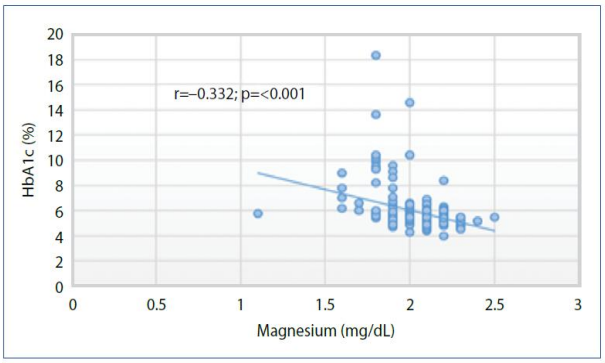

Figure-2: Correlation between serum magnesium and HbA1c levels.

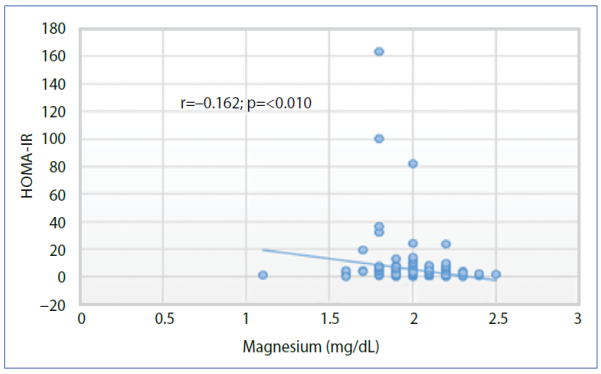

Figure-3: Correlation between serum magnesium and HOMA-IR values.

DISCUSSION

Hypomagnesaemia is a common electrolyte disturbance in diabetic patients[34]. Magnesium is essential for glucose and insulin metabolism, particularly through its influence on the tyrosine kinase enzyme. By modulating the activity of glucose transporter proteins, magnesium also regulates cellular glucose uptake[35]. One study reported that diabetes was an independent risk factor for reduced magnesium levels in individuals aged 55 years and older[36]. Several mechanisms may contribute to this decline, including alterations in insulin metabolism, poor glycemic control, and osmotic diuresis[12]. Consistent with previous studies[19,24], we also observed a negative correlation between magnesium levels and glucose, HbA1c, and HOMA-IR. Chen et al.[37] examined the association between magnesium levels, prediabetes, and diabetes, and reported findings similar to ours: magnesium levels progressively declined from the normal glucose tolerance group to the prediabetic and diabetic groups. These results suggest that reduced magnesium levels in diabetic patients may adversely affect glucose regulation. A 2011 meta-analysis reviewed 13 studies, 9 of which demonstrated a significant inverse relationship between magnesium intake and diabetes risk[38]. It was further reported that each 100 mg/day increase in dietary or supplemental magnesium intake was associated with a 15% reduction in diabetes incidence[39]. Moreover, magnesium deficiency has been linked to a higher risk of diabetic complications, whereas supplementation improves insulin sensitivity and reduces metabolic complications[13,40]. Restoring magnesium levels in diabetic patients may therefore help mitigate diabetes-related complications by reducing oxidative stress[31]. In a randomized, double-blind meta-analysis evaluating the effect of oral magnesium supplementation on glycemic control, 370 diabetic patients received an average of 15 mmol/day (≈360 mg/day) of magnesium for 4–16 weeks. The results showed significant reductions in glucose levels and increases in HDL-C, with long-term benefits for metabolic control[41]. Mahalle et al.[42] reported inverse correlations between serum magnesium levels and diabetes, dyslipidemia, and hypertension, noting that magnesium deficiency was associated with significantly higher total cholesterol, triglyceride, and

LDL-C levels, along with lower HDL-C levels. Similarly, Corica et al.[43] found that magnesium supplementation reduced total cholesterol and LDL-C while increasing HDL-C. In contrast, other studies have suggested no clear relationship between serum magnesium and lipid parameters[9,44]. In our study, although triglyceride levels were significantly elevated in both prediabetic and diabetic patients—as expected from a metabolic standpoint—no significant association was observed between serum magnesium and triglycerides or other lipid profile markers. This study has certain limitations. To minimize variability related to device differences or methodological changes, data collection was restricted to a defined time period. Patient classification into prediabetic and diabetic groups was based solely on fasting glucose and HbA1c values available in the hospital information system. Oral glucose tolerance test results were not available, and therefore impaired glucose tolerance could not be assessed. As a result, some patients with impaired glucose tolerance may have been misclassified into the nondiabetic group, leading to potential selection bias, which is a common limitation of retrospective studies. Despite conflicting reports in the literature, most studies, including ours, indicate that low serum magnesium adversely affects glucose regulation. For diabetic patients, optimizing glycemic control is important to prevent osmotic diuresis—the major contributor to magnesium depletion. Furthermore, a magnesium-rich diet and/or oral magnesium supplementation may enhance insulin sensitivity and reduce the risk of diabetes-related complications in patients with magnesium deficiency.

CONCLUSIONS

Although findings in the literature are somewhat inconsistent, most studies, including ours, indicate that low magnesium levels negatively affect glucose regulation. In diabetic patients, optimizing glycemic control is essential to prevent osmotic diuresis, the primary contributor to magnesium deficiency. Incorporating a magnesium-rich diet and/or providing oral magnesium supplementation may improve insulin sensitivity and help reduce the risk of diabetes-related complications in patients with low serum magnesium.

REFERENCES

1. Fawcett WJ, Haxby EJ, Male DA. Magnesium: physiology and pharmacology. Br J Anaesth. 1999;83(2):302-20.
2. Jahnen-Dechent W, Ketteler M. Magnesium basics. Clin Kidney J. 2012;5(Suppl 1):i3-14.
3. Goldman J, Fisher V. Magnesium is required in addition to calcium for insulin stimulation of glucose transport. Endocrinology. 1983;112(1):271-5.
4. Yajnik CS, Smith RF, Hockaday TD, Ward NI. Fasting plasma magnesium concentrations and glucose disposal in diabetes. Br Med J (Clin Res Ed). 1984;288(6423):1032-4.
5. Sundaram G, Ramakrishnan T, Parthasarathy H, Moses J, Lalitha T. Evaluation of micronutrient (zinc, magnesium, and copper) levels in serum and glycemic status after nonsurgical periodontal therapy in type 2 diabetic patients with chronic periodontitis. Contemp Clin Dent. 2017;8(1):26-32.
6. Chutia H, Lynrah KG. Association of serum magnesium deficiency with insulin resistance in type 2 diabetes mellitus. J Lab Physicians. 2015;7(2):75-8.
7. Cruz KJ, de Oliveira AR, Pinto DP, Morais JB, Lima FS, Colli C, et al. Influence of magnesium on insulin resistance in obese women. Biol Trace Elem Res. 2014;160(3):305-10.
8. Yadav C, Manjrekar PA, Agarwal A, Ahmad A, Hegde A, Srikantiah RM. Association of serum selenium, zinc and magnesium levels with glycaemic indices and insulin resistance in pre-diabetes: a cross-sectional study from South India. Biol Trace Elem Res. 2017;175(1):65-71.

9. Corsonello A, Ientile R, Buemi M, Cucinotta D, Mauro VN, Macaione S, et al. Serum ionized magnesium levels in type 2 diabetic patients with microalbuminuria or clinical proteinuria. Am J Nephrol. 2000;20(3):187-92.
10. Pickup JC, Chusney GD, Crook MA, Viberti GC. Hypomagnesaemia in IDDM patients with microalbuminuria and clinical proteinuria. Diabetologia. 1994;37(6):639-43.
11. Allegra A, Corsonello A, Buemi M, D'Angelo R, Di Benedetto A, Bonanzinga S, et al. Plasma, erythrocyte and platelet magnesium levels in type 1 diabetic patients with microalbuminuria and clinical proteinuria. J Trace Elem Med Biol. 1997;11(3):154-7.
12. Pham PC, Pham PM, Pham SV, Miller JM, Pham PT. Hypomagnesemia in patients with type 2 diabetes. Clin J Am Soc Nephrol. 2007;2(2):366-73.
13. Dasgupta A, Sarma D, Saikia UK. Hypomagnesemia in type 2 diabetes mellitus. Indian J Endocrinol Metab. 2012;16(6):1000-3.
14. Liamis G, Liberopoulos E, Barkas F, Elisaf M. Diabetes mellitus and electrolyte disorders. World J Clin Cases. 2014;2(10):488-96.
15. Tosiello L. Hypomagnesemia and diabetes mellitus: a review of clinical implications. Arch Intern Med. 1996;156(11):1143-8.
16. Ma J, Folsom AR, Melnick SL, Eckfeldt JH, Sharrett AR, Nabulsi AA, et al. Associations of serum and dietary magnesium with cardiovascular disease, hypertension, diabetes, insulin, and carotid arterial wall thickness: the ARIC study. J Clin Epidemiol. 1995;48(7):927-40.
17. Mooren FC, Krüger K, Völker K, Golf SW, Wadepuhl M, Kraus A. Oral magnesium supplementation reduces insulin resistance in non-diabetic subjects: a double-blind, placebo-controlled, randomized trial. Diabetes Obes Metab. 2011;13(3):281-4.
18. Guerrero-Romero F, Rodríguez-Morán M. Magnesium improves the beta-cell function to compensate variation of insulin sensitivity: double-blind, randomized clinical trial. Eur J Clin Invest. 2011;41(4):405-10.
19. Sales CH, Pedrosa LF. Magnesium and diabetes mellitus: their relation. Clin Nutr. 2006;25(4):554-62.
20. Latham JR, Pathirathna S, Jagodic MM, Choe WJ, Levin ME, Nelson MT, et al. Selective T-type calcium channel blockade alleviates hyperalgesia in ob/ob mice. Diabetes. 2009;58(11):2656-65.
21. Raz I, Havivi E. Trace elements in blood cells of diabetic subjects. Diabetes Res. 1989;10(1):21-4.
22. Lind L, Lithell H, Hvarfner A, Ljungall S. Indices of mineral metabolism in subjects with an impaired glucose tolerance. Exp Clin Endocrinol. 1990;96(1):109-12.
23. Rohn RD, Pleban P, Jenkins LL. Magnesium, zinc, and copper in plasma and blood cellular components in children with IDDM. Clin Chim Acta. 1993;215(1):21-8.
24. Kim DJ, Xun P, Liu K, Loria C, Yokota K, Jacobs DR Jr, et al. Magnesium intake in relation to systemic inflammation, insulin resistance, and the incidence of diabetes. Diabetes Care. 2010;33(12):2604-10.

25. Sales CH, Pedrosa LF, Lima JG, Lemos TM, Colli C. Influence of magnesium status and magnesium intake on the blood glucose control in patients with type 2 diabetes. Clin Nutr. 2011;30(3):359-64.
26. Galli-Tsinopoulou A, Maggana I, Kyrgios I, Mouzaki K, Grammatikopoulou MG, Stylianou C, et al. Association between magnesium concentration and HbA1c in children and adolescents with type 1 diabetes mellitus. J Diabetes. 2014;6(4):369-77.
27. Ramadass S, Basu S, Srinivasan AR. Serum magnesium level as an indicator of status of diabetes mellitus type 2. Diabetes Metab Syndr. 2015;9(1):42-5.
28. Sinha S, Sen S. Status of zinc and magnesium levels in type 2 diabetes mellitus and its relationship with glycemic status. Int J Diabetes Dev Ctries. 2014;34(4):220-3.
29. Mikhail N, Ehsanipoor K. Ionized serum magnesium in type 2 diabetes mellitus: its correlation with total serum magnesium and hemoglobin A1c levels. South Med J. 1999;92(12):1162-6.
30. Salmonowicz B, Krzystek-Korpacka M, Noczyńska A. Trace elements, magnesium, and the efficacy of antioxidant systems in children with type 1 diabetes mellitus and in their siblings. Adv Clin Exp Med. 2014;23(2):259-68.
31. Matthiesen G, Olofsson K, Rudnicki M. Ionized magnesium in Danish children with type 1 diabetes. Diabetes Care. 2004;27(5):1216-7.
32. Wegner M, Araszkiewicz A, Zozulińska-Ziółkiewicz D, Wierusz-Wysocka B, Pioruńska-Mikołajczak A, Pioruńska-Stolzmann M. The relationship between concentrations of magnesium and oxidized low density lipoprotein and the activity of platelet activating factor acetylhydrolase in the serum of patients with type 1 diabetes. Magnes Res. 2010;23(3):97-104.
33. American Diabetes Association. Standards of medical care in diabetes-2017. Diabetes Care. 2017;40(Suppl 1):S4-5.
34. Song Y, Manson JE, Buring JE, Liu S. Dietary magnesium intake in relation to plasma insulin levels and risk of type 2 diabetes in women. Diabetes Care. 2004;27(1):59-65.
35. Suárez A, Pulido N, Casla A, Casanova B, Arrieta FJ, Rovira A. Impaired tyrosine-kinase activity of muscle insulin receptors from hypomagnesaemic rats. Diabetologia. 1995;38(11):1262-70.
36. Liamis G, Rodenburg EM, Hofman A, Zietse R, Stricker BH, Hoorn EJ. Electrolyte disorders in community subjects: prevalence and risk factors. Am J Med. 2013;126(3):256-63.
37. Chen S, Jin X, Liu J, Sun T, Xie M, Bao W, et al. Association of plasma magnesium with prediabetes and type 2 diabetes mellitus in adults. Sci Rep. 2017; 7:12763.
38. Dong JY, Xun P, He K, Qin LQ. Magnesium intake and risk of type 2 diabetes: meta-analysis of prospective cohort studies. Diabetes Care. 2011;34(9):2116-22.
39. Larsson SC, Wolk A. Magnesium intake and risk of type 2 diabetes: a meta-analysis. J Intern Med. 2007;262(2):208-14.

40. Guerrero-Romero F, Simental-Mendía LE, Hernández-Ronquillo G, Rodriguez-Morán M. Oral magnesium supplementation improves glycaemic status in subjects with prediabetes and hypomagnesaemia: a double-blind placebo-controlled randomized trial. Diabetes Metab. 2015;41(3):202-7.
41. Song Y, He K, Levitan EB, Manson JE, Liu S. Effects of oral magnesium supplementation on glycemic control in type 2 diabetes: a meta-analysis of randomized double-blind controlled trials. Diabet Med. 2006;23(10):1050-6.
42. Mahalle N, Kulkarni MV, Naik SS. Is hypomagnesaemia a coronary risk factor among Indians with coronary artery disease? J Cardiovasc Dis Res. 2012;3(4):280-6.
43. Corica F, Allegra A, Di Benedetto A, Giacobbe MS, Romano G, Cucinotta D, et al. Effects of oral magnesium supplementation on plasma lipid concentrations in patients with non-insulin-dependent diabetes mellitus. Magnes Res. 1994;7(1):43-7.
44. Arpaci D, Tocoglu AG, Ergenc H, Korkmaz S, Ucar A, Tamer A. Associations of serum magnesium levels with diabetes mellitus and diabetic complications. Hippokratia. 2015;19(2):153-7.

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