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CASE REPORT

Effect of magnesium supplementation on glycemic control in type 2 diabetes mellitus: evidence-based case report

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Abstract

Background: Diabetes mellitus is one of the most serious and common chronic diseases, leading to life-threatening, disabling and costly complications, and reducing life expectancy. Poor intracellular magnesium concentration may contribute to insulin resistance, whereas higher magnesium levels are associated with increased insulin sensitivity. This study aimed to investigate the effect of magnesium supplementation on glycemic control in patients with type 2 diabetes.

Objective: This study aimed to investigate the effect of magnesium supplementation on glycemic control in patients with type 2 diabetes.

Methods: A literature search was conducted using three major databases: PubMed, Cochrane Library, and EBSCOhost. MeSH terms, advanced search, and eligibility criteria were used for title and abstract screening after removing duplicates. Critical assessment tools and levels of evidence of the final articles are based on the Oxford Center for Evidence-Based Medicine.

Results: A meta-analysis and two RCTs met the PICO and eligibility criteria. One meta-analysis found that magnesium supplementation significantly reduced fasting blood glucose and HbA1c. One RCT reported that magnesium supplementation significantly improved HbA1c, insulin levels, and HOMA-IR. Another RCT found that there were no differences in HbA1C and continuous glucose monitoring.

Conclusion: Magnesium supplementation may have a beneficial effect on glycemic control in patients with type 2 diabetes. However, further research is needed to establish optimal dosage and the most effective form of magnesium supplementation.

Keywords: blood glucose, glycated hemoglobin, glycemic control, magnesium supplementation, type 2 diabetes mellitus

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Case scenario

Mr. IR, a 45-year-old male, visited the nutrition clinic complaining of fatigue, body aches, and poor sleep quality. He brought his laboratory results, which showed a fasting blood glucose level 160 mg/dL, a 2-hour post prandial blood glucose level of 220 mg/dL, and HbA1c of 7%. The patient had been diagnosed with type 2 diabetes and had been taking metformin 500 mg twice a day for the past four months. He wanted to modify his diet to improve glycemic control. The patient had heard that magnesium supplementation could help

improve blood glucose and HbA1c, so he asked the clinical nutrition specialist if it would be appropriate for him to take the supplement.

Introduction

Diabetes is a major global health issue and is recognized as one of the most important non-communicable diseases driven by unhealthy modern lifestyles.[1] The International Diabetes Federation (IDF) estimated that 536.6 million people worldwide were living with diabetes (diagnosed or undiagnosed) in 2021, and this number is expected to increase by 46%, reaching 783.2 million by 2045. According to the IDF, Indonesia ranks fifth among countries with the highest number of diabetes cases globally. The prevalence of diabetes in Indonesia was 10.6% in 2021 and increased to 11.7% in 2023.[2]

Diabetes mellitus can impact individuals in various ways, ranging from acute threats due to decompensated metabolism leading to severe hyperglycemic hyperosmolar coma, ketoacidosis, or severe hypoglycemia, to long-term serious complications affecting both large and small blood vessels as well as the nervous system. Additionally, it poses lifelong challenges to quality of life due to a wide range of psychosocial problems.[1] Studies have shown that individuals can remain in an asymptomatic phase of prediabetes and type 2 diabetes mellitus for 5-6 years before being diagnosed, during which microvascular and macrovascular complications may arise. Therefore, maintaining good glycemic control is fundamental in preventing diabetes-related complications.[2]. Self-monitored blood glucose, flash glucose monitoring, continuous glucose monitoring, and glycated hemoglobin (HbA1c) are the best information for assessing glycemic control.[3]

Type 2 diabetes is frequently associated with magnesium deficiency in both extracellular and intracellular compartments, which is related to hyperglycemia. An increased prevalence of magnesium deficiency has been observed in patients with type 2 diabetes, particularly in those with poor glycemic control, longer duration of the disease, and presence of microvascular and macrovascular complications.[4] Previous studies have shown that hypomagnesemia occurs in 9–48% of individuals with type 2 diabetes, and low serum magnesium levels have been implicated in the pathogenesis of type 2 diabetes and its cardiovascular complications.[5]

Magnesium ion plays a major role in carbohydrate metabolism and insulin function. Magnesium acts as a cofactor in glucose transport across cell membranes and in enzymes involved in carbohydrate oxidation and also plays a role in insulin release.[6] Magnesium improves insulin sensitivity by modulating tyrosine kinase activity of insulin receptors and promoting their autophosphorylation. Magnesium also inhibits calcium entry into adipocytes. Reduced intracellular magnesium levels lead to calcium accumulation in adipocytes, which subsequently increases inflammation, oxidative stress, and insulin resistance.[7] Several studies have reported that insulin resistance and/or hyperinsulinemia reduces renal tubular reabsorption of magnesium, increased magnesium excretion, and promotes its shift from extracellular to intracellular compartments. Additionally, inadequate magnesium intake is another major contributor to hypomagnesemia in diabetic patients.[8]

Accumulating evidence has shown that increased magnesium intake improved insulin release and sensitivity, alleviated dyslipidemia, mitigated endothelial cells dysfunction, and reduced thrombotic risk and vascular contractility.[9] A recent meta-analysis revealed that, compared to a placebo, magnesium supplementation enhances glucose and insulin sensitivity markers in individuals with diabetes or conditions that increase the risk of developing diabetes, such as obesity.[10] Some studies have demonstrated that magnesium supplementation is associated with improved glycemic control and could prevent chronic complications of diabetes. However, other studies have not demonstrated such results.[11,12] The available clinical trials are still insufficient to establish guidelines for clinical practice. Therefore, this study was conducted to evaluate the effect of magnesium supplementation on glycemic control in patients with type 2 diabetes.

Clinical question

P : Type 2 diabetes mellitus patients
I : Magnesium supplementation
C : Placebo
O : Glycemic control
Clinical Question: Could magnesium supplementation help control glycemia in patients with type 2 diabetes mellitus?

Methods

A literature search was performed using a combination of MeSH terms and Title/Abstract across three large databases: PubMed, Cochrane Library, and EBSCOhost. The search was conducted on September 10th, 2024. The keywords used were blood glucose, glycated hemoglobin, glycemic control, magnesium supplementation, and type 2 diabetes mellitus. Critical appraisal tools and determination of the level of evidence were created based on the Oxford Centre for Evidence-Based Medicine.

Eligibility criteria

The inclusion criteria consist of subjects aged 18 years or older with type 2 diabetes mellitus who received magnesium supplementation. Eligible studies must be randomized controlled trials (RCTs), systematic reviews, or meta-analyses that report glycemic control outcome, were published between 2020-2024, and were written in English. The exclusion criteria encompass patients with type 1 diabetes mellitus, pregnant or lactating women, patients with malignancy, animal studies, and articles without full-text availability.

Results

The author found 35 articles in the PubMed database, 17 articles in the Scopus database, and 6 articles in EBSCOhost (**Table 1**). Duplicate removal was conducted using *Covidence.* The articles were evaluated for eligibility based on PICO and the eligibility criteria (**Figure 1**), resulting in the selection of three studies. The characteristics of these studies are detailed in **Table 2,** and their levels of evidence are presented in **Table 3**. All selected studies relevant to answering the clinical question are presented in **Table 4**.

Discussion

Type 2 diabetes mellitus is a chronic metabolic disorder caused by impaired insulin secretion by pancreatic β-cells, insulin resistance in target organs, or both, resulting in hyperglycemia. Although individual susceptibility to type 2 diabetes due to non-modifiable risk factors like ethnicity and family history has a strong genetic basis, epidemiological studies suggest that many cases of type 2 diabetes can be prevented by improving the key modifiable factors such as obesity, physical activity, and unhealthy diet.[14] Mechanisms for development of microvascular and macrovascular complications due to hyperglycemia include endothelial dysfunction, advanced glycation end-product formation, hypercoagulability, increased platelet reactivity, and sodium-glucose co-transporter-2 (SGLT-2) hyperexpression. The incretin effect, alterations in the gut microbiome, immune dysregulation, and inflammation have been identified as significant pathophysiological factors. Compared with individuals without diabetes, patients with type 2 diabetes have a 15% increased risk of all-cause mortality. This is twice as high in young individuals, particularly those younger than 55 years of age with glycated haemoglobin (HbA1c) levels of 6.9% or lower, compared with individuals without diabetes.[15]

Magnesium is the second most abundant intracellular cation after potassium and the fourth most common mineral in the human body after calcium, potassium, and sodium.[4,15] As a cofactor of >600 enzymes, magnesium plays a role in various metabolic pathways, such as glycolysis, β-oxidation, and insulin signaling.[16] The kidneys regulate normal plasma magnesium levels within the range of 1.7 to 2.4 mg/dL.[6] Magnesium deficiency can occur without hypomagnesemia. However, when hypomagnesemia is present, it typically indicates a significant systemic magnesium deficiency.[4,15] Signs and symptoms of

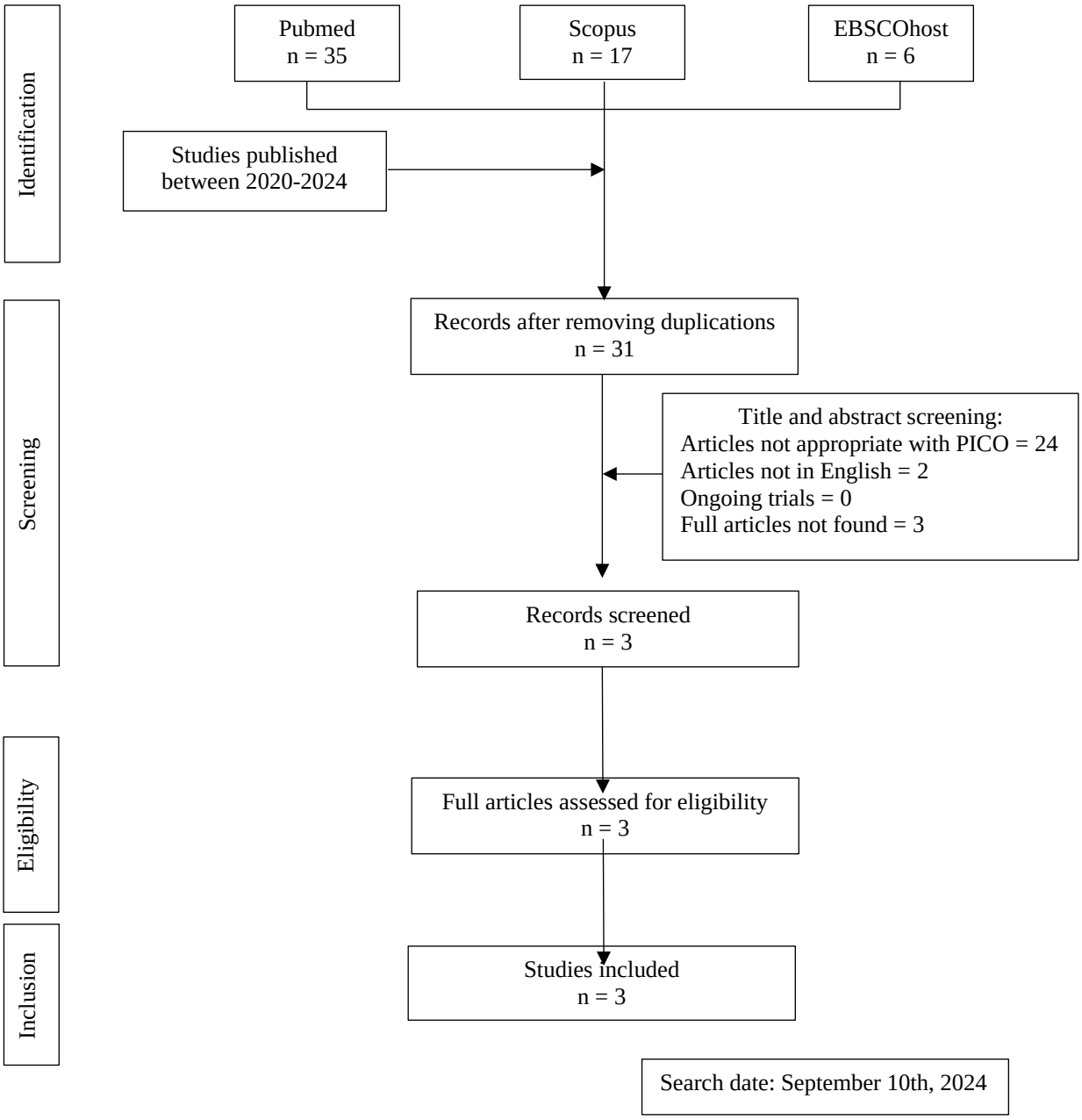

Figure 1. Prisma's flow chart

Table 1. Resources and search strategy

Database	Terminology	Hits	Eligible
Pubmed	(((diabetes[MeSH Terms]) OR (diabetes[Title/Abstract])) AND ((((magnesium supplementation[MeSH Terms]) OR (Mg supplementation[MeSH Terms])) OR (magnesium supplementation[Title/Abstract])) OR (Mg supplementation[Title/Abstract]))) AND (((((((blood glucose[MeSH Terms]) OR (plasma glucose[MeSH Terms])) OR (glycated hemoglobin[MeSH Terms])) OR (glycemic control[MeSH Terms])) OR (blood glucose[Title/Abstract])) OR (plasma glucose[Title/Abstract])) OR (glycated hemoglobin[Title/Abstract])) OR (glycemic control[Title/Abstract])) Filters: Meta-Analysis, Randomized Controlled Trial, Systematic Review	35	3
Scopus	TITLE-ABS-KEY (diabetes AND mellitus) AND TITLE-ABS-KEY (magnesium) AND TITLE-ABS-KEY (blood AND glucose OR plasma AND glucose OR glycemic AND control OR glycated AND hemoglobin) AND TITLE-ABS-KEY (randomized AND controlled AND trial OR systematic AND review OR meta-analysis)	17	1
EBSCOhost	magnesium AND diabetes mellitus AND (glycemic control or HbA1c or glycated hemoglobin or blood glucose) AND (systematic review or meta-analysis or randomized control trial)	6	1

Table 2. Study characteristics

No.	Author	Study design	Population characteristics	Number of subjects	Outcomes	Results
1.	Asbaghi et al. (2022)	Systematic review and meta-analysis	Adults with type 2 diabetes who received an oral elemental magnesium dose ranging from 36.49 to 500 mg/day for 4 to 24 weeks	1097 (18 RCTs)	-HbA1C -Fasting blood sugar	The estimated mean difference in HbA1c at 500 mg/d was −0.73% (95% CI: −1.25, −0.22, *p*= 0.004). Fasting blood sugar (FBS) at 360 mg/d was −7.11 mg/dl (95% CI: −14.03, −0.19, *p*=0.092). The mean difference in FBS and HbA1c at 24 weeks was estimated to be −15.58 mg/dl (95 % CI: −24·67, −6·49, *p*=0.034) and −0.48% (95% CI: −0.77, −0.19, *p*=0.001), respectively.
2.	Albaker et al. (2022)	RCT, double blind	Adults with type 2 diabetes and normal baseline magnesium levels who received magnesium chloride added to desalinated drinking water and were distributed into three groups: group A (0 mg/L), group B (20 mg/L), and group C (50 mg/L) for 3 months	102	-HbA1C -HOMA.IR -Insulin level -Fasting blood glucose -C-peptide	The median level of HbA1c showed a significant improvement (8.0 vs 8.2%, *p*=0.04) along with median insulin levels (7.5 vs 9.9 µIU/mL, *p*=0.03), and homeostasis model assessment- estimated insulin resistance (HOMA-IR) (2.5 vs 2.9, *p*=0.002) in group C (high dose) after three months compared to baseline value. However,

No.	Author	Study design	Population characteristics	Number of subjects	Outcomes	Results
						there were no significant changes in FBS level C-peptide.
3.	Drenthen et al. (2024)	RCT, double blind	Adults (aged ≥18 years) with type 2 diabetes who had been treated with insulin for at least 1 year and had a serum magnesium concentration ≤0.79 mmol/l received oral magnesium gluconate or placebo as a liquid solution (50 mL) three times a day (equivalent to 360 mg) for 6 weeks	14	-Mean glucose infusion rate (GIR) during the final 30 min of the clamp (*M* value) -Continuous glucose monitoring outcomes, HbA1c, insulin dose, lipid profile and blood pressure	The *M* value of the glucose clamp did not differ between the magnesium and placebo study arms (4.6 ± 0.5 vs 4.4 ± 0.6 mg/kg/min, *p*=0.108). Glucose monitoring outcomes, HbA1c, insulin dose, lipid profile, and blood pressure also did not differ, except for a lower HDL-cholesterol concentration after magnesium supplementation compared with placebo (1.14 ± 0.08 vs 1.20 ± 0.09 mmol/l, *p*=0.026).

Table 3. Validity criteria

	Study design	Number of patients	Randomization	Similarity treatment and control	Blinding comparable treatment	Domain	Determinant	Measurement of outcomes	Quality of evidence	Level of evidence
Asbaghi *et al.* (2022)[12]	+	+	+	+	+	+	+	+	Moderate	1A
Albaker et al. (2022)[8]	+	+	+	+	+	+	+	+	Moderate	1B
Drenthen *et al.* (2024)[13]	+	+	+	+	+	+	+	+	Moderate	1B

* Quality of evidence according to GRADE guidelines, https://www.ncbi.nlm.nih.gov/pubmed/21208779
**Level of evidence according to Oxford Center of Evidence-based Medicine (CEBM), http://www.cebm.net.
+ clearly mentioned in the article; - not done; ? Not stated clearly
- Systematic review and meta-analysis with troublesome heterogeneity

Table 4. Relevance Criteria

Article	Similarity Population	Similarity determinant/intervention /indicators	Similarity outcome
Asbaghi et al. (2022)[12]	+	+	+
Albaker et al. (2022)[8]	+	+	+
Drenthen et al. (2024)[13]	+	+	+

hypomagnesemia usually appear when serum magnesium levels drop below 1.2 mg/dL. Various factors can negatively affect magnesium balance in the body, including reduced magnesium intake from food or water, increased magnesium loss through the kidneys, and prolonged use of certain medications that induce hypomagnesemia.[15]

Hypomagnesemia is associated with insulin resistance, decreased pancreatic insulin secretion, impaired cellular glucose transport, impaired glucose tolerance and rapid decline in kidney function. Consequently, hypomagnesemia in diabetes patients can accelerate disease progression and risk of end-stage kidney disease, cardiovascular disease, nephropathy, retinopathy and foot ulcers.[12,16] Intracellular magnesium plays a critical role in regulating glucokinase, K-ATP channels, and L-type Ca^{2+} channels in pancreatic β-cells, which are essential for insulin secretion. Moreover, the autophosphorylation of insulin receptors relies on intracellular magnesium levels, making magnesium a key factor in the development of insulin resistance. Conversely, insulin is an important regulator of magnesium homeostasis. In the kidney, insulin activates the transient receptor potential melastatin type 6 (TRPM6) channel which controls urinary magnesium excretion. Therefore, patients with type 2 diabetes and hypomagnesemia enter a vicious cycle in which hypomagnesemia leads to insulin resistance and insulin resistance reduces serum magnesium concentration.[7,8]

Previous studies have shown that oral magnesium supplementation significantly decreased the fasting and postprandial plasma glucose, glycated hemoglobin, fasting insulin levels and homeostatic model assessment-insulin resistance (HOMA-IR) score in type 2 diabetes patients.[9,17]

We reviewed 1 meta-analysis and 2 RCTs to address the clinical question of the effectiveness of magnesium supplementation on glycemic control in patients with type 2 diabetes.

Asbaghi et al. conducted a recent meta-analysis in 2022 to evaluate the effects of magnesium supplementation on glycemic control in individuals with type 2 diabetes, including 18 studies published between 1989 and 2019. The oral magnesium supplementation in these studies ranged from 36.49 to 500 mg/day with intervention duration varying between 4 and 24 weeks. At a dosage of 500 mg/day, the estimated mean difference in HbA1c was −0.73% (95% CI: −1.25, −0.22) suggesting a modest improvement in HbA1c with strong evidence (p=0.004). Meanwhile, a dose of 360 mg/day resulted in a fasting blood glucose reduction of −7.11 mg/dl (95% CI: −14.03, −0.19), suggesting minimal improvement with weak evidence (p= 0.092). Over a 24-week period, the estimated mean differences in fasting blood glucose and HbA1c were −15.58 mg/dl (95% CI: −24.67, −6.49) and −0.48% (95 % CI: −0.77, −0.19), respectively, suggesting modest improvement in fasting blood glucose (p=0.034) and HbA1c (p=0.001) with strong evidence.[12]

The strength of this present meta-analysis was in performing correct analysis in the homogenous populations, rather than relying on pooled individual data analysis. The limitations are high heterogeneity among the studies and the effects of the confounding variables, including the genetic background and lifestyle factors, were ignored. Although the health benefits of oral magnesium supplementation have been reported, caution is warranted when administering it in certain medical conditions, such as chronic kidney disease and end-stage renal disease. Additionally, it may pose unsafe for patients using certain diuretics and heart medications.[12]

In a randomized controlled trial (RCT) conducted by Albaker et al. in 2022, involving 102 diabetic patients on any anti-diabetic therapy, the addition of magnesium chloride to desalinated bottled water led to significant improvement in the median HbA1c level (8.0 vs 8.2%, p=0.04), median insulin level (7.5 vs 9.9 μIU/mL, p=0.03), and homeostasis model assessment-estimated insulin resistance (HOMA-IR) (2.5 vs 2.9, p=0.002) in the high-dose group after three months compared to baseline. However, no significant changes were observed in fasting blood glucose and C-peptide levels. The intervention (bottled water) contained sodium, potassium, calcium, chloride, and bicarbonate. Each water bottle had a volume of one litre, and the participants consumed one litre of the supplied water per day for three months.[8]

The strength of this study is the inclusion of a relatively large number of patients and the evaluation of different doses of magnesium supplement. However, the limitations include the sole measurement of serum magnesium levels, which may not accurately reflect intracellular magnesium stores, and the relatively low magnesium doses administered.[8]

In an RCT by Drenthen et al. in 2024, involving 14 participants with insulin-treated type 2 diabetes and low serum magnesium level, magnesium supplementation increased both mean serum magnesium level (0.75 ± 0.02 vs 0.70 ± 0.02 mmol/l, p=0.016) and urinary magnesium excretion (magnesium/creatinine ratio, 0.23 ± 0.02 vs 0.15 ± 0.02, p=0.005), compared to placebo. The primary outcome in this study was the mean glucose infusion rate during the final 30 minutes of a hyperinsulinemic-euglycemic clamp, also referred to *M* value. The *M* value of the glucose clamp did not differ between the magnesium and placebo study arms (4.6 ± 0.5 vs 4.4 ± 0.6 mg/kg/min, p=0.108). Variables of glucose control are secondary outcomes, including HbA1C and continuous glucose monitoring. There were no differences in HbA1C and continuous glucose monitoring. Despite a slight increase in magnesium levels, oral magnesium supplementation does not appear to improve insulin sensitivity in individuals with insulin-treated type 2 diabetes and low magnesium levels.[13]

The strengths of the study include its randomized, placebo-controlled, double-blind, crossover design, the use of the glucose clamp technique which is the gold standard for measuring insulin sensitivity, and the inclusion of a study population clearly identified as insulin resistant. However, the limitations include the lack of dietary intake monitoring, participants' magnesium levels remained at the lower end of the normal range, and a relatively small sample size.[13]

Magnesium does not naturally exist in its pure elemental form. Instead, it forms 'salts' when combined with other substances. Magnesium supplements are available in various forms, including magnesium lactate, magnesium citrate, magnesium glycinate, magnesium chloride, magnesium gluconate, and magnesium oxide. The term 'elemental magnesium' refers to the actual amount of magnesium present in each compound.[18] Several studies have found that magnesium in citrate, glycinate, chloride, and gluconate has higher bioavailability than magnesium oxide. Among these, magnesium gluconate has the highest oral bioavailability of magnesium salts and is recommended for magnesium supplementation due its superior absorption and lower likelihood of causing diarrhea. Magnesium chloride salts are highly soluble in water, but they have a bitter and astringent. In contrast, magnesium gluconate has a milder taste and less bitter taste. However, palatability can still vary depending on the dosage and the formulation whether in tablet, powder, or liquid form.[19]

Conclusion

Based on a critical review of the studies, magnesium supplementation may have beneficial effects on glycemic control in patients with type 2 diabetes by modulating carbohydrate metabolism and insulin function. The recommended oral elemental magnesium dose ranges from 36.49 to 500 mg/day for 4 to 24 weeks. However, further research is needed to determine the optimal dosage and the most effective form of magnesium supplementation for improving glycemic control in patients with type 2 diabetes.

Conflict of interest

The authors declared no conflict of interest regarding this article.

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