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Effect of oral magnesium citrate on blood pressure in type 2 diabetes; a single-blind, randomized, placebo-controlled clinical trial study

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ABSTRACT

Introduction: Hypertension accelerates kidney damage in type 2 diabetes mellitus (T2DM), making effective blood pressure control essential for renal protection. Due to magnesium deficiency as a common disorder in this population.

Objectives: This study aimed to assess the effect of oral magnesium supplementation on blood pressure.

Materials and Methods: The study was a randomized, single-blind, placebo-controlled clinical trial conducted at Loqman Hakim Hospital, Tehran, Iran, from 2025 to 2026. Adults with T2DM were enrolled and randomly assigned to receive either 300 mg of oral magnesium citrate daily (n = 30) or a matching placebo (n = 30) for a period of two months. Demographic data and informed written consent were collected at baseline, and blood pressure parameters, including systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), were measured at baseline, one month, and two months using standardized procedures. The outcomes were compared both within and between groups across the follow-up period.

Results: After one month, compared to the baseline, the magnesium group showed decreases of −6.30 mmHg in SBP (4.16%↓), −4.30 mmHg in DBP (4.83%↓), and −3.10 mmHg in MAP (2.83%↓), all with $P<0.05$, while the control group showed smaller, non-significant changes. By two months, reductions in the magnesium group increased to −12.00 mmHg in SBP (7.94%↓), −8.70 mmHg in DBP (9.78%↓), and −7.90 mmHg in MAP (7.21%↓), each with $P<0.001$, whereas the control group again demonstrated non-significant declines. In comparison between one and two months, the magnesium group continued to improve, with additional reductions of −5.70 mmHg in SBP (3.93%↓), −4.40 mmHg in DBP (5.19%↓), and −4.80 mmHg in MAP (4.51%↓), all statistically significant ($P<0.05$), while the control group showed minimal, non-significant fluctuations across all parameters.

Conclusion: The results indicate that magnesium citrate is an effective adjunct strategy for improving blood pressure control in patients with T2DM.

Trial Registration: The trial protocol was approved by the Iranian Registry of Clinical Trials with code (identifier: IRCT20230522058258N2), and received ethical approval from Shahid Beheshti University of Medical Sciences (IR.SBMU.MSP.REC.1404.602).

Implication for health policy/practice/research/medical education:
In this clinical trial study, we found that magnesium supplementation led to meaningful and sustained reductions in systolic, diastolic, and mean arterial blood pressure among adults with type 2 diabetes, with improvements evident after one month and further strengthened by two months, whereas the control group showed only minimal, non-significant fluctuations across these blood pressure parameters.

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Clinical Trial

Introduction

Type 2 diabetes mellitus (T2DM) constitutes a major global public health crisis, with its prevalence projected to reach 783 million cases worldwide by 2045, driven by hyperglycemia and associated microvascular and macrovascular complications such as cardiovascular disease, hypertension, and diabetic kidney disease (1-3). According to recent global estimates, the prevalence of T2DM continues to rise, and a significant proportion of these individuals will also develop hypertension (4). Effective blood pressure control in patients with T2DM is thus a critical public health priority, as even modest reductions in blood pressure can yield substantial reductions in cardiovascular events and mortality (5). Despite the availability of antihypertensive pharmacotherapy, BP control remains suboptimal in many patients with T2DM, highlighting the need for adjunctive strategies, including dietary and micronutrient interventions, to improve outcomes (6).

Magnesium, the fourth most abundant mineral in the human body, plays a pivotal role in numerous physiological processes relevant to cardiovascular and metabolic health (7,8). It serves as a cofactor for over 300 enzymatic reactions, including those involved in energy metabolism, nucleic acid synthesis, and regulation of vascular tone (9,10). Mechanistically, magnesium modulates vascular smooth muscle contraction by acting as a natural calcium antagonist, promotes endothelial-dependent vasodilation through enhanced synthesis of nitric oxide and prostacyclin, and attenuates oxidative stress and inflammation within the vascular wall (11). In addition, magnesium influences insulin secretion and action, and its deficiency has been implicated in the pathogenesis of insulin resistance, a hallmark of T2DM (7). Meta-analysis studies have consistently reported an inverse association between dietary magnesium intake and blood pressure levels in both general and diabetic populations (12). Notably, hypomagnesemia is frequently observed in individuals with T2DM, with prevalence varying depending on the population and diagnostic criteria used (13). This deficiency may exacerbate insulin resistance, endothelial dysfunction, and vascular calcification, further contributing to the development and progression of hypertension in diabetic patients (14,15). These findings have prompted considerable interest in the potential antihypertensive effects of magnesium supplementation, particularly as a low-cost, well-tolerated adjunctive therapy in populations at high cardiovascular risk.

Objectives

This study was conducted to evaluate the effect of oral magnesium supplementation on blood pressure in patients with type 2 diabetes, with the aim of improving hypertension management and supporting non-pharmacologic strategies for kidney disease risk reduction.

Materials and Methods

Study design and participants

This study employed a randomized, single-blind, placebo-controlled clinical trial design and adhered to the SPIRIT (Standard Protocol Items: Recommendations for Interventional Trials) (16) guidelines for preparing the trial protocol, as well as the CONSORT (Consolidated Standards of Reporting Trials) (17) guidelines for reporting trial outcomes. The study was conducted at Loqman Hakim Hospital in Tehran, Iran, between 2025 and 2026. Adults with a confirmed diagnosis of T2DM were screened for eligibility based on predefined inclusion and exclusion criteria. After initial assessment, eligible participants who provided written informed consent were randomly assigned to either the magnesium supplementation group or the placebo control group. A total of 60 participants (30 in each group) completed the study and were included in the final analysis.

Inclusion and exclusion criteria

Participants were eligible for enrollment if they met the following criteria: a confirmed diagnosis of T2DM based on the American Diabetes Association (ADA) diagnostic standards (18); age between 30 and 70 years; a documented diagnosis of controlled or uncontrolled hypertension undertreatment exclusively with valsartan and amlodipine; and provision of written informed consent before participation. Individuals were required to have no evidence of renal disease or heart failure and no history of magnesium supplementation or use of medications known to influence serum magnesium levels within the preceding three months. Additional criteria included the absence of severe hypertension at baseline requiring urgent medical intervention or hospitalization, no current pregnancy, and no gastrointestinal disorders that could interfere with magnesium absorption. Patients were excluded if they were receiving antihypertensive medications other than valsartan and amlodipine during the study period, declined to continue participation, or developed any condition that could interfere with adherence or study procedures. Individuals who had taken magnesium supplements or medications known to alter serum magnesium levels during the study, as well as those who failed to complete the study protocol, were also excluded.

Sample size

Using a 95% confidence level, 80% statistical power, an effect size of 0.8 (the minimum effect size based on Cohen's equation), and the G*Power software (19), the required sample size was calculated to be 26 participants per group. To account for potential attrition, 30 participants were included in each group.

Randomization/allocation

Participants were randomly assigned to either the

magnesium supplementation group or the placebo control group. Randomization was performed using a computer-generated simple random sequence to ensure an unbiased allocation process. Allocation was conducted by an independent researcher not involved in data collection or outcome assessment to maintain allocation concealment. Participants were assigned to their respective groups in a single-blind manner, meaning that they were unaware of their group assignment, while the research team responsible for administering the intervention had access to allocation information.

Blinding

This study employed a single-blind design in which participants were unaware of their assigned treatment group throughout the trial. Both the magnesium supplement and the placebo were prepared in identical capsules with similar appearance, packaging, and administration instructions to prevent participants from distinguishing between interventions. The research personnel responsible for distributing the study medications were aware of group assignments; however, all individuals involved in outcome measurement and data collection remained blinded to treatment allocation to minimize assessment bias. Blinding was maintained until completion of data collection and final analysis.

Intervention

Participants assigned to the intervention group received 300 mg of oral magnesium citrate (Biomagnelyte Magnesium, Sagepad Darou, Iran) once daily for a period of two months. The control group received a matching placebo administered on the same schedule. Both the magnesium supplement and placebo were provided in identical capsules to maintain blinding. All participants were instructed to take the assigned capsule at a consistent time each day and to report any missed doses during follow-up visits. Blood pressure parameters, including systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), were measured at three time points: before the initiation of the intervention (baseline), one month, and two months after the start of treatment. The measured blood pressure parameters were compared between the two groups as well as within each group throughout the study period.

Data collection

Data collection was performed at baseline, one month, and two months after randomization using standardized clinical procedures. At enrollment, demographic information, including age, sex, and body mass index (BMI), was obtained through patients' interviews. Height and weight were measured using calibrated equipment, and BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2), following internationally accepted criteria (20, 21). Written informed consent was obtained from all participants before data collection. Blood pressure measurements (SBP, DBP, and MAP) were obtained at each visit by trained personnel using a calibrated digital sphygmomanometer after a minimum five-minute seated rest. Two consecutive readings were taken on the same arm, and their average was recorded to ensure accuracy. All collected data were documented in structured case report forms and subsequently entered into SPSS for statistical analysis.

Outcomes measurement

The outcome of this study is a comparison of changes in blood pressure parameters, including SBP, DBP, and MAP, from baseline to one and two months after the intervention within the intervention and control groups, as well as a comparison of these parameters between these two groups at these time point.

Statistical analysis

All statistical procedures were conducted using SPSS software, version 27 (IBM Corp., USA). Before analysis, the distribution of continuous variables was examined using the Shapiro–Wilk test to verify normality. The Levene test was applied to assess the homogeneity of variances between the study groups. Because parametric and non-parametric analyses produced comparable P values, parametric methods were selected to take advantage of their greater statistical efficiency. Baseline demographic and clinical characteristics were compared using the independent samples t-test for continuous variables and the chi-square test for categorical variables. Changes in blood pressure within each group over time were evaluated using the paired samples t-test, while between-group comparisons at each measurement point were performed using the independent samples T-test. All analyses were two-tailed, and a P value < 0.05 was considered statistically significant.

Results

The study assessed 76 individuals for eligibility, after which 11 were excluded, with 7 not meeting the inclusion criteria and 4 declining to participate. A total of 65 participants were then randomized, with 33 allocated to the magnesium group and 32 to the control group; all participants in each group received their assigned intervention. During follow-up, 3 participants in the magnesium group were lost due to lack of adherence to the treatment protocol, while 2 participants in the control group were lost because they were not referred for blood pressure measurement. Ultimately, 30 participants from each group were included in the final analysis (Figure 1). The study enrolled a total of 60 patients with T2DM, allocating 30 participants to the magnesium intervention group and 30 to the control group. The distribution of gender was similar between the magnesium group and the control group, with no statistically significant difference

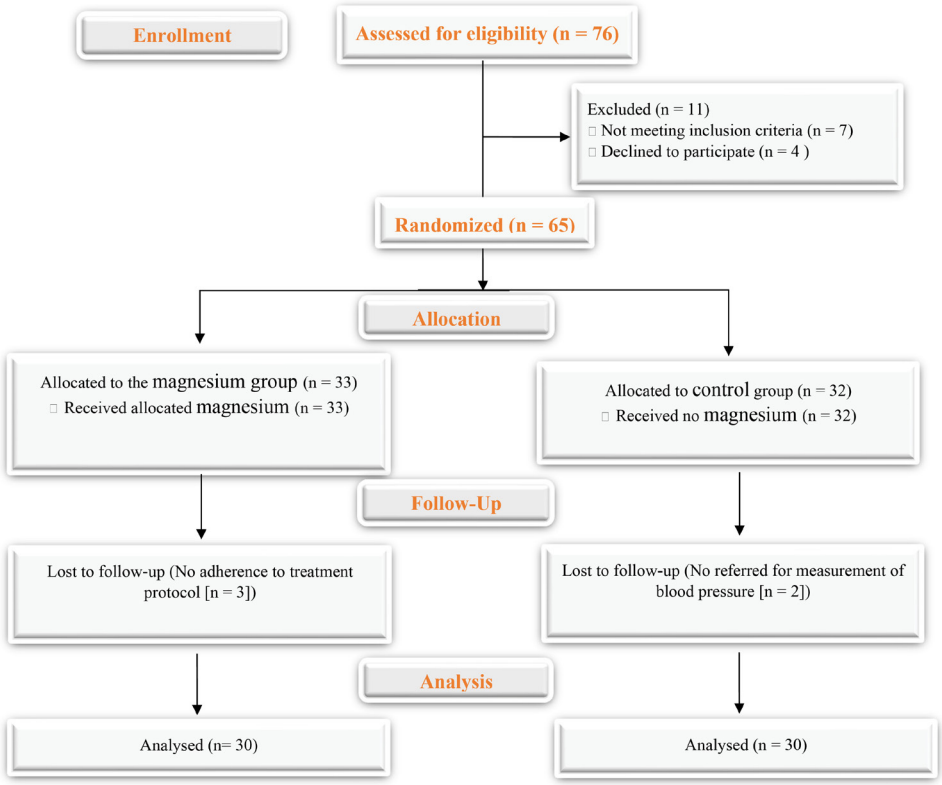

Figure 1. CONSORT flow diagram of the study.

observed. Likewise, the quantitative variables showed no meaningful differences between groups. Participants in both groups had comparable average ages and body mass index values, and statistical testing indicated that none of these baseline characteristics differed significantly between the treatment and control groups (Table 1).

The results indicated that at the time of baseline, SBP, DBP, and MAP were comparable between the magnesium and control groups, with no statistically significant differences. One month after the intervention, SBP and MAP demonstrated a significant difference between groups, whereas DBP remained similar. By the second month, SBP and MAP no longer differed significantly, while DBP showed a clear between-group difference (Table 2).

Across the three measurement time points, both groups demonstrated gradual changes in SBP, DBP, and MAP, with the magnesium group generally showing a more consistent downward trajectory. At baseline, blood pressure values were broadly comparable between groups. After one month of intervention, the magnesium group exhibited noticeable reductions in SBP, DBP, and MAP, whereas the control group showed smaller changes, resulting in a divergence between groups at this stage. By the second month, the magnesium group continued to decline while the control group increased slightly. Overall, the pattern suggests that magnesium supplementation produced more pronounced improvements in blood pressure parameters during the follow-up duration (Figure 2).

Within-group comparisons from baseline to one month after the intervention showed that participants receiving magnesium experienced meaningful reductions in SBP, DBP, and MAP, with all changes reaching statistical significance. In contrast, the control group demonstrated

Table 1. Comparison of baseline demographic characteristics between the treatment groups of magnesium consumption and the control group

Demographic characteristics		Treatment group		*P* value*
		Mg (n = 30)	Control (n = 30)	
		N (%)	N (%)	
Gender	Male	13 (43.7)	12 (40)	0.793
	Female	17 (56.3)	18 (60)	
Quantitative variables		**Mean ± SD**	**Mean ± SD**	***P* value****
Age (year)		61.71 ± 9.14	59.40 ± 5.49	0.369
BMI (kg/m^2)		27.81 ± 3.84	26.57 ± 4.17	0.236

BMI: Body mass index, SD: Standard deviation, *Chi-square, **Independent T-test.

Table 2. Comparison of blood pressure parameters among the treatment groups during the study period

BP parameters	Treatment group				*P* value*
	Mg (N = 30)		Control (N = 30)		
	Mean	SD	Mean	SD	
Baseline					
SBP (mmHg)	151.10	10.51	145.50	13.65	0.080
DBP (mmHg)	88.93	6.51	87.83	5.79	0.492
MAP (mmHg)	109.51	6.32	106.78	6.70	0.111
1-month after intervention					
SBP (mmHg)	144.80	6.34	140.67	6.07	0.012
DBP (mmHg)	84.63	4.80	84.87	6.01	0.869
MAP (mmHg)	106.41	3.48	104.30	4.01	0.033
2-month after intervention					
SBP (mmHg)	139.10	7.39	141.33	7.30	0.244
DBP (mmHg)	80.23	5.45	85.33	5.68	<0.001
MAP (mmHg)	101.61	4.56	103.45	4.85	0.137

BP: Blood pressure, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, SD: Standard deviation, *Independent T-test.

smaller declines across the same parameters, none of which achieved statistical significance (Table 3).

Over the two months following the intervention, participants in the magnesium group demonstrated substantial and statistically significant reductions in all 3 parameters of blood pressure, including SBP, DBP, and MAP, when compared with their baseline values, while the control group exhibited modest declines across these parameters, with no statistical significance (Table 4).

When comparing blood pressure values between one and two months after the intervention, the magnesium group demonstrated continued and statistically significant reductions in SBP, DBP, and MAP, with the rate of change reflecting further improvement beyond the first month. In contrast, the control group showed minimal fluctuations across all blood pressure parameters, none of which reached statistical significance (Table 5).

Discussion

The findings of this clinical trial demonstrated that magnesium supplementation serves as a beneficial adjunctive intervention for enhancing blood pressure control in individuals with T2DM, producing meaningful reductions across blood pressure parameters, including SBP, DBP, and MAP. These results align with prior evidence from randomized trials and meta-analyses reporting that magnesium supplementation can modestly but significantly reduce blood pressure in adults, including those with metabolic disorders. For example, a meta-analysis of randomized controlled trials by Zhang et al found that magnesium supplementation was associated with reductions in both SBP and DBP across diverse populations, supporting the plausibility of the present findings (22). Similarly, a pooled analysis of 24 randomized trials in T2DM reported improvements in hypertension-related

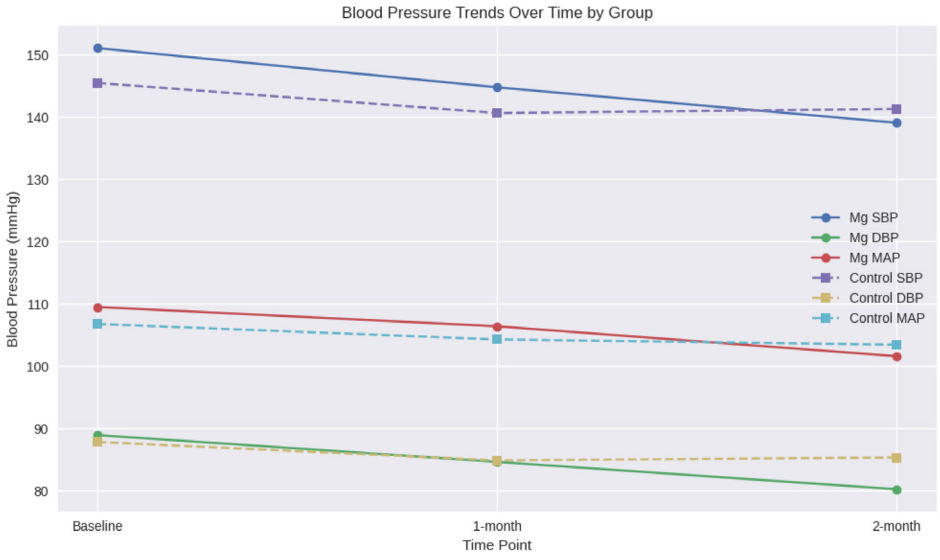

Figure 2. Trends in blood pressure parameters over the study period in the magnesium and control groups. Mg: Magnesium, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure.

Table 3. Comparative analysis of blood pressure parameters within treatment groups, comparing one month post-intervention values with those at baseline

BP parameters	Mean difference				Changing rate (%)	*P* value*
	Mean	Std. error	95% CI			
			Lower	Upper		
Mg (N = 30)						
SBP (mmHg)	- 6.30	1.95	- 10.29	- 2.30	4.16 ↓	0.003
DBP (mmHg)	- 4.30	1.48	- 7.33	- 1.26	4.83 ↓	0.007
MAP (mmHg)	- 3.10	1.11	- 5.37	- 0.80	2.83 ↓	0.010
Control (N = 30)						
SBP (mmHg)	- 4.83	2.91	- 10.80	1.13	3.31 ↓	0.108
DBP (mmHg)	- 2.96	1.53	- 6.10	0.17	3.37 ↓	0.063
MAP (mmHg)	- 2.48	1.36	- 5.28	0.30	2.32 ↓	0.079

Mg: Magnesium, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, CI: Confidence interval, *Paired samples T-test.

Table 4. Comparative analysis of blood pressure parameters within treatment groups, comparing two-month post-intervention values with those at baseline

BP parameters	Mean difference				Changing rate (%)	*P* value*
	Mean	Std. error	95% CI			
			Lower	Upper		
Mg (N = 30)						
SBP (mmHg)	- 12.00	2.18	- 16.46	- 7.53	7.94 ↓	< 0.001
DBP (mmHg)	- 8.70	1.35	- 11.47	- 5.92	9.78 ↓	< 0.001
MAP (mmHg)	- 7.90	1.59	- 11.15	- 4.62	7.21 ↓	< 0.001
Control (N = 30)						
SBP (mmHg)	- 4.17	2.40	- 9.08	0.75	2.86 ↓	0.094
DBP (mmHg)	- 2.50	1.51	- 5.59	0.59	2.84 ↓	0.110
MAP (mmHg)	- 3.33	1.77	- 6.96	0.30	3.11 ↓	0.071

Mg: Magnesium, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, CI: Confidence interval, *Paired samples T-test.

Table 5. Comparative analysis of blood pressure parameters within treatment groups, comparing two-month post-intervention values at one month post-intervention

BP parameters	Mean difference				Changing rate (%)	*P* value*
	Mean	Std. error	95% CI			
			Lower	Upper		
Mg (N = 30)						
SBP (mmHg)	- 5.70	1.79	- 9.37	- 2.02	3.93 ↓	0.004
DBP (mmHg)	- 4.40	1.48	- 7.43	- 1.36	5.19 ↓	0.006
MAP (mmHg)	- 4.80	1.02	- 6.89	- 2.70	4.51 ↓	<0.001
Control (N = 30)						
SBP (mmHg)	0.66	1.60	- 2.61	3.94	0.46 ↑	0.681
DBP (mmHg)	0.46	1.56	- 2.73	3.67	0.54 ↑	0.768
MAP (mmHg)	- 0.85	1.28	- 3.47	1.78	0.81 ↓	0.516

Mg: Magnesium, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, CI: Confidence interval, *Paired samples T-test.

outcomes following magnesium supplementation, further corroborating the antihypertensive potential of this intervention (23). Maqrashi et al's systematic review and meta-analysis, by pooling data across available studies, found that magnesium supplementation was associated with a significant reduction in DBP in T2DM patients, suggesting a modest but meaningful hemodynamic benefit in this population (24). A relevant systematic review and meta-analysis by Amer et al provides additional context for interpreting the antihypertensive effects of magnesium. In their analysis of 24 randomized studies, magnesium supplementation was associated with a significant reduction in DBP (MD: –1.64 mmHg, 95% CI: –3.19 to –0.09; $P = 0.04$), while showing no significant effect on SBP ($P = 0.16$) or pulse rate ($P = 0.81$). These findings suggest that magnesium's primary hemodynamic influence may be exerted through mechanisms that preferentially lower peripheral vascular resistance rather than directly affecting systolic load. When considered alongside the current trial's observation of reductions across SBP, DBP, and MAP, the results of Amer et al highlight both the consistency of magnesium's diastolic-lowering effect and the possibility that certain populations, such as individuals with type 2 diabetes, may experience broader blood pressure

benefits (6). A systematic review and meta-analysis by Verma et al offers further evidence supporting the antihypertensive potential of magnesium in individuals with type 2 diabetes. Although the primary focus of their analysis was a broad range of cardiometabolic outcomes, magnesium supplementation was also associated with a significant reduction in SBP (weighted mean difference [WMD]: –3.06 mmHg, 95% CI: –5.51 to –0.60; $P = 0.015$). This finding reinforces the possibility that magnesium contributes to improved vascular function in diabetic populations, potentially through mechanisms linked to enhanced glycemic control, lipid modulation, and reduced vascular resistance. Notably, Verma et al reported that the antihypertensive effect was more pronounced in individuals with hypomagnesemia, suggesting that baseline magnesium status may influence treatment responsiveness (25). A systematic review and meta-analysis by Han et al provides additional population-level evidence supporting the role of magnesium in blood pressure regulation. Across ten prospective cohort studies involving more than 180,000 participants, the authors identified a clear inverse association between dietary magnesium intake and the risk of developing hypertension, with individuals in the highest intake category experiencing an 8% lower risk compared with those in the lowest (RR = 0.92; 95% CI 0.86–0.98). Moreover, each 100 mg/day increase in magnesium intake was associated with a 5% reduction in hypertension risk (RR = 0.95; 95% CI 0.90–1.00), suggesting a dose-response relationship that strengthens the biological plausibility of magnesium's antihypertensive effects (26). Although the magnitude of effect varies across studies, likely due to differences in baseline magnesium status, dosage, and formulation, the direction of effect observed here is consistent with the broader literature. A notable strength of the present study is its randomized, placebo-controlled design, which enhances internal validity. These findings support the idea that magnesium may serve as a supportive therapy alongside standard diabetes and hypertension management.

Overall, these findings indicate that magnesium supplementation may serve as a beneficial adjunctive strategy for improving blood pressure control in this high-risk population. The results are consistent with existing evidence suggesting that magnesium contributes to improved vascular function and blood pressure regulation. While the study design strengthens confidence in the observed effects, further research with larger sample sizes and longer follow-up periods is warranted to confirm the durability of the benefit and to refine clinical recommendations. Magnesium supplementation represents a practical, low-cost intervention that may complement established therapeutic approaches for hypertension management in T2DM.

Conclusion

The results indicated that magnesium citrate supplementation resulted in clinically meaningful reductions in systolic, diastolic, and mean arterial blood pressure among adults with T2DM, with improvements emerging within the first month and continuing through the second month of treatment, while patients who did not receive magnesium showed only minimal, non-significant variations in blood pressure over the same period. Collectively, these findings suggest that magnesium supplementation may serve as an effective adjunctive strategy for improving blood pressure control in this population, particularly in those requiring additional non-pharmacologic support for hypertension management.

Limitations of the study

The single-blind design, in which investigators administering the intervention were not blinded, may introduce a degree of performance bias. The short follow-up duration of two months restricts conclusions about the long-term effects of magnesium supplementation on blood pressure control. Additionally, adherence to the intervention was based on participant self-report, which may be subject to reporting bias. Finally, potential confounding factors such as dietary magnesium intake and physical activity were not fully controlled, which could influence the observed outcomes.

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Authors' contribution

Conceptualization: Arezoo Ranjbar Arani, Zahra Abbasi, and Yekta Ghasemi
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Investigation: Bahador Oshidari, Sahar Kavand, and Zahra Sahraei.
Methodology: Masood Zangi and Mohsen Soori.
Project management: Mahdi Amirdosara and Sahar Kavand.
Resources: All authors.
Supervision: All authors.
Validation: Arezoo Ranjbar Arani and Mohsen Soori.
Writing–original draft: All authors.
Writing–review and editing: All authors.

Conflicts of interest

The authors declare no conflict of interest.

Data availability statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of generative artificial intelligence (AI) and AI-assisted technologies in the writing process

While preparing this work, the authors utilized AI (Grammarly and Copilot) to refine grammar points and language style. Subsequently, they thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

The research was conducted in accordance with the principles the Declaration of Helsinki.. This study was conducted at Loghman Hakim Hospital and is derived from the thesis of Yekta Ghasemi (Thesis#43016488), with the Ethical code (IR.SBMU.MSP.REC.1404.602; https://ethics.research.ac.ir/form/dspuhut7e4jjtnyw.pdf), approved by the Shahid Beheshti University of Medical Sciences, Tehran, Iran. The study protocol was also registered as a clinical trial at the Iranian Registry of Clinical Trials (identifier: IRCT20230522058258N2; https://irct.behdasht.gov.ir/trial/88047). Besides, written informed consent was taken from all participants before any intervention. Additionally, the authors have ultimately observed ethical issues (including plagiarism, data fabrication, and double publication).

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