RESEARCH PAPER

Effect of magnesium supplementation in Hypomagnesemia on wound healing in diabetic foot ulcer

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ABSTRACT

Background: Hypomagnesemia is linked to the onset of neuropathy and altered platelet function, and increase the risk of diabetic foot ulcer (DFU). The present study aimed to assess the effect of magnesium supplementation on wound healing in individuals with diabetic foot ulcers and hypomagnesemia.

Materials and Methods: This intervention study included ninety patients with DFU, allocated to two groups: intervention and control. Serum magnesium levels and ulcer dimensions such as length, width and wound characteristics like discharge, sloping edge, and surrounding skin condition were assessed among both groups. Statistical analysis was performed using SPSS software to compare intervention and control groups.

Results: Both groups showed improvement in ulcer healing parameters; however, the intervention group showed a bigger decrease in ulcer length (3.91±0.51 to 2.61±0.51 cm) and width (2.77±0.43 to 1.68±0.44 cm) than the control group (length: 3.86±0.52 to 3.12±0.60 cm; width: 2.67±0.42 to 1.81±0.42 cm). The intervention group had a higher rate of discharge resolution (91.1% vs. 86.7%) and a higher percentage of people who achieved normal surrounding skin (93.3% vs. 88.9%) at 12 weeks. Both groups saw a big improvement in the formation of sloping edges (88.9%).

Conclusion: At the 12-week follow-up, patients with diabetic foot ulcers (DFUs) who had low magnesium levels showed greater improvement in wound-healing metrics than those without hypomagnesemia. This suggests that correcting magnesium deficiency could accelerate ulcer healing.

Keywords: DFU, ulcer healing, magnesium, hypomagnesemia, and prospective study

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Introduction

Diabetes mellitus (DM) is a group of metabolic illnesses characterized by

elevated blood glucose levels due to insufficient insulin secretion by β-cells or insulin receptor insensitivity.[1] Recent findings indicate that approximately one-third of the 500 million individuals living with diabetes globally may face the risk of developing a diabetic foot ulcer at some point in their lives.[2] A diabetic foot ulcer (DFU) is characterized by a disruption of the epidermal layer and at least a portion of the dermal layer in individuals diagnosed with diabetes mellitus. Lesions that are more superficial or closed, which do not extend into the dermis (such as calluses, blisters, warmth, or erythema), are classified as preulcerative and exhibit a significant risk of advancing to ulceration.[3] It is estimated that around 20% of individuals who experience a diabetic foot ulcer (DFU) will necessitate lower-extremity amputation, which may be classified as a minor (below the ankle), major (above the ankle), or a combination of both.[4] Moreover, 10% of these patients could die within one year following their initial DFU diagnosis.[5]

Research indicates that a significant proportion of amputations associated with diabetes, approximately 85%, could potentially be avoided through early detection and proper management of wounds.[6] It is recognized that specific vitamins and minerals are crucial to wound healing, and deficiencies in these essential nutrients can impair healing outcomes.[7]

Magnesium is the fourth most abundant mineral in the human body. This substance is acknowledged as a cofactor in over 300 enzymatic reactions, playing a vital role in the metabolism of adenosine triphosphate (ATP). In addition to its role in DNA and RNA synthesis, protein synthesis, and reproduction, magnesium regulates several physiological processes, including heart rate, blood pressure, insulin sensitivity, neuromuscular conduction, and cardiac excitability.[8] Low magnesium levels are linked to Alzheimer's disease, insulin resistance, type-2 diabetes, high blood pressure, cardiovascular conditions (e.g., stroke), headaches associated with migraines, and ADHD.[8] Hypomagnesemia can lead to the onset of neuropathy and irregular platelet function, both of which contribute to the increased risk of foot ulcer progression.[9] Magnesium supplementation shows potential benefits for the healing of diabetic foot ulcers (DFUs). So, this study aimed to study whether magnesium supplementation improves wound healing in diabetic foot ulcer

Objectives

- To study magnesium supplementation improving wound healing in patients with diabetic foot ulcer and Hypomagnesemia
- To compare the wound healing assessment based on wound characteristics like size, edge, margin, floor, discharge, and surrounding skin

Review of literature

Razzaghi et al.[10] discovered that following the 12-week treatment, magnesium supplementation, in comparison to the placebo, led to a notable increase in serum magnesium levels (+0.3 ± 0.3 vs. -0.1 ± 0.2 mg/dL, $P < 0.001$) and significant reductions in ulcer length (-1.8 ± 2.0 vs. -0.9 ± 1.1 cm, $P = 0.01$), width (-1.6 ± 2.0 vs. -0.8 ± 0.9 cm, $P = 0.02$), and depth (-0.8 ± 0.8 vs. -0.3 ± 0.5 cm, $P = 0.003$). Moreover, notable decreases in fasting plasma glucose (-45.4 ± 82.6 vs. -10.6 ± 53.7 mg/dL, $P = 0.04$), serum insulin levels (-2.4 ± 5.6 vs. +1.5 ± 9.6 μIU/mL, $P = 0.04$),

and HbA1c (-0.7 ± 1.5 vs. -0.1 ± 0.4%, P = 0.03), along with a significant increase in the quantitative insulin sensitivity check index (+0.01 ± 0.01 vs. -0.004 ± 0.02, P = 0.01), were observed following magnesium supplementation compared to the placebo. Furthermore, in comparison to the placebo, magnesium supplementation led to a notable reduction in serum high-sensitivity C-reactive protein (hs-CRP) levels and a considerable elevation in plasma total antioxidant capacity (TAC) concentrations (+6.4 ± 65.2 vs. -129.9 ± 208.3 mmol/L). Magnesium supplementation for 12 weeks in participants with diabetic foot ulcers shown advantageous effects on ulcer size, glucose metabolism, serum high-sensitivity C-reactive protein, and plasma total antioxidant capacity levels. According to the findings of Afzali et al, administration of magnesium and vitamin E supplements resulted in a decrease in ulcer length (β [difference in the mean of outcomes measurements between treatment groups] −0.56 cm), width (β −0.35 cm), and depth (β −0.18 cm; 95% confidence interval, −0.33, −0.02; p = 0.02). It is also worth mentioning that the co-supplementation was linked to a significant reduction in fasting plasma glucose (β -13.41 mg/dL; 95% CI, -20.96, -5.86; p = 0.001), insulin (β -1.45 μIU/ml; 95% CI, -2.37, -0.52; p = 0.003), insulin resistance (β -0.60; 95% CI, -0.99, -0.20; p = 0.003) and HbA1c (β -0.32%; 95% CI, -0.48, -0.16; p < 0.003) and with a significant increase in insulin sensitivity (β 0.007; 95% CI, 0.003, 0.01; p < 0.001) compared to the placebo.

Material and Methods

This prospective comparative study was conducted over 12 months in the Department of General Surgery at Trichy SRM Medical College Hospital and Research Center, Tamil Nadu, to assess the impact of oral magnesium supplementation on wound-healing parameters in patients with diabetic foot ulcers. A total of 90 adult patients aged 18 to 65 years with chronic non-healing diabetic foot ulcers (DFUs) were enrolled in the study. The participants were assessed for serum magnesium levels, and patients with hypomagnesemia were enrolled in the intervention group. The patients with normal magnesium levels were enrolled in the control group. There were two groups of participants. 45 patients in the intervention group took magnesium supplements by mouth. In the control group, on the other hand, 45 patients received regular diabetic foot care without additional magnesium. Participants were enrolled sequentially through a process called consecutive sampling that adhered to strict inclusion and exclusion criteria after obtaining informed written consent

. Individuals who administered medications that are known to influence serum magnesium concentrations, such as aminoglycosides, amphotericin B, digoxin, and loop diuretics, were not included in the study. To avoid confounding factors that affect magnesium metabolism and wound healing, patients with renal failure, acute or chronic diarrhea, malabsorption syndromes, cancer, sepsis, pregnant or lactating women, and those already taking magnesium or multivitamin-mineral supplements, antioxidant supplements, or anti-inflammatory agents were also excluded. The IEC approval was sought in the teaching Hospital for the conduct of the study. Baseline demographic and clinical information, such as age, gender, duration of diabetes, and glycemic status, was documented after participants provided

informed written consent. Serum magnesium levels were assessed before supplementation.

A comprehensive evaluation of the wound was conducted at presentation, using established wound-healing parameters. This assessment included examining the type and characteristics of discharge, the quality of granulation tissue, the features of the wound edges (whether sloping or non-sloping), the condition of the surrounding skin, the dimensions of the wound, and any exposure of bone. The dimensions of the wound were assessed using established methods, measuring length and width in centimeters with a sterile measuring tool and reporting the area in square centimeters.

To reduce inter-observer variability, wound assessment during the study period was conducted by the same surgical team utilizing a semi-structured proforma. Individuals in the intervention group received oral magnesium oxide at 400 mg once daily, alongside standard diabetic foot management protocols. These protocols included strict glycemic management, wound debridement, when necessary, the use of sterile dressings, infection-control strategies based on clinical and microbiological evaluations, and suitable offloading methods. The control group received the same standard care, except for magnesium supplementation.

At each follow-up visit (at 4, 8, and 12 weeks), wound parameters were systematically reassessed for all patients. The wound size was re-evaluated using the same standardized technique, and serum magnesium levels were measured to assess the biochemical response. Adherence to medication and wound care protocols was assessed at each appointment. In individuals from the supplementation cohort with consistently low serum magnesium levels and inadequate wound-healing progress, the magnesium dose was increased under clinical observation following a thorough reassessment. All observations and laboratory values were meticulously recorded in a structured data-collection format to ensure consistency and completeness. The main outcome measure focused on the enhancement of wound-healing parameters over the 12 weeks, evaluated by reductions in wound size and discharge, improvements in healthy granulation tissue formation, development of sloping wound edges, improvement in surrounding skin condition, and the absence of bone exposure. The secondary outcome assessed the change in serum magnesium levels from baseline to 12 weeks and examined its relationship with wound-healing progression.

The data were systematically entered and analyzed utilizing IBM SPSS Statistics version 26.0. Continuous variables were evaluated for normality using appropriate statistical tests and reported as the mean and standard deviation for normally distributed variables. The independent-samples t-test was used to compare the mean values of the two groups. In contrast, the paired t-test was used to assess within-group changes from baseline to follow-up. Frequencies and percentages were used to express categorical variables, with comparisons conducted through the Chi-square test or Fisher's exact test as deemed suitable. A two-sided p-value below 0.05 was deemed statistically significant.

Results

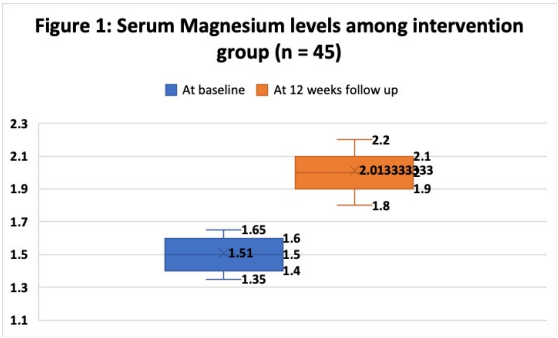
This study included 45 patients with diabetic foot ulcer and hypomagnesemia who received magnesium supplementation,

and 45 patients with diabetic foot ulcer who received standard care. The average age of patients in the intervention group was 50.33 (9.18) years, and in the control group, 49.36 (10.34) years. The average duration of diabetes mellitus was 12.82 ± 4.6 years in the DFU with hypomagnesemia group and 12.78 ± 5.13 years in the DFU group. There were 29 men (64.4%) and 16 women (35.6%) in the intervention group. In the control group, 30 patients (66.7%) were male, and 15 patients (33.3%) were female. The average HbA1c levels were 10.16 ± 1.38% in the intervention group and 8.43 ± 1.35% in the control group.

Table 1: Descriptive data of participants

SNo	Variables		Intervention group (n = 45)	Control group (n = 45)
1	Mean age		50.33 ± 9.18	49.36 ± 10.34
2	Gender	Male	29 (64.4%)	30 (66.7 %)
		Female	16 (35.6%)	15 (33.3 %)
3	Mean duration of DM		12.82 ± 4.6	12.78 ± 5.13
4	Mean HbA1C		10.16 ± 1.38	8.43 ± 1.35

Figure 1 shows serum magnesium levels in the intervention group. The intervention group had a much lower baseline serum magnesium level (1.51 ± 0.10 mg/dL) than the control group (2.13 ± 0.13 mg/dL). The mean serum magnesium level in the intervention group rose to 2.01 ± 0.12 mg/dL at 12 weeks, whereas it remained approximately the same in the DFU group (2.14 ± 0.11 mg/dL).



Ulcer characteristics

At baseline, the average ulcer length was similar in both groups: 3.91 ± 0.51 cm in the intervention group and 3.86 ± 0.52 cm in the control group. The average width of the ulcers was also similar, with the intervention group measuring 2.77 ± 0.43 cm and the control group measuring 2.67 ± 0.42 cm. This means that the ulcers were the same size at the start. After 12 weeks, the intervention group had a greater decrease in ulcer size. In this group, the average ulcer length decreased to 2.61 ± 0.51 cm, whereas in the control group it remained at 3.12 ± 0.60 cm. The average width of the ulcers also went down in the intervention group, from 1.81 ± 0.42 cm to 1.68 ± 0.44 cm.

Table 2: Variation in ulcer size at baseline and follow-up

SNo	Ulcer size		Intervention group (n = 45)	Control group (n = 45)
1	Length	At baseline	3.91 ± 0.51	3.86 ± 0.52

		At follow-up	2.61 ± 0.51	3.12±0.60
2	Width	At baseline	2.77 ± 0.43	2.67 ± 0.42
		At follow-up	1.81±0.42	1.68±0.44

At baseline, a higher percentage of patients in both groups exhibited ulcer discharge: 41 patients (91.1%) in the intervention group and 37 patients (82.2%) in the control group. A lower percentage exhibited no discharge (8.9% vs 17.8%). After 12 weeks, there was a significant reduction in ulcer discharge in both groups. In the intervention group, only four patients (8.9%) had ulcer discharge, while 41 patients (91.1%) did not. In the control group, 6 patients (13.3%) had ulcer discharge, while 39 patients (86.7%) did not.

At baseline, 9 patients (20.0%) in the intervention group had a sloping edge, compared with only 3 (6.7%) in the control group. At first, most of the patients in both groups did not have a sloping edge (80.0% vs. 93.3%, respectively). There was a big rise in the number of sloping edges in both groups after 12 weeks. In the intervention group, 40 patients (88.9%) exhibited a sloping edge, whereas only 5 patients (11.1%) did not. In the control group, 37 patients (82.2%) exhibited a sloping edge, while 8 patients (17.7%) did not.

Table 3: Ulcer characteristics at baseline and follow-up

SNo	Ulcer		Intervention group (n = 45)	Control group (n = 45)
1	Presence of discharge	At baseline	41 (91.1%)	37 (82.2%)
		At follow-up	4 (8.9%)	6 (13.3%)
2	Presence of sloping edge	At baseline	9 (20%)	3 (6.7%)
		At follow-up	40 (88.9%)	37 (82.2%)
3	Presence of normal surrounding skin	At baseline	33 (73.3%)	34 (75.6%)
		At follow-up	42 (93.3%)	40 (88.9%)

At the start of the study, 33 patients (73.3%) in the intervention group and 34 patients (75.6%) in the control group had normal skin. Twelve patients (26.7%) had abnormal skin around them, and eleven patients (24.4%) had abnormal skin around them, which shows that the two groups had similar baseline characteristics. After 12 weeks, both groups saw an improvement in the condition of the skin around the area. Of the 42 patients in the intervention group (93.3%), their surrounding skin was normal. Only 3 patients (6.7%) had abnormal skin. In the control group, 40

patients (88.9%) had normal skin, while 5 patients (11.1%) still had abnormal findings.

Discussion

Hypomagnesemia can worsen the glycaemic control in DM, and both micro- and macrovascular complications of diabetes are strongly associated with hyperglycemia and/or uncontrolled glycemia. Keshek et al.[8] reported in their study among the Turkish population that low serum magnesium levels are associated with diabetic foot ulcers. Our study showed that low magnesium levels were found among Diabetic foot ulcers allocated to the intervention group. A meta-analysis by Kurian et al[11] reported that patients with diabetic foot ulcers exhibit markedly reduced magnesium levels compared with both healthy individuals and people with diabetes without diabetic foot ulcers. Hypomagnesemia is associated with neuropathy, endothelial dysfunction, and altered platelet activity, all of which are pertinent to the development of ulcers and impaired healing processes. [12]

Magnesium supplementation in DFU

Our study found that both groups showed significant improvements in ulcer-healing parameters over time. However, the intervention group showed a comparatively greater reduction in ulcer dimensions. Also, a higher percentage of patients in the hypomagnesemia group showed wound closure and normalization of the surrounding skin at 12 weeks, indicating better wound healing. The formation of sloping edges, a key clinical sign of healing, improved significantly in both groups in a similar manner. These results are consistent with findings from other studies on how magnesium can help wounds heal. Afzali et al[13] reported that magnesium supplementation in combination with Vitamin E significantly reduced ulcer length, width, and depth compared to placebo.Similarly, Garcia et al[14] demonstrated 250 mg magnesium oxide daily for 12 weeks in DFU patients reported reduced ulcer length, width, and depth, better HbA1c, fasting glucose, and lower CRP vs placebo. Moore et al[15] reported improved wound-healing outcomes and metabolic parameters with concomitant magnesium and vitamin E supplementation, although the specific effect of magnesium could not be definitively established. Basiri et al[16] documented expedited wound healing with a nutrient-dense formulation containing magnesium; however, the specific impact of magnesium alone is unclear owing to the presence of other micronutrients. Razzaghi et al[10]. also demonstrated that a 250 mg dose of magnesium among DFU patients without prior serum magnesium documentation resulted in improved ulcer healing, as measured by ulcer dimensions, compared with placebo. However, Paturi et al[17] reported in a randomized controlled trial that no significant differences in patient distribution with respect to wound-healing characteristics, such as discharge, granulation tissue, edge sloping, surrounding normal skin, cellulitis, and exposed bone, were observed between patients who received magnesium supplementation and those who did not at four weeks of follow-up treatment. In this study, the intervention group received 400 mg of magnesium, and the control group received conventional treatment.

Reviews suggest correcting magnesium deficiency may support DFU healing, but emphasize the need for more high-quality RCTs before firm

recommendations.[18] Amini et al[19]. Reported in a systematic review indicating that certain dietary and herbal supplements, including Vitamin D, Magnesium, Vitamin E, Probiotic, Zinc, and Pycnogenol, may be beneficial for the wound healing of diabetic foot ulcers (DFUs). Hristova et al[20] in a review stated that magnesium, zinc, selenium, and copper have been postulated as beneficial for DFU as they function as antioxidants modulate immunity, cell replication and differentiation, and tissue repair.

Low magnesium levels are distinctly linked to an increased risk of diabetic foot ulcers and a decline in metabolic health. Preliminary studies indicate that magnesium, whether administered alone or in conjunction with vitamin E, may reduce ulcer size and improve glycemic and inflammatory markers. However, the evidence supporting these findings is of low certainty and does not specifically target patients with hypomagnesemia. Monitoring and addressing magnesium deficiency in diabetic foot ulcers is prudent; however, using magnesium supplementation as a specific therapy for wound healing requires more significant clinical evidence. Therefore, the magnesium levels should be controlled in patients with diabetes (with or without foot ulcers), and magnesium supplementation can be a complementary treatment in such cases.

Conclusion

Our study shows that patients in the intervention group had better healing indicators, including shorter, wider ulcers, reduced discharge, and improved restoration of the surrounding skin's integrity. The results suggest that hypomagnesemia may be an important factor in the progression and healing of diabetic foot ulcers, and that fixing it could lead to better healing outcomes. Over time, serum magnesium levels in the intervention group improved, suggesting that restoring magnesium levels may be linked to better wound-healing outcomes. The results suggest that low magnesium levels may have a clinically important effect on the progression and healing of diabetic foot ulcers, and that fixing this problem could lead to better healing outcomes.

Limitations

The sample size was smaller, and a single-center study limits its generalizability. Nutrient and drug intake for diabetes were not compared between the intervention and control groups, which are confounding factors for ulcer healing.

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