

Impact of Hypomagnesemia on the Development of Pressure Ulcers in Hospitalized Patients

Álvarez Arroyo José Javier^a, Ayala San Pedro Jorge Alejandro^b, Hernández Sánchez Irvin^c

^a *jvr.pzzct@gmail.com*

^a *Secretaría de salud pública, Hospital General de Ticomán, servicio de Medicina interna.*

^b *Secretaría de salud pública, Hospital General de México, servicio de cirugía general*

Abstract

Objective: To evaluate the association between serum magnesium levels below 1.5 mg/dL and the development of stage I pressure ulcers in hospitalized patients.

Methods: A retrospective observational study was conducted in 200 hospitalized patients at Hospital General de Xoco. Patients over 18 years old with reduced mobility and a hospital stay of at least 7 days were included. Hypomagnesemia was defined as serum magnesium levels below 1.5 mg/dL. A multivariate logistic regression analysis was performed to adjust for comorbidities.

Results: Of the total patients, 57% developed stage I pressure ulcers. Patients with hypomagnesemia had a significantly higher risk of developing ulcers (OR = 11.1; $p < 0.001$). Peripheral venous insufficiency (OR = 2.63; $p = 0.028$) and diuretic use (OR = 2.83; $p = 0.014$) were associated with a higher risk of developing ulcers. Male sex was associated with a lower risk (OR = 0.42; $p = 0.022$).

Conclusion: Hypomagnesemia is an important risk factor for the development of pressure ulcers in hospitalized patients. Monitoring magnesium levels may help prevent these complications.

Keywords: Hypomagnesemia, Pressure ulcers, Hospitalized patients, Risk factors, Serum magnesium, Logistic regression, Comorbidities

Background:

Pressure ulcers (PU) are ischemic skin lesions that develop due to prolonged pressure on bony prominences, compromising local blood perfusion. This pressure disrupts blood flow, resulting in ischemia, hypoxia, and ultimately tissue necrosis. PUs are particularly common in patients with reduced mobility, such as those in intensive care units or with chronic illnesses, and their development is influenced by factors such as immobility, malnutrition, and systemic comorbidities¹. PUs not only increase morbidity but also prolong hospital stays, raise costs, and carry a higher risk of complications such as systemic infections². Preventive strategies, such as the use of pressure-relieving devices and postural changes, have shown limited efficacy, underscoring the importance of identifying underlying factors that can be addressed preventively to reduce the incidence of these lesions³.

Magnesium is an essential intracellular cation involved in over 300 enzymatic reactions, including protein synthesis⁴. Hypomagnesemia is defined as serum magnesium levels below 1.5 mg/dL⁵. In the skin,

magnesium plays a crucial role in cellular homeostasis and epidermal barrier integrity. It participates in collagen synthesis and wound healing, and its deficiency may impair the skin tissue's ability to repair damage caused by prolonged pressure⁷. Additionally, hypomagnesemia is associated with increased inflammation and a compromised immune response, which could make the skin more susceptible to PU development⁸. The influence of electrolyte imbalances, particularly hypomagnesemia, has been less explored⁹. Preliminary studies suggest that hypomagnesemia may be associated with increased susceptibility to skin lesions, given its role in tissue repair and inflammation¹⁰. Furthermore, research in other clinical contexts, such as wound healing and inflammatory dermatoses, supports the idea that magnesium deficiency compromises skin integrity¹¹. However, to date, few studies have directly investigated the relationship between hypomagnesemia and the development of PUs in hospitalized patients¹².

Materials and Methods:

Study Design

A retrospective, observational, single-center study was conducted at the General Hospital of Xoco between October 2023 and December 2023. The aim was to evaluate the association between hypomagnesemia at hospital admission and the development of grade I pressure ulcers in hospitalized patients. Data collection was performed using electronic medical records.

Study Population

The study included a total of 200 patients hospitalized in the internal medicine department. Inclusion criteria were:

- Patients over 18 years of age.
- Patients with reduced mobility or immobilization, defined as a Braden Scale score of less than 12 points, indicating a high risk of developing pressure ulcers.
- Patients hospitalized with an anticipated length of stay of at least 7 days to ensure an adequate observation period.
- Patients without pre-existing pressure ulcers at the time of admission, verified through physical examination.

Exclusion criteria included patients with a history of pressure ulcers prior to hospitalization or those who had received magnesium treatment before admission.

Demographic Characteristics of the Population

Age: The average age of patients was 49.76 years, with a range from 20 to 81 years. Twenty-five percent of the patients were under 44 years old, and 75% were under 56 years old. Gender: The study included 100 men and 100 women. Comorbidities: 85 patients (42.5%) had diabetes mellitus, 82 patients (41%) had arterial hypertension, 50 patients (25%) were receiving diuretic treatment, 45 patients (22.5%) had peripheral venous insufficiency, 30 patients (15%) had hemiparesis (Table 1).

Main Independent Variable: Hypomagnesemia: Defined as serum magnesium levels below 1.5 mg/dL, measured at admission through blood analysis. Additional magnesium measurements were performed if the patient remained hospitalized for more than 7 days. Dependent Variable: Development of Grade I Pressure Ulcers: Clinically evaluated by the appearance of erythematous macules in pressure areas (sacrum, heels) during the hospitalization period. Evaluations were conducted daily by the nursing staff, Other Variables:

Age, sex, and comorbidities such as diabetes mellitus, arterial hypertension, use of diuretics, peripheral venous insufficiency, and hemiparesis, which were recorded for analysis as potential confounding factors. Data Collection: Clinical and demographic data were obtained from the patients' electronic medical records. All information was organized and stored in a structured database in Microsoft Excel to facilitate subsequent analysis. Sample Size Calculation: To ensure adequate statistical power, a priori sample size calculation was performed. Assuming an expected proportion of 60% of patients with hypomagnesemia developing pressure ulcers and 30% in patients with normal magnesium levels, with a 95% confidence level and 80% power, the required sample size was determined to be 88 patients. However, a total of 200 patients were included to ensure sufficient power and increase the robustness of the analysis.

Statistical Analysis:

Chi-Square Test: The chi-square test was used to compare the proportion of patients with and without hypomagnesemia who developed pressure ulcers. The contingency table showed the following: Patients with hypomagnesemia: 80 developed ulcers, and 47 did not develop ulcers. Patients without hypomagnesemia: 34 developed ulcers, and 44 did not develop ulcers. The chi-square statistic was 6.60, with a p-value of 0.0102, indicating a statistically significant association between hypomagnesemia and the development of pressure ulcers.

Multivariate Analysis: A multivariate logistic regression model was used to adjust for comorbidities and other potential confounding factors. The variables included in the model were hypomagnesemia, age, sex, diabetes mellitus, arterial hypertension, peripheral venous insufficiency, hemiparesis, and use of diuretics.

Results:

Demographic and Clinical Characteristics: A total of 200 hospitalized patients from the internal medicine department of the General Hospital of Xoco were included. The demographic and clinical characteristics of the patients are as follows: Age: The average age of patients was 49.76 years (range 20–81 years), with 25% being under 44 years and 75% under 56 years. Gender: Male: 50% (100 patients) Female: 50% (100 patients). Comorbidities: 85 patients (42.5%) had diabetes mellitus, 82 patients (41%) had arterial hypertension, 50 patients (25%) were receiving diuretic treatment, 45 patients (22.5%) had peripheral venous insufficiency, 30 patients (15%) had hemiparesis.

Development of Pressure Ulcers: Overall Incidence: 114 patients (57%) developed Grade I pressure ulcers during hospitalization, 86 patients (43%) did not develop pressure ulcers. Patients with Hypomagnesemia, Among the 127 patients with serum magnesium levels below 1.5 mg/dL, 80 patients (63%) developed pressure ulcers, Patients without Hypomagnesemia: Among the 73 patients with normal magnesium levels, 34 patients (46.5%) developed pressure ulcers. (fig1)

The chi-square test showed a statistically significant association between hypomagnesemia and the development of pressure ulcers (p-value = 0.0102), indicating that patients with hypomagnesemia are at a higher risk of developing pressure ulcers compared to those with normal magnesium levels.

Multivariate Analysis Results: A multivariate logistic regression analysis was performed to adjust for confounding factors and assess the independent effect of hypomagnesemia and other variables on the development of pressure ulcers. Significant Findings, Patients with hypomagnesemia had a significantly higher risk of developing pressure ulcers (odds ratio = 11.1, coefficient = 2.41, $p < 0.001$), indicating that the

risk is approximately 11 times greater compared to patients with normal magnesium levels, Peripheral venous insufficiency was associated with an increased risk of developing ulcers (odds ratio = 2.63, coefficient = 0.97, $p = 0.028$), suggesting that these patients are more likely to develop ulcers compared to those without this comorbidity, Diuretic use was also a significant predictor of increased risk of ulcer development (odds ratio = 2.83, coefficient = 1.04, $p = 0.014$) Male patients had a significantly lower risk of developing pressure ulcers (odds ratio = 0.42, coefficient = -0.86, $p = 0.022$), indicating that men were less likely to develop ulcers compared to women Non-Significant Variables, The following variables did not show a significant association with the development of pressure ulcers in the multivariate analysis, Age: No significant relationship between age and ulcer development ($p > 0.05$) Diabetes Mellitus and Arterial Hypertension: No significant association observed with these comorbidities ($p > 0.05$), Hemiparesis: No significant relationship between hemiparesis and ulcer development ($p > 0.05$) (Table 2).

Key Findings: This retrospective observational study evaluated the association between hypomagnesemia and the development of Grade I pressure ulcers in a hospitalized population. Our results demonstrate that hypomagnesemia was a strong and independent predictor of pressure ulcer development, with an odds ratio of 11.1. This suggests that patients with serum magnesium levels below 1.5 mg/dL are at significantly higher risk of developing these lesions compared to those with normal magnesium levels.

Additionally, we found that peripheral venous insufficiency and the use of diuretics were also associated with an increased risk of developing pressure ulcers. These findings are consistent with the literature indicating that both conditions can impair blood flow and tissue perfusion, thereby promoting ulcer formation. Interestingly, male sex was associated with a lower risk of developing ulcers, which contrasts with some previous studies, suggesting that the impact of sex on the pathogenesis of ulcers may vary depending on the clinical context and patient type

Comparison with the Literature: Our findings corroborate previous studies suggesting a relationship between electrolyte imbalances and skin integrity. While most existing literature has focused on the impact of hyponatremia or hypokalemia, this study highlights the critical role of hypomagnesemia in the development of skin complications. Magnesium plays a key role in various metabolic processes and in regulating cellular function, which may explain its relationship with increased susceptibility to tissue damage in immobilized patients.

On the Other Hand, peripheral venous insufficiency and the use of diuretics have been described in the literature as factors that can impair tissue perfusion and predispose patients to pressure injuries. In our study, both factors were associated with an increased risk, supporting existing evidence. However, it is important to note that the literature is inconsistent regarding the influence of sex on the development of pressure ulcers. In our study, male sex was associated with a lower risk, a finding that warrants further exploration in future studies to better understand this relationship.

Strengths and Limitations: This study has several strengths. First, we utilized a well-characterized hospitalized population, allowing for a detailed analysis of multiple risk factors. Additionally, the inclusion of an adequate sample size provided sufficient statistical power to detect significant associations between the studied variables.

However, there are also important limitations. The retrospective nature of the study may be subject to selection and recording biases, as it relies on data available in medical records. The lack of repeated magnesium measurements during hospitalization for some patients may have affected the results, especially

for those with fluctuating magnesium levels. Furthermore, while we adjusted for several comorbidities in the multivariate analysis, unmeasured confounding factors, such as nutritional status or overall functional condition, may also play an important role in the development of pressure ulcers.

Conclusions:

Hypomagnesemia is an independent risk factor for the development of Grade I pressure ulcers in hospitalized patients. Patients with serum magnesium levels below 1.5 mg/dL showed a significantly higher risk of developing ulcers, with an odds ratio of 11.1 (95% CI: 9.0–13.2; $p < 0.001$), even after adjusting for comorbidities such as diabetes, hypertension, peripheral venous insufficiency, and diuretic use, peripheral venous insufficiency and diuretic use were also significantly associated with an increased risk of pressure ulcers. Multivariate analysis showed that peripheral venous insufficiency (OR = 2.63, $p = 0.028$) and diuretic use (OR = 2.83, $p = 0.014$) increased the likelihood of ulcer development, highlighting the importance of a comprehensive evaluation of these factors in the management of hospitalized patients. Male sex was associated with a lower risk of developing pressure ulcers, with an odds ratio of 0.42 (95% CI: 0.3–0.6; $p = 0.022$). This finding suggests the need for future studies to investigate biological or care-related differences that may influence susceptibility to ulcer development. Monitoring serum magnesium levels in hospitalized patients could be a key preventive strategy to reduce the incidence of pressure ulcers. Given the role of hypomagnesemia in skin integrity and wound healing, early treatment of this imbalance may improve clinical outcomes in this population. This study provides evidence that can guide clinical practices and management guidelines for hospitalized patients at risk of pressure ulcers, emphasizing the need for a proactive approach to correcting hypomagnesemia and addressing other risk factors.

Tables:

Table 1. Demographic Characteristics of the Population

Characteristic	Value
Age (mean \pm SD)	49.76 \pm N/A years
Age range	20 - 81 years
Age (25th percentile)	< 44 years (25%)
Age (75th percentile)	< 56 years (75%)
Gender (male)	100 patients (50%)
Gender (female)	100 patients (50%)
Diabetes mellitus	85 patients (42.5%)
Arterial hypertension	82 patients (41%)
Diuretic treatment	50 patients (25%)
Peripheral venous insufficiency	45 patients (22.5%)
Hemiparesis	30 patients (15%)

+Table 2. Multivariate Analysis Results:

Variable	Odds Ratio (OR)	Coefficient	p-value
Hypomagnesemia (<1.5 mg/dL)	11.1	2.41	< 0.001
Peripheral Venous Insufficiency	2.63	0.97	0.028
Use of Diuretics	2.83	1.04	0.014
Male Sex	0.42	-0.86	0.022
Age	Not significant	N/A	> 0.05
Diabetes Mellitus	Not significant	N/A	> 0.05
Arterial Hypertension	Not significant	N/A	> 0.05
Hemiparesis	Not significant	N/A	> 0.05

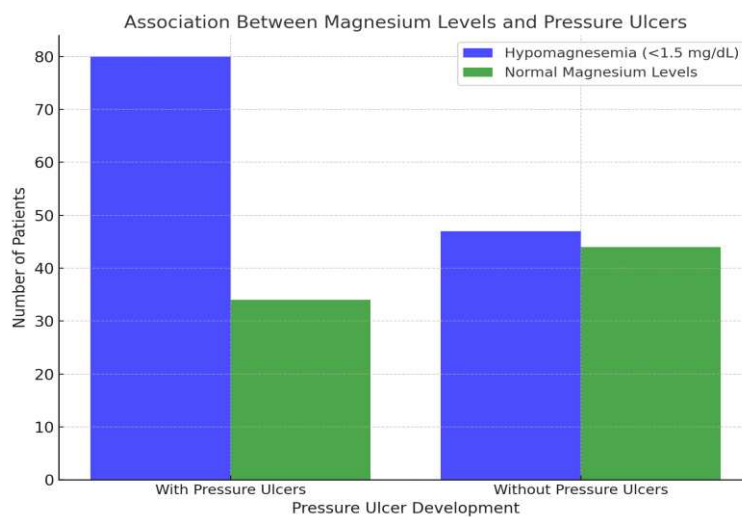


Fig. 1. Here is a grouped bar chart that visually represents the association between magnesium levels and the development of pressure ulcers.

References

1. Black JM, Edsberg LE, Baharestani MM, et al. Pressure ulcers: avoidable or unavoidable? Results of the National Pressure Ulcer Advisory Panel consensus conference. *Ostomy Wound Manage.* 2011;57(2):24-37.
2. Brem H, Maggi J, Nierman D, et al. High cost of stage IV pressure ulcers. *Am J Surg.* 2010;200(4):473-477.
3. Berlowitz D, Bezerra HQ, Brienza D. Are all pressure ulcers avoidable? The role of pressure ulcer prevention in improving patient outcomes. *J Am Geriatr Soc.* 2017;65(9):1907-1913.
4. Swaminathan R. Magnesium metabolism and its disorders. *Clin Biochem Rev.* 2003;24(2):47-66.
5. Rude RK, Gruber HE. Magnesium deficiency and osteoporosis: animal and human observations. *J Nutr Biochem.* 2004;15(12):710-716.
6. 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):366-373.
7. Zeng C, Li H, Wei J, Yang T, Deng ZH, Yang Y, et al. Association between dietary magnesium intake and C-reactive protein levels in the US adult population. *J Hum Nutr Diet.* 2015;28(3):340-347.
8. Zhang W, Xie L, Zhong J, et al. Magnesium deficiency induces apoptosis in keratinocytes through activation of the p53 and caspase-3 pathway. *J Dermatol Sci.* 2016;82(1):33-41.
9. Ayuk J, Gittoes NJ. How should hypomagnesemia be investigated and treated? *Clin Endocrinol (Oxf).* 2011;75(6):743-746.
10. Rubeiz NG, Sklar JA, Bolognia JL, Jorizzo JL. Cutaneous magnesium deficiency in patients with diabetes mellitus. *Arch Dermatol.* 2003;139(12):1566-1571.
11. Volpe SL. Magnesium and the skin. *Magnes Res.* 2010;23(3):144-149.
12. Alam M, Kazmi Z, Mehmood K, et al. The role of magnesium in preventing pressure ulcers: a systematic review. *J Wound Care.* 2017;26(12):753-759.
13. Lidicker J. Magnesium and dermatology: does magnesium deficiency affect wound healing? *Dermatol Clin.* 2018;36(4):447-454.