ORIGINAL ARTICLE

THE RELATIONSHIP BETWEEN NEUROPATHIC PAIN AND SERUM MAGNESIUM LEVELS IN STROKE PATIENTS

İnme Hastalarında Nöropatik Ağrı ile Serum Magnezyum Düzeyleri Arasındaki İlişki

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Geliş tarihi/Received: 04.11.2025
Kabul tarihi/Accepted: 22.01.2026
DOI: 10.16919/bozoktip.1817687

Bozok Tıp Derg 2026;16(1):78-86
Bozok Med J 2026;16(1):78-86

ABSTRACT

Objective: This study aimed to investigate the relationship between neuropathic pain and serum magnesium levels in post-stroke patients.

Material and Methods: Consecutive patients who had completed the acute phase and were eligible for rehabilitation were included. Sociodemographic and clinical data, comorbidities, and medications were recorded at baseline. Neuropathic pain was assessed using the Douleur Neuropathique 4 (DN4) questionnaire, and motor and gait functions were evaluated using the Brunnstrom Recovery Stages and Functional Ambulation Classification (FAC). Assessments were repeated after a four-week rehabilitation program.

Results: A total of 110 patients (74.5% ischemic stroke) were analyzed, with a median time since onset of 6 months (IQR: 4-12.8). Brunnstrom stages and FAC scores significantly improved post-treatment (all $p<0.001$), while DN4 scores decreased (median (3 (1-5) vs. 2 (0-3), $p<0.001$). Baseline serum magnesium was negatively correlated with post-treatment DN4 scores ($\rho=-0.219$, $p=0.022$) and positively correlated with baseline and post-treatment FAC and upper-limb Brunnstrom stages ($\rho=0.274$, 0.271 and 0.210, respectively, $p<0.05$ for all).

Conclusion: Lower serum magnesium concentrations were associated with higher neuropathic pain intensity and poorer functional improvement in this cohort. As the observational design of our study limits the ability to establish causality, further prospective trials are necessary to confirm this association and explore potential pathophysiological pathways.

Keywords: *Pain; Stroke; Stroke Rehabilitation; Neuropathic Pain; Magnesium*

ÖZET

Amaç: Bu çalışma, inme sonrası hastalarda nöropatik ağrı ile serum magnezyum düzeyleri arasındaki ilişkiyi araştırmıştır.

Gereç ve Yöntemler: Akut dönemi tamamlamış ve rehabilitasyon için uygun hastalar çalışmaya dâhil edilmiştir. Katılımcıların sosyodemografik ve klinik özellikleri, eşlik eden hastalıkları ve ilaç kullanımları kaydedilmiştir. Nöropatik ağrı Douleur Neuropathique 4 (DN4) anketiyle; motor ve yürüme fonksiyonları ise sırasıyla Brunnstrom Evreleri ve Fonksiyonel Ambulasyon Sınıflaması (FAS) ile değerlendirilmiştir. Değerlendirmeler, başlangıçta ve dört haftalık rehabilitasyon programı sonrasında tekrarlanmıştır.

Bulgular: Analize alınan 110 hastanın %74,5'i iskemik inme geçirmiş olup, inme başlangıcından itibaren medyan süre 6 aydı (IQR: 4-12,8). Tedavi sonrası Brunnstrom evreleri ve FAS skorlarında anlamlı artış (tümü $p<0,001$), DN4 skorlarında ise azalma saptandı (ortanca (3 (1-5)'den 2 (0-3)'ye, $p<0,001$). Serum magnezyum düzeyi, tedavi sonrası DN4 skorlarıyla negatif ($\rho=-0,219$, $p=0,022$) ve motor ile fonksiyonel göstergelerle pozitif korelasyon gösterdi (sırasıyla $\rho=0,274$, 0,271 ve 0,210, $p<0,05$).

Sonuç: Bu çalışmada daha düşük serum magnezyum düzeyleri, daha yüksek nöropatik ağrı skorları ve daha düşük fonksiyonel iyileşme çıktıları ile ilişkili bulunmuştur. Çalışmanın gözlemsel tasarımı nedensel çıkarıma izin vermediğinden, bu ilişkinin doğrulanması ve olası mekanizmaların araştırılması için ileriye dönük çalışmalar gereklidir.

Anahtar Kelimeler: *Ağrı; İnme; İnme Rehabilitasyonu; Nöropatik Ağrı; Magnezyum*

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INTRODUCTION

Stroke is a common neurological condition worldwide, associated with high rates of disability and mortality, and increased healthcare burden (1). Stroke not only affects the patient's functional status and independence, but may also lead to stroke-related pain syndromes, which further compromise recovery and functional outcomes (2).

Neuropathic pain is defined as pain associated with sensory symptoms and signs resulting from a lesion in the peripheral or central nervous system, or both (3). The pathophysiology of neuropathic pain comprises peripheral and central sensitization. Altered in ion channel activity, triggering of immune cells, mediators released by glial cells play part in the sensitization (4). Posttranslational modification of the N-Methyl-D-Aspartate (NMDA) receptors are the basis of central sensitization and trigger subsequent process. These changes cause a significant increase in excitability by eliminating the voltage-dependent magnesium block even in the absence of depolarization and extending the time the channel remains open (5). Post-stroke neuropathic pain occurs in approximately 11% of individuals who have experienced a stroke (6). Symptoms including hyperalgesia and allodynia may be present. Symptoms may appear right after the stroke, most patients develop symptoms within one to three months (7). The most common form is central post-stroke pain, which typically develops as a result of lesions in the thalamus or other central structures. However, its diagnosis is often delayed in clinical practice due to the possibility of being confused with other types of pain, such as spasticity-related pain or musculoskeletal pain. Magnesium is being investigated as an alternative treatment for chronic pain in various review studies (8, 9).

Magnesium ranks as the fourth most plentiful element in the human body (10). Over 99% of the total magnesium (Mg^{2+}) in the human body is located within the intracellular compartment. A significant portion of this intracellular magnesium is stored in the bones, muscles, soft tissues, and various organs. In contrast, only 1 to 2 is present in the blood and extracellular fluids (11,12). A serum magnesium concentration between 1.82 and 2.07 mg/dL is regarded as reflective of an adequate systemic magnesium balance (13). Assessment of magnesium status remains critically important but is complicated by methodological limitations. Serum magnesium measurement is most frequently employed due to its convenience, yet it lacks a reliable correlation with the total body magnesium content or tissue-specific magnesium concentrations (14). Magnesium functions a blocker of the ion channel on the NMDA receptor, thereby inhibiting extracellular calcium influx into the cell. It is believed that this mechanism plays important role in preventing central sensitization (15). Neuropathic pain remains a challenging condition to treat despite advancements in medical science. Therefore, NMDA receptor-dependent pathways and magnesium, a physiological antagonist of NMDA receptors, have attracted interest in neuropathic pain research (16). Magnesium, known for its regulatory effects on neuronal excitability and NMDA receptor activity, is thought to play a potential role in pain modulation; however, its association with post-stroke neuropathic pain has not yet been adequately investigated.

The aim of this study is to investigate the possible relationship between the neuropathic pain and serum magnesium levels in individuals with post-stroke hemiplegia.

MATERIAL AND METHOD

This observational study included consecutive patients with a diagnosis of stroke who presented to the Department of Physical Medicine and Rehabilitation outpatient clinic at Izzet Baysal Physical Therapy and Rehabilitation Education and Research Hospital. The study protocol was approved by the Non-Interventional Clinical Research Ethics Committee of Bolu Abant İzzet Baysal University (approval no: 2025/205). All participants provided written informed consent prior to inclusion, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Inclusion Criteria: Patients aged 18 years or older who had completed the acute stroke phase and were able to participate in the rehabilitation program were considered eligible. Patients who have developed hemiplegia following a cerebrovascular event. Patients aged between 18 and 75 years. Patients possessing the intellectual and social capacity to provide informed consent, comply with questionnaires, and adhere to

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treatment.

Exclusion criteria: Patients with a history of malignancy, active infection, hepatic or renal failure, or magnesium supplementation within the previous six months were excluded. In addition, those with severe cognitive impairment, unstable systemic disease, missing laboratory data, or unwillingness to provide informed consent were not included in the study. A total of 110 patients met the inclusion and exclusion criteria and were included in the final analysis.

Patients' sociodemographic and clinical characteristics (age, sex, marital status, educational level, occupation, body mass index (BMI), current medications (proton pump inhibitors (PPI), antihypertensive, gabapentin, pregabalin and vitamin B12 supplementation), habits (smoking and alcohol), comorbidities, disease duration, stroke type and side of hemiplegia) were recorded at baseline. Neuropathic pain was evaluated using the Douleur Neuropathique 4 (DN4) questionnaire and motor function was assessed by the Brunnstrom Recovery Stages for the upper limb, hand, and lower limb. Gait ability was evaluated using the Functional Ambulation Classification (FAC). The DN4 questionnaire and functional assessments were performed at the baseline and repeated after 4-week rehabilitation program.

The Douleur Neuropathique 4 (DN4) questionnaire is a frequently used tool to evaluate the presence of neuropathic pain. It includes questions about the presence of burning sensations, painful cold, electric shocks, tingling, pins and needles, numbness, and itching. Each 'yes' response scores 1 point, and 'no' scores 0. The clinical examination assesses the presence of hypoesthesia; if present, 1 point is given. The total score ranges from 0 to 10, with scores of ≥4 indicating a high likelihood of neuropathic pain. The validity and reliability of the Turkish version have been previously established (17,18).

The Brunnstrom Staging is a commonly used scale to assess functionality after stroke. It evaluates the gradual recovery of motor abilities, from passive to active movements, as well as the degree of spasticity and whether movements are performed in a coordinated manner. The scale consists of 7 stages for the upper extremity, 6 stages for the lower extremity, and 6 stages for hand assessment (19).

The FAC is one of the most commonly used scales for evaluating a patient's ambulation ability. It consists of six levels, ranging from 0 to 5, with higher scores indicating greater independence in ambulation (19,20).

Venous blood samples were collected at baseline to determine routine biochemical and hemogram parameters, including serum magnesium, albumin, C-reactive protein (CRP), folate, calcium, creatinine, hemoglobin, erythrocyte sedimentation rate (ESR), and vitamin B12 levels. Analyses were performed in the central hospital laboratory using standardized automated methods. Laboratory tests and clinical evaluations were conducted on the same day for each participant.

Statistical Analysis

All analyses were performed using JASP statistical software (version 0.95.1; University of Amsterdam, Netherlands). Data distribution was evaluated with the Shapiro-Wilk test and histograms. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR), and categorical variables as frequencies and percentages. Between-time comparisons were made using the Wilcoxon signed-rank test. Associations between biochemical markers and clinical scores were examined using Spearman's rank correlation coefficient (ρ).

A p-value <0.05 was considered statistically significant.

RESULTS

A total of 110 post-stroke patients (52 females, 58 males) with a median age of 64.5 (57-71.8) years were included in the study. The median time since stroke onset was 6 (4-12.8) months. Most participants had ischemic stroke (74.5%), and hypertension was the most common comorbidity (86.4%). Baseline demographic, clinical, and laboratory parameters are summarized in Table 1.

Following the rehabilitation program, significant improvements were observed in motor and functional outcomes. Brunnstrom stages of the upper limb, hand, and lower limb, as well as the Functional Ambulation Classification (FAC), all increased significantly from baseline to post-treatment (all p<0.001; Table 2). Additionally, DN4 questionnaire scores, indicating

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Table 1. Demographic, clinical and laboratory parameters of patients

Parameters	
Age, years	64.5 (57-71.8)
Sex, female/male	52 (47.3%) / 58 (52.7%)
Body mass index, kg/m²	27.7 (24.3-30.1)
Marital status, married / single	104 (94.5%) / 6 (5.5%)
Education, illiterate / primary school / secondary school / high school / university	8 (7.3%) / 70 (63.6%) / 13 (11.8%) / 13 (11.8%) / 6 (5.5%)
Occupation, employed / retired / housewife	29 (26.4%) / 33 (30%) / 48 (43.6%)
Stroke Type, hemorrhagic / ischemic	28 (25.5%) / 82 (74.5%)
Affected Side, right / left	53 (48.2%) / 57 (51.8%)
Lesion Location, MCA / ACA / PCA / basal ganglia / thalamus / pons / cerebellum	62 (56.4%) / 7 (6.4%) / 4 (3.6%) / 17 (15.5%) / 14 (12.7%) / 3 (2.7%) / 3 (2.7%)
Time since stroke onset, months	6 (4 to 12.8)
Hypertension	95 (86.4%)
Diabetes mellitus	40 (36.4%)
Coronary artery disease	42 (38.2%)
Other additional disease	65 (59.1%)
Smoking	31 (28.2%)
Alcohol	8 (7.3%)
Proton pump inhibitor Tx	19 (17.3%)
Antihypertensive Tx	95 (86.4%)
Vitamin B supplementation	29 (26.4%)
Pregabalin Tx	4 (3.6%)
Gabapentin Tx	10 (9.1%)
Magnesium, mg/dL	1.9 (1.8 to 2)
Albumin, g/L	43 (39.5 to 45)
C-Reactive protein, mg/L	3.6 (2 to 10)
Folate, ng/mL	5.4 (3.9 to 7.88)
Calcium, mg/dL	9.4 ± 0.5
Creatinine, mg/dL	0.8 (0.647 to 0.907)
Hemoglobin, g/dL	13.6 ± 1.6
Erythrocyte sedimentation Rate, mm/h	19.5 (13 to 28.8)
Vitamin B12, pg/mL	360 (285 to 489)

Data are expressed as mean ± SD, median (IQR), or n (%). Tx: Treatment, MCA: Middle cerebral artery, ACA: Anterior cerebral artery, PCA: Posterior cerebral artery, kg/m²: Kilogram/square meter, mg/dL: Milligram/deciliter, g/L: Gram/liter, mg/L: Milligram/liter, ng/mL: Nanogram/mililitre, g/dL: Fram/deciliter, mm/h: Millimeter/hour, pg/mL: Picogram/milliliter

Table 2. Changes in motor, functional, and neuropathic pain scores before and after rehabilitation

	Baseline	Post-treatment	Delta change	P value*
Brunnstrom stage - upper Limb	3 (2-5)	3 (2-5)	0 (0 to 1)	<0.001
Brunnstrom stage - hand	2 (1-4)	2.5 (1-5)	0 (0 to 1)	<0.001
Brunnstrom stage - lower Limb	4 (3-4)	4 (3-5)	0 (0 to 1)	<0.001
Functional ambulation classification	2.5 (0-5)	4 (1-5)	0 (0 to 1)	<0.001
DN4 questionnaire	3 (1-5)	2 (0-3)	0 (-2 to 0)	<0.001

Data are presented as median (interquartile range). *:Wilcoxon signed-rank test. DN4: Douleur Neuropathique 4

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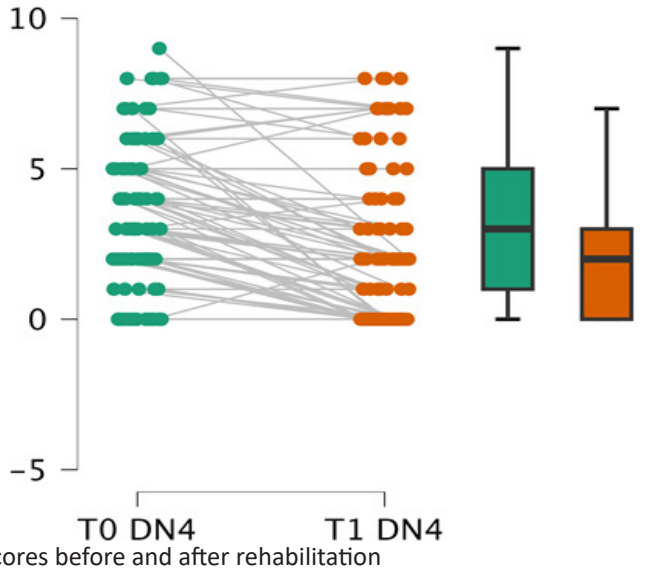


Figure 1. Distribution of DN4 scores before and after rehabilitation
The box-and-whisker plots illustrate the distribution and individual changes in DN4 (Douleur Neuropathique 4) scores before and after the rehabilitation program. Each gray line on the left panel represents an individual patient's change between baseline and post-rehabilitation assessments, demonstrating that most participants experienced a reduction in DN4 scores. The central box shows the interquartile range (IQR; Q1-Q3), with the horizontal line indicating the median value. Whiskers denote the minimum and maximum values.

neuropathic pain, significantly decreased after treatment (3 (1-5) vs. 2 (0-3), p<0.001; Figure 1).

Correlation analyses revealed a negative association between baseline serum magnesium levels and post-treatment DN4 scores (ρ=-0.219, p=0.022). In addition, magnesium showed positive correlations with motor and functional indices, including baseline Brunnstrom stages of the upper limb (ρ=0.210, p=0.028), baseline FAC (ρ=0.274, p=0.004), and post-treatment FAC (ρ=0.271, p=0.004). Serum albumin demonstrated similar positive correlations with functional outcomes (ρ=0.210-0.318, p<0.05).

No significant correlations were found between DN4 scores and serum folate, calcium, creatinine, hemoglobin, or vitamin B12 levels (Table 3).

DISCUSSION

In this study, we investigated the possible relationship between serum magnesium levels and neuropathic pain, as assessed by the DN4 questionnaire, in patients undergoing post-stroke rehabilitation. The analyses indicated that lower magnesium levels tended to be associated with higher post-treatment DN4 scores, while higher magnesium concentrations showed positive correlations with motor and functional measures such as Brunnstrom stages and ambulation scores. These findings may suggest a potential link between magnesium status and both sensory and motor recovery after stroke. Although the results cannot establish causality, they point toward the possibility that magnesium balance could have a role in post-stroke pain perception and functional improvement.

Magnesium has been increasingly discussed in the context of pain modulation through its physiological interaction with NMDA receptors and downstream effects on neuronal excitability. Recent narrative reviews synthesize experimental and clinical observations suggesting that suboptimal magnesium may facilitate hyperexcitability and pro-nociceptive signaling, including pathways linked to central sensitization (21,22). Clinically, contemporary syntheses show mixed but generally modest analgesic signals that appear context-dependent: recent meta-analyses and reviews report reductions in postoperative pain and opioid consumption with intravenous magnesium in perioperative settings, while evidence specific to neuropathic pain phenotypes

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Table 3. Correlations between biochemical parameters and clinical measures in post-stroke patients

		T0 DN4	T1 DN4	Delta DN4	T0 BS- UL	T0 BS- Hand	T0 BS- LL	T0 FAC	T1 BS- UL	T1 BS- Hand	T1 BS- LL	T1 FAC	T1 BS- UL	Delta BS- Hand	Delta BS- LL	Delta FAC
Age	ρ	0.095	0.132	0.156	-0.111	-0.028	-0.064	-0.181	-0.063	0.000	-0.088	-0.220	0.066	0.044	-0.034	-0.074
	p	0.326	0.168	0.103	0.247	0.771	0.504	0.058	0.514	0.996	0.362	0.021	0.493	0.648	0.724	0.439
BMI	ρ	0.256	0.149	-0.196	-0.012	0.040	0.006	-0.038	-0.022	0.044	-0.014	-0.108	-0.002	0.009	0.015	-0.080
	p	0.007	0.121	0.040	0.905	0.675	0.947	0.692	0.818	0.649	0.882	0.263	0.987	0.925	0.875	0.403
Time since stroke onset	ρ	-0.162	-0.196	-0.022	0.195	0.209	0.133	0.295	0.106	0.151	0.065	0.217	-0.213	-0.097	-0.122	-0.112
	p	0.090	0.040	0.823	0.041	0.028	0.167	0.002	0.271	0.115	0.498	0.023	0.026	0.311	0.206	0.243
Magnesium	ρ	-0.149	-0.219	-0.106	0.210	0.135	0.122	0.274	0.163	0.129	0.150	0.271	-0.140	0.068	0.026	0.005
	p	0.121	0.022	0.271	0.028	0.158	0.205	0.004	0.089	0.180	0.117	0.004	0.146	0.478	0.789	0.960
Albumin	ρ	-0.025	-0.071	-0.169	0.195	0.109	0.210	0.295	0.118	0.090	0.176	0.318	-0.152	0.030	-0.007	-0.040
	p	0.794	0.461	0.078	0.041	0.258	0.027	0.002	0.220	0.350	0.066	0.001	0.113	0.757	0.942	0.675
CRP	ρ	0.030	0.001	-0.071	-0.130	-0.067	-0.248	-0.220	-0.028	-0.053	-0.158	-0.171	0.198	0.092	0.153	0.187
	p	0.758	0.994	0.461	0.175	0.490	0.009	0.021	0.768	0.580	0.098	0.074	0.039	0.340	0.109	0.050
Folate	ρ	0.050	0.082	-0.012	-0.046	-0.008	0.107	0.053	0.015	-0.008	0.124	0.110	0.118	-0.097	0.000	0.030
	p	0.602	0.392	0.905	0.636	0.934	0.267	0.584	0.880	0.933	0.198	0.254	0.218	0.313	0.998	0.758
Calcium	ρ	0.050	0.059	-0.064	-0.003	-0.003	0.072	0.013	0.009	-0.011	0.119	0.067	0.025	-0.055	0.092	0.056
	p	0.603	0.542	0.507	0.978	0.972	0.454	0.893	0.925	0.908	0.217	0.487	0.798	0.567	0.340	0.561
Creatinine	ρ	0.153	0.155	-0.065	0.069	0.109	0.122	0.157	0.083	0.101	0.145	0.099	0.041	0.093	0.071	-0.079
	p	0.110	0.106	0.499	0.473	0.257	0.205	0.100	0.387	0.295	0.131	0.303	0.671	0.332	0.463	0.412
Hemoglobin	ρ	-0.111	-0.083	0.013	0.205	0.169	0.179	0.219	0.203	0.189	0.167	0.268	0.032	0.065	0.056	-0.068
	p	0.249	0.386	0.894	0.032	0.077	0.062	0.022	0.033	0.048	0.081	0.005	0.741	0.497	0.562	0.480
ESR	ρ	0.183	0.193	-0.017	-0.265	-0.173	-0.251	-0.325	-0.226	-0.191	-0.195	-0.301	0.061	-0.042	0.069	0.195
	p	0.056	0.043	0.862	0.005	0.071	0.008	0.001	0.017	0.046	0.042	0.001	0.525	0.662	0.476	0.041
Vitamin B12	ρ	-0.128	-0.014	0.056	-0.045	-0.003	-0.047	-0.065	0.028	-0.027	0.013	-0.025	0.159	-0.075	0.135	0.071
	p	0.181	0.885	0.559	BMI0.642	0.976	0.624	0.501	0.769	0.780	0.891	0.795	0.097	0.439	0.161	0.461

T0: Baseline, T1: Post-treatment, DN4: Douleur Neuropathique 4, BS: Brunnstrom stage, UL: Upper limb, LL: Lower limb, FAC: Functional ambulation classification, BMI: Body mass index, ESR: Erythrocyte sedimentation rate, ρ: Spearman's correlation coefficient

remains limited and heterogeneous (23,24). In stroke populations, newer cohort data indicate that lower serum magnesium levels are associated with worse functional outcomes after ischemic stroke or transient ischemic attack (TIA). Conversely, higher-normal electrolyte profiles, including magnesium, may be related to reduced adverse events. These findings do not establish causality but provide important clinical context for exploring magnesium status alongside pain and recovery metrics in rehabilitation (25,26). Additionally, a recent study on neuropathic-pain screening supports the continued clinical utility of DN4, strengthening the interpretability of our pain phenotyping in this cohort (27).

Several studies have investigated the potential role of magnesium-either in terms of serum levels or therapeutic supplementation-in relation to neuropathic pain across different clinical conditions. A study demonstrated that magnesium treatment can reduce the severity of pain in patients with treatment-resistant chronic low back pain with a neuropathic component (28). In another study involving patients with postherpetic neuralgia, intravenous magnesium sulfate infusion resulted in a significant reduction in neuropathic pain compared with placebo (15). In the study conducted by Pickering et al., no significant difference in pain scores was observed between the oral magnesium treatment group and the placebo group after four weeks of therapy. (29). In a study involving patients with fibromyalgia, no significant association

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was found between pain scores and serum magnesium levels (30). However, another study reported a significant association between serum magnesium concentrations and pain intensity, suggesting that magnesium status may influence pain perception in this population (31). The results have been inconsistent-some studies have demonstrated significant associations or treatment effects, whereas others have found no meaningful relationship. These discrepancies may reflect differences in patient populations, pain mechanisms, or magnesium formulations and dosing protocols. In our study, a significant association was observed between baseline serum magnesium levels and post-rehabilitation neuropathic pain scores in stroke patients with neuropathic components, suggesting that magnesium status may be a relevant factor influencing pain modulation during recovery. However, since total serum magnesium is often influenced by serum albumin and does not fully reflect total body or ionized magnesium levels, these findings should be interpreted as a preliminary link that warrants confirmation with more comprehensive intracellular or ionized magnesium assessments.

While our findings provide preliminary insight into the relationship between magnesium status and neuropathic pain after stroke, several limitations should be acknowledged. The study design was cross-sectional, which precludes causal inference, and magnesium levels were assessed only once, without adjustment for dietary intake or diurnal fluctuation. Moreover, because total serum magnesium is partly protein-bound and influenced by plasma proteins (including albumin), and although albumin was measured in our cohort, residual confounding related to protein binding, nutritional status, or inflammation cannot be excluded; therefore, total serum magnesium may still incompletely reflect physiologically relevant magnesium availability. The DN4 questionnaire, although validated and clinically practical, represents a screening rather than a diagnostic tool for neuropathic pain, and its use may have led to partial misclassification. The single-center design of this study may limit the generalizability of our findings. Furthermore, the inclusion of patients receiving gabapentinoids or vitamin B supplements represents a potential source of confounding, as these treatments may influence neuropathic pain levels and associated clinical outcomes; therefore, our results should be interpreted with caution. Additionally, the sample size, although adequate for correlation analyses, may limit the generalizability of the results to broader stroke populations or different rehabilitation settings. Future studies employing longitudinal designs, repeated biochemical measurements, intracellular magnesium analyses, and multimodal pain assessments would be valuable to clarify whether magnesium supplementation or optimization of serum levels could influence neuropathic pain trajectories and functional recovery after stroke.

In conclusion, this study suggests that lower serum magnesium levels may be associated with higher neuropathic pain scores and poorer functional outcomes in patients undergoing post-stroke rehabilitation. Although the cross-sectional nature of the analysis limits causal interpretation, the findings highlight a possible link between magnesium homeostasis and post-stroke recovery processes. Further longitudinal and interventional studies are warranted to determine whether optimizing magnesium status could contribute to improved pain control and functional recovery in this population.

CONCLUSION

In conclusion, this study suggests that lower serum magnesium levels may be associated with higher neuropathic pain scores and poorer functional outcomes in patients undergoing post-stroke rehabilitation. Although the cross-sectional nature of the analysis limits causal interpretation, the findings highlight a possible link between magnesium homeostasis and post-stroke recovery processes. Further longitudinal and interventional studies are warranted to determine whether optimizing magnesium status could contribute to improved pain control and functional recovery in this population.

Acknowledgment

The authors declared that this study has received no financial support.

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