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Serum magnesium status in patients with Bell's palsy: Findings from case-control, cross- sectional, and correlational analyses

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Abstract

Background

It has been found a relationship between Bell’s palsy and hipomagnesemia. Also, diabetic cases have low serum magnesium levels and Bell’s palsy patients have high rates of diabetes. Therefore, there is a need to address the potential confounding effect of diabetes in finding that Bell’s palsy patients have low serum magnesium levels. This study aimed to investigate serum magnesium levels in patients with Bell’s palsy, considering type 2 diabetes mellitus (T2DM).

Methods

In this study, 50 Bell’s palsy patients, 50 healthy controls, and 50 T2DM patients were compared statistically in terms of serum magnesium levels. Additionally, correlation analysis was performed between serum magnesium levels and Bell’s palsy severity. Both healthy controls and T2DM patients were selected to match the characteristics of Bell’s palsy patients. The House-Brackmann Scale was used to measure the severity of Bell’s palsy.

Results

The three groups were similar in terms of demographics ($p > 0.05$). Fourteen patients with Bell's palsy had also T2DM (28%). The Bell’s palsy group had lower serum magnesium levels than the healthy controls (but not significant, $p = 0.261$). The Bell’s palsy group had higher serum magnesium levels than the T2DM group (but not significant, $p = 0.274$). However, the healthy group had significantly higher serum magnesium levels than the T2DM group ($p = 0.002$). Bell’s palsy patients with T2DM had significantly higher House-Brackmann grades than those without T2DM ($p = 0.028$). A negative correlation was found between serum magnesium levels and the House-Brackmann grades (r:-0.138, $p = 0.340$).

Conclusion

In this study, 28% of Bell’s palsy patients also had T2DM, which is associated with low serum magnesium levels. Serum magnesium levels in Bell’s palsy patients were statistically similar to those in a healthy control group. However, the co-occurrence of T2DM (28%) and the observed trend of decreased magnesium levels require further research.

Introduction

Bell’s palsy is characterized by sudden unilateral facial nerve paralysis and is the most common form of acute facial paralysis. The role of inflammation, infection, ischemia, immunity, anatomy, and cold in its

etiopathogenesis has been emphasized, but the exact causative factor remains unknown (1). Despite high recovery rates, Bell's palsy patients may experience high concerns and neurological deficits that impact their quality of life (1, 2). Therefore, there is an ongoing need for both etiological research and more effective treatment.

Type 2 diabetes mellitus (T2DM) is a common endocrine disorder associated with high morbidity and mortality due to various complaints, comorbidities, and complications (3, 4). The pathophysiology of T2DM is complex and involves increased insulin resistance, reduced β-cell function, mitochondrial dysfunction, and oxidative stress (3, 4). Also, it interacts with genetic and environmental factors (4). Successful management of T2DM is possible with medications and lifestyle interventions such as exercise and diet, but there is currently no cure (5, 6).

Magnesium is a multifunctional mineral abundant in the body. It plays a role in the immune response as a cofactor for immunoglobulin synthesis and T and B cell function. Magnesium is also essential for the biosynthesis, transport, and activation of vitamin D, which is involved in the pathogenesis of infectious diseases. As a result, magnesium has an indirect interaction with the immune system, and magnesium deficiency is associated with infections and inflammation (7–9). In addition, magnesium plays a role in many biological functions, including nerve signalling, muscle contraction, and the reduction of inflammation. It acts as a natural calcium antagonist, helping to stabilize nerve excitability and prevent excessive neuronal firing. Magnesium's neuroprotective roles are particularly important in preventing neuronal damage (10). Chronic magnesium deficiency can lead to oxidative stress and inflammation (7, 8) and has been linked to various neurological conditions, such as migraine, stroke, and peripheral neuropathy (10).

It is known that T2DM patients have low serum magnesium levels (11, 12) and Bell's palsy patients have high rates of T2DM (13). Furthermore, the findings of some recent studies suggest a relationship between Bell's palsy and hypomagnesemia (14, 15). However, these studies have not addressed the role of T2DM in this relationship, and the subject need to be investigated in more detail due to the limited data. Therefore, this study aimed to investigate serum magnesium levels in patients with Bell's palsy, also taking T2DM into consideration.

Materials and Methods

This prospective study was conducted on three different groups and includes case-control, cross-sectional, and correlational analyses. After obtaining Ethics Committee approval (Gazi Yaşargil Hospital Clinical Research Ethics Committee; Number:148; Date: September 06, 2024), 50 Bell's palsy patients, 50 healthy controls, and 50 T2DM patients were included in the study. Since it is known that T2DM patients have hypomagnesemia (11, 12), a comparison group was created with these patients. Healthy controls consisted of individuals who underwent health checks during the same period (2024–2025). Both healthy controls and T2DM patients were selected to match the demographics of patients with Bell's

palsy. Signed informed consent was obtained from each participant. The study was conducted as a prospective analysis in accordance with the ethical principles of the Declaration of Helsinki.

The severity of Bell's palsy was measured using the House-Brackmann grading scale (16). This scale is widely used and has six grades (grade I: normal - grade VI: complete paralysis) to evaluate the severity of Bell's palsy. Serum magnesium levels were measured early morning on an empty stomach using Architect Plus c16000 (Abbott, Illinois, USA). Applied inclusion and exclusion criteria in the study were as follows: Newly diagnosed cases of Bell's palsy who give informed consent were included in the study. Cases with potentially confusing factors such as traumatic or iatrogenic palsy, epilepsy, multiple sclerosis, malignancy, age < 18 and ≥ 65 years, fasting, pregnancy, lactation, mental and intake disorders, immunodeficiency, malabsorption, magnesium supplements use and missing data were excluded from the study.

Statistical Analysis

Descriptive statistics were expressed as median (IQR) or mean ± SD (min-max) (for continuous variables) or n (%) (for categorical variables). The normal distribution of variables was examined using the Kolmogorov-Smirnov test. When continuous variables exhibited a skewed distribution, at least in one of three groups, the Independent Samples Kruskal-Wallis test was used to determine the significance of differences between group medians. When continuous variables exhibited a normal distribution in all of three groups, One-Way ANOVA with Bonferroni post-hoc test was used to determine the significance of differences between group means. In addition, the Shapiro-Wilk test, the Mann-Whitney U test, the Student t-test, and the Spearman correlation test were used in subgroup analyses. Chi-square test was used to compare categorical variables between the groups. A significance level of 5% was adopted, and calculations were performed using the SPSS software program (version 28).

Results

The healthy controls and the T2DM patients were selected to match the demographics of patients with Bell's palsy. The three groups were statistically similar in terms of age ($p = 0.582$), gender ($p = 1.0$), BMI ($p = 0.097$), and working status ($p = 0.655$). The presence of T2DM among patients with Bell's palsy was 14 (28%) (Table 1).

Table 1
Comparison of the groups for demographic characteristics

Age, years	**Bell's palsy (n = 50)**	**Healthy group (n = 50)**	**Diabetes mellitus (n = 50)**	**p**
	46.0 (14.50)	**44.50 (12.25)**	**47.50 (6.25)**	**90.582**
Gender, female, n (%)	30 (60)	30 (60)	30 (60)	
Weight, kg	74.50 (13.75)	75.50 (17.25)	80.0 (12.75)	0.040
Height, m	1.65 (0.10)	1.65 (0.09)	1.68 (0.10)	0.168
BMI, kg/m^2	27.40 (4.41)	27.18 (5.27)	28.55 (6.08)	0.097
Working, A/B/C, n	27/20/3	31/16/3	26/18/6	0.655
Diabetes mellitus, n (%)	14 (28)	0 (0)	50 (100)	
Data were expressed as median (IQR) or n (%)				
A/B/C: Housewife/Employed/Unemployed				
Independent-Samples Kruskal-Wallis test was use in comparisons				

On the other hand, considering serum magnesium levels, there were significant differences among the three groups according to the One-Way ANOVA test (p = 0.004). No significant difference was found between the Bell's palsy patients and healthy controls in terms of serum magnesium levels (1.95 ± 0.13 and 2.01 ± 0.15, respectively) (p = 0.261; 95% CI -0.133 to 0.022). Similarly, the Bell's palsy patients and the T2DM patients had statistically similar serum magnesium levels (1.95 ± 0.13 and 1.90 ± 0.20, respectively) (p = 0.274; 95% CI -0.023 to 0.132). However, the healthy controls had significantly higher serum magnesium levels than the T2DM patients (2.01 ± 0.15 and 1.90 ± 0.20, respectively) (p = 0.002; 95% CI 0.032 to 0.187) (Table 2). Figure 1 illustrates the serum magnesium levels (mg/dL) in the three groups.

Table 2
Comparison of the groups for serum magnesium levels

	Bell's palsy (n = 50)	Healthy group (n = 50)	Diabetes mellitus (n = 50)	p
Serum Mg level, mg/dL	1.95 ± 0.13 (1.70–2.20)	2.01 ± 0.15 (1.75–2.51)	1.90 ± 0.20 (1.34–2.31)	0.004[a] 0.261[b] 0.274[c] 0.002[d]
Data were expressed as mean ± SD (min-max)				
[a]: One-Way ANOVA with Bonferroni post-hoc test				
[b]: Bell's palsy vs Healthy				
[c]: Bell's palsy vs Diabetes mellitus				
[d]: Healthy vs Diabetes mellitus				

In the Bell's palsy patients, a subgroup analysis including a comparison of serum Mg levels between patients with (n = 14) and without T2DM (n = 36) was performed (Student t-test). Accordingly, no statistically significant difference was found between the Bell's palsy patients with and without T2DM (p = 0.086) (Table 3). However, Bell's palsy patients with T2DM had significantly higher House-Brackmann grades than those without T2DM (p = 0.028). In addition, a negative correlation was found between serum magnesium levels and the House-Brackmann grades, but this did not reach a significant level statistically (Spearman test; r=-0.138; p = 0.340).

Table 3
Comparison of the groups for demographic characteristics

Age, years	**Bell's palsy with Diabetes (n = 14)**	**Bell's palsy without Diabetes (n = 36)**	**p**
	53.57 ± 6.94 (42–64)	**41.67 ± 9.06 (27–58)**	**0.001[a]**
Gender, female, n (%)	8 (57.14)	22 (61.11)	1.0
Weight, kg	80.14 ± 14.02 (65–116)	73.17 ± 11.22 (52–104)	0.072[a]
Height, m	1.65 (0.08)	1.65 (0.09)	0.760[b]
BMI, kg/m^2	27.98 (5.11)	27.26 (4.47)	0.082[b]
Working, HW/E/UE, n	7/5/2	20/15/1	0.305
House-Brackmann, grade	4.50 (3.0)	4.0 (1.0)	0.028[b]
Serum Mg, mg/dL	1.90 ± 0.14 (1.70–2.16)	1.97 ± 0.12 (1.72–2.20)	0.086[b]
Data were expressed as mean ± SD (min-max) or median (IQR) or n (%)			
HW/E/UE: Housewife/Employed/Unemployed			
[a]: Student t-test			
[b]: Mann-Whitney U test			

Discussion

This prospective study included case-control, cross-sectional, and correlational analyses on the serum magnesium status in patients with Bell's palsy, considering T2DM. The study results showed that serum magnesium levels tended to be lower in Bell's palsy patients compared to healthy controls, and tended to be higher in compared to patients with T2DM. However, these trends were not statistically significant. Given that patients with T2DM have decreased serum magnesium levels (11, 12), these results may be related to the fact that 28% of Bell's palsy patients in this study also had T2DM. Consistent with previous findings (11, 12), T2DM patients had significantly lower serum magnesium levels than the healthy individuals in this study.

There is limited data on the relationship between serum magnesium status and Bell's palsy. To date, only two studies have addressed this subject (14, 15). Moreover, in the previous studies, the potential role of T2DM in this relationship has not been considered. Demir et al. (14) have used atomic absorption spectrometry and have revealed that patients with Bell's palsy have significantly lower magnesium concentration compared to healthy controls (14). Also, Kumar et al. (15) have focused the potential

effect of magnesium on the Bell's palsy progression and recovery. They found that higher serum magnesium levels are associated with better recovery in Bell's palsy patients (15).

In addition to low serum magnesium levels in patients with T2DM (11, 12), the development of Bell's palsy is associated with T2DM (13), and magnesium therapy improves hyperglycemia (17). Therefore, it is possible that a decreased serum magnesium levels in patients with Bell's palsy (14) may be due to diabetic cases, and the better recovery in Bell's palsy patients with higher serum magnesium levels (15) may be due to improvement in glycemic control thanks to magnesium effect. Indeed, 28% of patients with Bell's palsy in the current study had T2DM, and patients with T2DM had lower mean magnesium levels than those without T2DM. However, previous studies on the relationship between magnesium and Bell's palsy have not considered the presence of T2DM (14, 15). Therefore, comparisons cannot be made between the findings of different studies in this respect.

Although the Bell's palsy group in this study had lower serum magnesium levels than the healthy controls, this was not statistically significant. However, Demir et al. (14) reported that patients with Bell's palsy have significantly lower serum magnesium levels than healthy controls (14). This difference may be related to the different measurement methods used in the two studies.

Although the Bell's palsy patients in this study had higher serum magnesium levels than the T2DM patients, this was not statistically significant. This is likely because the difference was reduced due to the presence of T2DM (28%) among patients with Bell's palsy in this study. Because the findings of the current study and literature data have shown that patients with T2DM have significantly lower serum magnesium levels than healthy individuals (11, 12).

According to the current study, Bell's palsy patients with T2DM exhibited significantly higher disease severity (House-Brackmann grade) than those without ($p = 0.028$). Also, there was a negative correlation between serum magnesium levels and House-Brackmann grades in Bell's palsy patients. Previous studies have reported that Bell's palsy is more severe and has a worse prognosis in those with type T2DM (18). However, previous studies have not evaluated the link between Bell's palsy severity and serum magnesium levels. Therefore, the potential link between magnesium deficiency and disease severity in Bell's palsy deserves further investigation in the future.

In conclusion, we found no statistically significant difference in serum magnesium levels between patients with Bell's palsy and healthy controls. Nevertheless, the coexistence of T2DM and the observed trend toward lower magnesium concentrations warrant further investigation in larger prospective studies.

Declarations

Conflicts of interest/Competing interests:

The authors have no conflicts of interest to declare.

Consent to participate:

Confirmation of participation has been received from all participants.

Written Consent for publication

Written consent was obtained from all participants.

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Author Contribution

Concept: S.G.K., Ö.E.; Design-Methods: S.G.K., Ö.E.; Formal Analysis: S.G.K., Ö.E.; Data Collection: Ö.E.; Writing-Original Draft: S.G.K; Writing-Review-Editing: S.G.K., Ö.E.

Availability of data and material:

The data that support the findings of this study are available on request from the corresponding author.

Code availability:

Not applicable.

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Figures

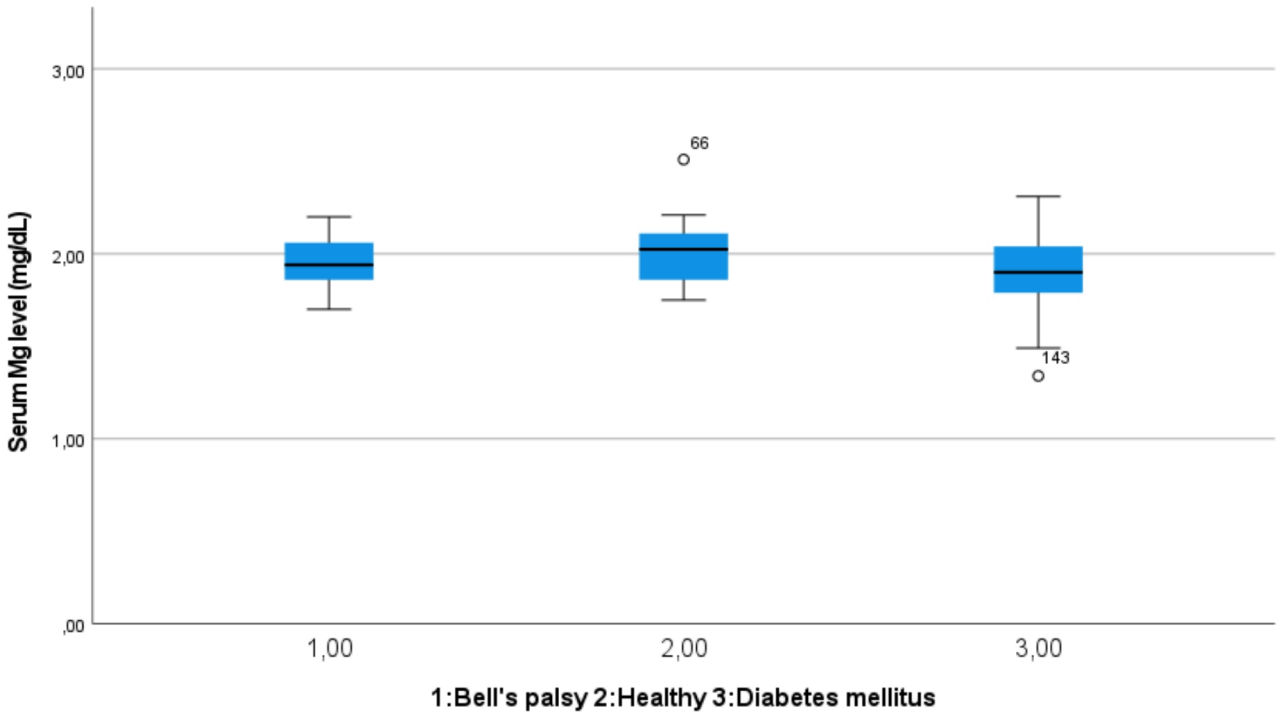

Figure 1

Comparison of serum magnesium levels between groups