

Original Article

Investigation of the Relationship Between Epstein-Barr Virus and Serum Magnesium Levels in Lymphoma Patients

✉ Mehmet Batuhan Duman¹, ✉ Fuat Erdem², ✉ Gülden Sincan²

¹Gebze Fatih State Hospital, Clinic of Internal Medicine, Kocaeli, Türkiye

²Atatürk University Faculty of Medicine, Department of Hematology, Erzurum, Türkiye

ABSTRACT

Aim: Lymphoma pathogenesis involves complex interactions of genetic, environmental, and immunological factors. While Epstein-Barr virüs (EBV) is recognized as a contributing factor in several lymphoma subtypes, and magnesium is essential for immune regulation. This study investigated the relationship between serum magnesium levels and EBV positivity in lymphoma patients, and the impact of magnesium on survival.

Methods: This retrospective study included 108 lymphoma patients. Demographic, clinical, EBV status, and serum magnesium levels were collected. Statistical analyses, including the chi-square test, Kaplan-Meier estimation, and receiver operating characteristic curve analysis, were performed.

Results: Serum magnesium levels were significantly lower in EBV-positive patients (1.82 mg/dL) compared to EBV-negative patients (1.98 mg/dL) ($p=0.049$). Patients with magnesium levels ≤ 1.68 mg/dL had significantly shorter overall survival and disease-free survival, particularly in Hodgkin lymphoma (HL). In diffuse large B-cell lymphoma, diminished magnesium levels were associated with inferior survival outcomes. Low magnesium levels were also associated with advanced disease stage ($p=0.006$).

Conclusion: Lower serum magnesium concentrations in lymphoma patients are associated with EBV positivity and poorer survival, particularly within the HL subgroup. Magnesium levels may be associated with prognosis in lymphoma patients; however, larger prospective studies are needed to determine their independent prognostic value.

Keywords: Magnesium, EBV, lymphoma, prognosis

Introduction

Lymphoma is a malignancy characterized by the uncontrolled proliferation of lymphocytes, with a pathobiology driven by complex interactions among genetic, environmental, and immunological factors. Risk factors contributing to lymphoma development include autoimmune conditions, chronic immunosuppression (e.g., post-transplant medications), infectious agents [e.g., Epstein-Barr virus (EBV) and human immunodeficiency virus], chemical exposures, and genetic predispositions. Furthermore, chronic inflammation and immune dysregulation within lymphoid tissues play a pivotal role in lymphoma pathogenesis [1-3].

EBV is a ubiquitous human herpesvirus that frequently persists asymptomatically in the general population. However, EBV

infection has been associated with various systemic diseases and malignancies. EBV is a well-established etiological factor in several lymphoma subtypes due to its tropism for B lymphocytes [4]. In EBV-associated malignancies, expression of latent viral proteins, including EBNA-1, LMP-1, and LMP-2, has been demonstrated [5-7]. The association between EBV and Burkitt lymphoma (BL) is closely related to c-myc translocations, where EBV infection may contribute to oncogenic activation [8]. In addition to B-cell involvement, EBV has also been shown to infect NK and T cells, contributing to extranodal NK/T-cell lymphomas [9]. In Hodgkin lymphoma (HL), EBV is more commonly associated with nodular sclerosis and mixed cellularity subtypes, whereas nodular lymphocyte-predominant HL is typically EBV-negative. Approximately 10-15% of diffuse large B-cell lymphoma (DLBCL) cases are EBV-

Address for Correspondence: Gülden Sincan MD, Atatürk University, Yakutiye Research Hospital, Clinic of Hematology, Erzurum, Türkiye

E-mail: guldensincan@gmail.com **ORCID ID:** orcid.org/0000-0002-7671-7628

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positive, with a higher incidence observed in elderly patients. Magnesium is a critical regulator of immune function, particularly in T-cell activation and NK cell-mediated cytotoxicity. Intracellular magnesium flux is essential for proper T-cell receptor signaling and activation of cytotoxic pathways. Alterations in magnesium homeostasis may impair immune surveillance against viral infections such as EBV by affecting the cytotoxic functions of NK and CD8⁺ T cells. Studies of X-linked MAGT1 deficiency with increased susceptibility to EBV infection and N-linked glycosylation defects (XMEN disease) provide important mechanistic insights into this relationship. MAGT1 is involved in cellular magnesium homeostasis and N-linked glycosylation of several immune proteins, and MAGT1 deficiency is associated with reduced NKG2D expression and impaired cytotoxic function of NK and CD8⁺ T cells, which are critical for controlling EBV-infected cells [10-12]. These findings provide a biologically plausible framework linking magnesium homeostasis, impaired immune control of EBV, and EBV-associated lymphomagenesis.

Magnesium deficiency has been associated with impaired immune responses and altered inflammatory pathways, which may contribute to the development and progression of malignancies, including lymphoma [13-15]. Despite increasing evidence regarding intracellular magnesium dynamics in immune regulation, whether circulating serum magnesium levels reflect this immunological impairment in lymphoma patients remains unclear. Given that serum magnesium is a readily accessible clinical parameter, investigating its relationship with EBV positivity may provide clinical insights into the potential interplay between magnesium status and EBV-associated lymphoma biology [12].

Therefore, this study aimed to investigate the association between serum magnesium levels and EBV positivity in lymphoma patients, as well as to evaluate the prognostic significance of magnesium in terms of overall and disease-free survival.

Methods

This retrospective study included 108 patients diagnosed with lymphoma between 2020 and 2022 and followed at the Department of Hematology, Atatürk University Faculty of Medicine. Ethical approval for this study was obtained from the Ethics Committee of Atatürk University Faculty of Medicine (number: B.30.2.ATA.0.01.00/35, date: 29.12.2022). Informed consent was not obtained due to the retrospective nature of the study. Patients aged ≥ 18 years with available EBV status and serum magnesium measurements at the time of diagnosis or initial evaluation were included in the study. Patients with missing EBV status or serum magnesium measurements and those with incomplete clinical data were excluded.

Patient information, including demographic data (age, gender), lymphoma subtype, EBV status, disease stage, and remission status, was retrieved from patient files and the hospital information system. EBV status was determined using tissue-based and/or peripheral blood-based methods. Tissue-based

EBV positivity was defined as the presence of EBV-encoded RNA (EBER) expression in tumor cells detected by *in situ* hybridization (ISH). In addition, EBV DNA levels in peripheral blood were evaluated using polymerase chain reaction (PCR) assays. Because of the retrospective nature of the study, EBER and peripheral blood EBV DNA PCR results were not available for all patients. Some patients underwent both testing modalities, whereas others had results available from only one method. All EBV assessments were obtained at the time of diagnosis or during initial disease evaluation, and the results were retrospectively retrieved from patient records. No additional EBV testing was conducted specifically for this study. Because tissue-based EBER positivity and peripheral blood EBV DNA PCR reflect different biological aspects of EBV infection, these parameters were analyzed separately whenever possible to minimize potential methodological heterogeneity.

Serum magnesium concentrations were obtained from routine biochemical analyses performed at the time of diagnosis. Magnesium levels were measured using standard automated laboratory methods, and the initial values at diagnosis were used for analysis.

Survival data were obtained from the hospital information system and the Ministry of Health's Death Notification System.

Staging was primarily performed using positron emission tomography/computed tomography (PET/CT). Response assessment, based on the Lugano criteria, was performed using follow-up PET/CT after completion of the planned treatment regimen. Patients were classified as achieving complete response, partial response, stable disease, or progressive disease.

Statistical Analysis

Statistical analyses were performed to evaluate the relationships between study variables and survival outcomes. Categorical variables were presented as numbers and percentages, whereas continuous variables were expressed as mean $\pm$ standard deviation, median (minimum-maximum), or median [95% confidence interval (CI)], as appropriate. For the comparison of categorical data, the chi-square test, Fisher's exact test, and the trend chi-square test were used where applicable. The Kolmogorov-Smirnov test and histogram plots were used to assess the assumption of normal distribution for the measurements. Receiver operating characteristic (ROC) curve analysis was employed to determine threshold values for survival prediction based on magnesium levels. The log-rank test (Kaplan-Meier analysis) was used to compare survival times between groups. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 20.0 (IBM Corp., Armonk, NY, USA). A p value of <0.05 was considered statistically significant.

Results

Of the patients, 65 (60.2%) were male, and 43 (39.8%) were female. The mean age of the patients was 45.1 ± 16.8 years. Fifty-five patients (50.9%) were diagnosed with HL, while 53

patients (49.1%) were diagnosed with NHL. The most common subtype of HL was nodular sclerosis HL, observed in 23 patients (46.0%), followed by mixed cellularity HL in 16 patients (32%), nodular lymphocyte predominant HL in 9 patients (18%), and lymphocyte-rich HL in 2 patients (4%). The most frequently observed non-HL (NHL) subtype was DLBCL, accounting for 76.0% (38 patients). This was followed by mantle cell lymphoma in 3 patients (6%), follicular lymphoma in 3 patients (6%), T-cell lymphoma in 2 patients (4%), anaplastic large cell lymphoma in 2 patients (4%), marginal zone lymphoma in 1 patient (2%), and BL in 1 patient (2%). Among the 70 patients with available tissue-based EBER results, 22 (31.4%) were EBER-positive. Peripheral blood EBV DNA PCR results were available for 38 patients, of whom 9 (23.7%) were PCR-positive.

Upon examining the stages of the patients, one patient (0.9%) was at stage I, 28 patients (25.9%) were at stage II, 17 patients (15.7%) were at stage III, eight patients (7.4%) were at stage IIIS, and 54 patients (50.0%) were at stage IV. When evaluating the remission status of the patients, it was found that 22 patients (20.4%) achieved a complete response, 47 patients (43.5%) had a partial response, 28 patients (25.9%) had progressive disease, and 11 patients (10.2%) had stable disease.

The median serum magnesium level was 1.92 (1.35-2.47) mg/dL. The relationship between serum magnesium level and clinical and demographic data is presented in Table 1. Based on the ROC analysis, the optimal cut-off value for serum magnesium was determined as 1.68 mg/dL. In 8 patients (7.4%), magnesium levels were ≤ 1.68 mg/dL.

According to the 1.68 mg/dL cut-off-based grouping, a statistically significant association was observed between tissue-based EBER positivity and magnesium levels ($p=0.028$), whereas no significant association was found between

magnesium levels and peripheral blood EBV DNA PCR positivity ($p=0.574$). Additionally, lower magnesium levels were significantly associated with advanced disease stage ($p=0.006$) and remission status, particularly with progressive disease ($p=0.002$) (Table 1).

In the statistical analysis of Mg values in terms of predicting mortality, the area under the curve was 0.659 (95% CI: 0.561-0.747, $p=0.086$), with a sensitivity 38.46% and specificity of 96.84% at a cut-off value of 1.68 mg/dL. In EBV-positive patients, the Mg level was 1.82 (1.35-2.47) mg/dL, while in EBV-negative patients, it was 1.98 (1.70-2.42) mg/dL ($p=0.049$).

When comparing the survival status with demographic and clinical variables, statistically significant relationships were identified with age, type of diagnosis, and Mg levels. The mean age of deceased patients (61 ± 10 years) was statistically significantly higher than that of surviving patients (43 ± 16 years) ($p<0.001$). The survival rate of patients diagnosed with HL was 96.4%, whereas the survival rate of patients diagnosed with NHL was 79.2%, with a statistically significant difference ($p=0.01$).

When survival status (alive vs. exitus) was compared according to magnesium groups within lymphoma subgroups, a statistically significant association was observed in the DLBCL subgroup, where patients with magnesium levels ≤ 1.68 mg/dL had lower survival rates than those with higher magnesium levels (20% vs. 84.8%, $p=0.008$). In contrast, no statistically significant association was found in the HL subgroup using this categorical survival status analysis ($p=0.107$) (Table 2).

In Kaplan-Meier survival time analyses, different results were observed between subgroups. In the HL subgroup, overall survival was 11.69 (3.24-20.14) months in patients with low

Table 1. Comparison of grouped Mg levels with demographic and clinical data of patients

		Mg (mg/dL)				p value
		≤1.68		>1.68		
		n	(%)	n	(%)	
Gender	Male	4	(6.2)	61	(93.8)	0.710
	Female	4	(9.3)	39	(90.7)	
Age (years)		54±23		44±16		0.142
Type of diagnosis	HL	3	(5.5)	52	(94.5)	0.485
	NHL	5	(9.4)	48	(90.6)	
EBV pathology	Negative	0	-	48	(100)	0.028
	Positive	3	(13.6)	19	(86.4)	
EBV PCR	Negative	3	(10.3)	26	(89.7)	0.574
	Positive	2	(22.2)	7	(77.8)	
Stage	Stage IV	8	(14.8)	46	(85.2)	0.006
	Other stage	0	-	54	(100)	
Remission status	Complete response	0	-	22	(100)	0.002
	Partial response	1	(2.1)	46	(97.9)	
	Progressive disease	7	(25.0)	21	(75.0)	
	Stable disease	0	-	11	(100)	

HL: Hodgkin lymphoma, NHL: Non-Hodgkin lymphoma, EBV: Epstein-Barr virus, Mg: Magnesium

Table 2. The relationship between magnesium levels and survival rates in lymphoma subgroups

Groups	Mg (mg/dL)	Alive		Exitus		p value
		n	(%)	n	(%)	
HL	≤1.68	2	(66.7)	1	(33.3)	0.107
	>1.68	51	(98.1)	1	(1.9)	
DLBCL	≤1.68	1	(20.0)	4	(80.0)	0.008
	>1.68	28	(84.8)	5	(15.2)	
Non-DLBCL NHL	≤1.68	0	(0)	0	(0)	*
	>1.68	13	(86.7)	2	(13.3)	

*The p value could not be calculated due to the absence of data in one of the data groups being compared.
 HL: Hodgkin lymphoma, NHL: Non-Hodgkin lymphoma, DLBCL: Diffuse large B-cell lymphoma, Mg: Magnesium

Mg levels and 156.35 (149.03-163.67) months in those with higher Mg levels. Similarly, disease-free survival was 9.40 (3.39-15.41) months in patients with low magnesium levels and 31.38 (24.53-38.22) months in those with higher magnesium levels. Patients with magnesium levels below the threshold had significantly shorter overall and disease-free survival times ($p=0.001$ and $p=0.005$, respectively). In contrast, in the DLBCL subgroup, no statistically significant difference was observed between magnesium levels and survival times ($p=0.188$).

Discussion

EBV is strongly implicated in the pathogenesis of HL and certain NHL subtypes. EBV infection of lymphoid cells increases susceptibility to malignant transformation, thereby promoting uncontrolled proliferation and lymphomagenesis [16]. Although numerous studies have investigated the role of magnesium in immune function and malignancy, data specifically addressing the relationship between serum magnesium levels and EBV positivity in lymphoma patients remain limited. Therefore, our study aimed to evaluate this relationship and to assess the prognostic impact of magnesium on survival outcomes.

The relationship between Mg and EBV may be explained through immune dysregulation. Mg plays a critical role in cytotoxic T-cell and NK cell function, which are essential for controlling EBV-infected cells. Mg deficiency may impair cytotoxic activity, resulting in inadequate viral clearance and persistent EBV infection. This impaired immune response may contribute to lymphomagenesis. These findings support our hypothesis that magnesium status may influence EBV-related disease processes [10,12].

In the present study, serum magnesium levels were lower in EBV-positive patients compared to EBV-negative patients. Furthermore, patients with magnesium levels >1.68 mg/dL had longer overall and disease-free survival compared to those with magnesium levels ≤1.68 mg/dL. In addition, low magnesium levels were associated with advanced-stage and progressive disease. Tissue-based EBER positivity and peripheral blood EBV DNA PCR represent different biological aspects of EBV infection. While EBER positivity reflects EBV involvement within tumor tissue, PCR positivity indicates circulating viral DNA. Therefore, the observed association

between magnesium levels and EBV status should be interpreted with consideration of these methodological differences. In our study, the association between magnesium levels and EBV status was observed for tissue-based EBER positivity, whereas no significant association was found for peripheral blood EBV DNA PCR positivity.

The biological basis of this relationship may be further explained by magnesium-related immune dysfunction. Magnesium is known to regulate hematopoietic and immune cell function. In XMEN disease, MAGT1 deficiency disrupts magnesium homeostasis and N-linked glycosylation of immune-related proteins, contributing to impaired immune responses, particularly against viral infections such as EBV [11]. NK and CD8+ T lymphocytes play a central role in limiting EBV dissemination, and magnesium deficiency may compromise their cytolytic activity.

Chaigne-Delalande et al. [10] demonstrated that magnesium deficiency impairs NKG2D-mediated cytotoxic function in CD8+ T cells and NK cells, leading to inadequate control of EBV infection. Importantly, restoration of magnesium levels improved cytotoxic function *in vitro*. Similarly, Ravell et al. [12] reported significantly lower plasma magnesium levels in patients with EBV-positive BL and showed an inverse relationship between magnesium levels and EBV viral load. These findings are consistent with our results, which showed lower magnesium levels in EBV-positive patients.

The impact of magnesium on survival has also been reported in previous studies. Gile et al. [17] demonstrated that hypomagnesemia was associated with poorer survival outcomes in DLBCL patients. Similarly, in BL, low magnesium levels were associated with shorter overall survival [18]. In our study, the relationship between magnesium levels and survival differed according to both the lymphoma subtype and the type of survival analysis performed. In the DLBCL subgroup, low magnesium levels were associated with poorer survival status in the categorical analysis (alive vs. deceased). However, this association was not statistically significant in Kaplan-Meier survival time analysis. In contrast, in the HL subgroup, no significant association was observed in the categorical survival status analysis, whereas Kaplan-Meier analysis demonstrated significantly shorter overall and disease-free survival in patients with low magnesium levels. These differences may

reflect the methodological distinctions between categorical outcome comparisons and time-to-event analyses, as well as the limited sample size in subgroup analyses.

In addition, low magnesium levels were associated with advanced disease stage in our cohort. This finding is consistent with previous studies demonstrating a relationship between hypomagnesemia and higher tumor burden or worse prognostic indices, such as the International Prognostic Index [19]. Although a ROC-derived cut-off value of 1.68 mg/dL was used in the present analyses, the ROC analysis itself did not reach statistical significance. Therefore, this threshold should be regarded as exploratory and hypothesis-generating rather than a validated prognostic cut-off.

We acknowledge that evaluating magnesium levels alone may not fully explain survival outcomes, and additional prognostic factors should be considered. Furthermore, EBV status was not available in all patients and was assessed using different methods, which may introduce heterogeneity. The retrospective design and relatively small sample size also represent important limitations of this study.

Study Limitations

The current study has several limitations that should be acknowledged. First, the retrospective single-center design and the relatively small sample size may limit the generalizability of the findings. Second, EBV status was assessed using different methodologies, including tissue-based EBER ISH and peripheral blood EBV DNA PCR, which may have introduced methodological heterogeneity. Third, only eight patients had magnesium levels ≤ 1.68 mg/dL, limiting the statistical power and reliability of subgroup analyses. Furthermore, the ROC-derived magnesium threshold was based on an analysis that did not reach statistical significance and should therefore be interpreted cautiously. In addition, multivariable survival analyses could not be performed because of the limited number of death events, preventing assessment of whether serum magnesium is an independent prognostic factor. The study design did not include serial magnesium measurements, and serum magnesium levels were evaluated at a single time point. Data regarding renal function, previous magnesium replacement, and dietary magnesium intake were also unavailable. Therefore, the findings should be considered exploratory and hypothesis-generating and require confirmation in larger prospective studies.

Conclusion

Existing literature indicates that magnesium deficiency increases vulnerability to both malignancies and viral infections. This study contributes to the field by assessing the association between magnesium status and EBV positivity in lymphoma patients, as well as the relationship between magnesium levels and overall survival and disease-free survival. Our findings suggest a potential association between serum magnesium levels and clinical outcomes in lymphoma patients; however, these findings should be considered

exploratory. Larger prospective studies are required to determine whether magnesium has independent prognostic value. Future prospective interventional studies are needed to determine whether correction of magnesium deficiency may influence clinical outcomes in lymphoma patients.

Ethics

Ethics Committee Approval: Ethical approval for this study was obtained from the Ethics Committee of Atatürk University Faculty of Medicine (number: B.30.2.ATA.0.01.00/35, date: 29.12.2022).

Informed Consent: Informed consent was not obtained due to the retrospective nature of the study.

Author Contributions

Medical Practices: F.E., M.B.D., Concept: F.E., M.B.D., Design: F.E., Data Collection or Processing: M.B.D., Analysis or Interpretation: M.B.D., G.S., Literature Search: M.B.D., G.S., Writing: M.B.D., G.S., F.E.

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