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Serum Magnesium Level and the Extent of Clinical and Laboratory Presentations in Systemic Lupus Erythematosus. Is There Any Association? An Analytical Cross-Sectional Study

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Abstract

Background and Objectives: Recent studies have reported varying degrees of associations between the alterations of serum elements levels with the onset and severity of various autoimmune diseases. Therefore, we aimed to investigate the effects of changes in magnesium levels, which are either the primary or the essential cofactor in many metabolic and biochemical reactions, on the development of SLE.

Methods: This analytical cross-sectional study evaluated individuals suffering from SLE, visited between March 2021 and March 2022. Demographic data, clinical presentation, and laboratory indices related to SLE were documented in those meeting the eligibility criteria. Then, they were divided into different groups based on age, sex, and serum magnesium level, and their differences were analyzed.

Results: One hundred patients with SLE (90% women and 10% men) with a mean age of 41.7 years had (87%) normomagnesemia, 11% had hypomagnesemia (which were 100% women), and 2% had hypermagnesemia. serum magnesium was significantly lower in those with leukopenia than in those without leukopenia ($P < 0.01$). In addition, the mean serum magnesium levels were significantly different between patients based on their SLEDAI-2k classifications ($P < 0.01$) We found that as the disease activity increased, the serum magnesium level also increased, yet the latter never surpassed the normal upper limit.

Conclusion: Among the clinical manifestations of lupus, only serum magnesium was significantly lower in those with leukopenia than in those without leukopenia. Therefore, measuring magnesium in lupus patients with leukopenia should be considered. Of course, it should be noted that the small sample size affects the generalizability of the results, and a study with a larger sample size and a control group is recommended.

Keywords: Systemic Lupus Erythematosus, Autoimmune disorders, Trace Elements, Magnesium

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1. Introduction

Systemic Lupus Erythematosus (SLE) is a chronic progressive autoimmune disorder with various clinical and laboratory findings (1).SLE can involve several body organs, leading to significant morbidity and mortality in those suffering (e.g., the 5-year and 15-20-year survival rates are about 90 and 80%, respectively)

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↑*Question* *Is there a relationship between serum magnesium level and clinical and laboratory manifestations in systemic lupus erythematosus?*

Findings *We did not find a relationship between SLE score and disease activity with hypomagnesemia. Among the clinical manifestations of lupus, only serum magnesium was significantly lower in those with leukopenia than in those without leukopenia.*

→*Meaning* *Measuring magnesium in lupus patients with leukopenia should be considered.*

(1-3). Therefore, investigating the factors with potentially high impact on the disorder's frequency and severity of presentations is essential, one of which currently of high interest being the trace elements.

The magnitude of the effects of trace elements, including copper, zinc, manganese, iron, and magnesium, constituting about 0.01% of the total body weight, has been emphasized in many physiological and vital processes of the body (4-6). For instance, recent studies have revealed a significant relationship between the alterations of these elements' serum levels with the onset and severity of various autoimmune diseases such as Rheumatoid Arthritis (7-9). Furthermore, considering the significant effects of magnesium ions — which are divalent cations— as either the primary or the essential cofactor in many metabolic and biochemical reactions, including cellular respiration, glycolysis, membrane transportation, and DNA replication, the effects of changes in magnesium levels in the development of SLE should be sought with great attention to detail (10-12).

Unfortunately, studies on the relationship between serum magnesium levels and SLE have been scarce and have yielded contradictory findings (13-15). One study found lower serum magnesium levels in SLE patients, but these levels did not correlate with higher inflammatory parameters (13). Another study also reported lower serum magnesium levels in SLE patients, but viewed them as a presentation of the disease rather than a factor in its development (14). Additionally, fractional magnesium excretion differed significantly between SLE patients and those without the disorder in one study, but other factors like creatinine clearance, urine protein levels, and blood pressure did not show significant differences (16). Another study found that exogenous magnesium decreased activated partial thromboplastin time in SLE patients with high levels of lupus anticoagulants, while it increased in healthy controls (17). Furthermore, one study reported higher nutritional magnesium intake in SLE patients compared to healthy individuals, but another study found no difference in serum magnesium levels between the SLE and control groups (15, 18).

With all things mentioned in mind and amid the vast heterogeneity of findings, we aimed to determine serum magnesium levels in those with SLE and its association with the frequency and severity of the disease presentations.

2. Materials and Methods

2.1. Study Design

The current study's design was analytical cross-sectional and ethically approved by the local institutional review board (IRB) while also strictly abiding by the guidelines of the World Medical Association (WMA) provided in the declaration of Helsinki. Moreover, all the included individuals were provided with details of the conduction method of the study, and written informed consent was then obtained.

2.2. Participants and eligibility criteria

The population of interest was those suffering from SLE, visited between March 2021 and March 2022 in our region's Rheumatology referral center.

The inclusion criteria comprised having a confirmed diagnosis of SLE based on the Systemic Lupus International Collaborating Clinics (SLICC) criteria (i.e., either meeting a minimum 4 out of the 17 defined criteria, at least one of each being clinical and another immunologic or having a biopsy confirming lupus nephritis along with a higher than cut-off anti-nuclear titer (1/80) (19).

In addition, the exclusion criteria then included having any ongoing malignancy, infection, chronic kidney disease (stages three or above based on the Kidney Disease Outcomes Quality Initiative (KDOQI) 2002 (20), having an estimated Glomerular Filtration Rate (eGFR) < 30 mL/min/1.73m^2), or having hepatic failure and consumed alcohol. Finally, those meeting the eligibility criteria were selected for the next steps.

2.3. Data collection

The demographic data (i.e., age and sex), the clinical presentation, and the indices' values of obtained laboratory tests, brought below, were sought and documented:

- Presence or absence of:
  - General manifestations, including fever and chills
  - Neuropsychiatric manifestations (e.g., lupus headache, cranial nerves or visual disturbances, cerebrovascular accidents, seizure, and psychosis) or organic brain syndrome (i.e., cerebral dysfunction not due to either infection or metabolic causes, including the altered level of consciousness or mental status, and behavioral changes)
  - Musculoskeletal manifestations (e.g., myositis or arthritis)
  - Renal manifestations (e.g., hematuria, pyuria, or urinary casts)
  - Mucoso-cutaneous manifestations (e.g., rash, alopecia, and mucosal ulcers)
  - Lung involvement (e.g., pleurisy, lupus pneumonitis, pulmonary hypertension)
  - Cardiovascular manifestations (e.g, pericarditis, myocarditis, valvular involvement)
  - Abnormal laboratory indices and hematological manifestations (e.g., thrombocytopenia and leukopenia)
  - Immunologic manifestations (e.g., vasculitis, low serum Complements such as C3, C4, FANA, and increased anti-dsDNA)
  - Total SLE score and disease activity based on Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2k) scoring system: 0 = no activity, 1-5 = mild activity, 6-10 = Moderate activity, 11-19 = High activity, and >20 = Very High activity (21).

In addition, a morning 5-milliliter blood sample was obtained from each patient with minimal physical stress and sent to the lab for testing. After the centrifugation, the serum magnesium level was determined via colorimetric spectrophotometry (Mindray Bs800, CTA-2000, Chem-Tech + Kit from BioLab with Lot = 12501). Moreover, the resulting value was documented after comparing it with the given lab normal reference range (the range was 1.9-2.٩ mg/dl). The patients were categorized into three groups based on their serum magnesium levels: 1) low

magnesium group (serum magnesium less than 1.9 mg/dL), 2) normal group (serum magnesium between 1.9 and 2.9 mg/dL), and 3) high magnesium group (serum magnesium above 2.9 mg/dL).

2.4. Sample size determination

Due to the short time frame in which the study was carried out and the small number of available patients with SLE, all those meeting the inclusion and exclusion criteria were included.

2.5. Statistical analysis

We used the 26th version of the SPSS software package (IBM, USA) to analyze the data. Furthermore, mean ± standard deviation, median and Interquartile range (IQR), and frequency and ratio (in percent) were used to describe the distribution of normally distributed quantitative variables, not normally distributed quantitative variables, and qualitative variables, respectively. Moreover, due to the assumptions of regression analysis not being met (presence of significant outliers, not normally distributed variables, and the absence of homogeneity of variance), Mann-Whitney U and Kruskal Wallis H tests were used to analyze the differences of quantitative variables. Moreover, Pearson Chi-Squared (with Cramer's V as the post hoc analysis) and Kendall's tau B tests were used for analyzing the differences in qualitative variables. Moreover, the confidence level was 95%, and $P < 0.05$ were considered statistically significant.

3. Results

One hundred patients with SLE (90% women and 10% men) with a mean age of 41.7 years had their serum magnesium level and SLE severity evaluated. Most included cases (87%) had serum magnesium levels within the normal range, but 11% had hypomagnesemia) which were 100% women), and 2% had hypermagnesemia. Furthermore, most patients did not experience any adversities or complications related to SLE. Howbeit, among those with adversities, 35% had increased Anti-dsDNA more than 2 times normal reference, 23% had low serum Complements, and 23% had arthritis, which were the most frequent manifestations, respectively.

Table 1 shows the differences in variables were analyzed after dividing the patients into two groups based on their sex. women were significantly older ($P < 0.05$), and their manifestations related to vasculitis were more frequent in men ($P < 0.05$) (Table 1).

The frequency of seizures ($P = 1$), psychosis ($P = 0.74$), organic brain syndrome ($P = 0.74$), visual disturbances ($P= 0.74$), cranial nerves dysfunction ($P = 0.17$), lupus headache ($P = 0.44$), cerebrovascular accidents (p-value = 1), arthritis ($P = 0.58$), myositis ($P = 1$), urinary casts ($P = 1$), hematuria ($P = 1$), proteinuria ($P = 0.36$), pyuria ($P = 1$), rash ($P = 0.06$), alopecia ($P = 0.27$), mucosal ulcers ($P = 0.57$), pleurisy ($P = 0.63$), pericarditis ($P = 0.58$), low serum Complements levels ($P = 0.58$), fever ($P = 0.74$), thrombocytopenia $P = 0.4$), and leukopenia ($P = 0.29$) were not significant.

Table 2 shows the severity of SLE manifestations was also evaluated based on participants' sex and qualitative serum magnesium levels, which indicated that the frequency of vasculitis-related manifestations ($P < 0.05$) and mucosal ulcers ($P < 0.05$) were significantly different due to the higher incidence of the mentioned manifestations in individuals with normomagnesemia ($P < 0.05$). However, the frequency of seizure ($P = 1$), psychosis ($P = 0.93$), organic brain syndrome ($P = 0.93$), visual disturbances ($P = 0.93$), cranial nerves dysfunction ($P = 0.45$), lupus headache ($P = 0.68$), cerebrovascular accidents ($P = 1$), arthritis ($P = 0.67$), myositis ($P = 1$), urinary casts ($P = 1$), hematuria ($P = 1$), proteinuria ($P = 0.57$), pyuria ($P = 1$), rash ($P = 0.24$), alopecia ($P = 0.44$), pleurisy ($P = 0.86$), pericarditis ($p = 0.86$), low serum Complements ($P = 0.7$), increased anti-dsDNA ($P = 0.25$), fever ($P = 0.93$), thrombocytopenia ($P = 0.85$), and leukopenia ($P = 0.5$) were not

Table 1. Included individuals' characteristics

Variable	Total	Men	Women	P*
Age	41.7 ± 13.28	32.4 ± 6.85	42.74 ± 13.44	<0.05
Mean Serum magnesium	2.18 ± 0.32	2.29 ± 0.26	2.17 ± 0.32	0.186
Serum magnesium				
Low	11 (11%)	0 (0%)	11 (12.22%)	
Normal	87 (87%)	9 (90%)	78 (86.67%)	0.078
High	2 (2%)	1 (10%)	1 (1.11%)	
Lupus score	2[0-7.5]	4[0-7.5]	2[0-8]	0.45
SLEDAI-2k				
No activity	39 (39%)	3 (30%)	36 (40%)	0.42
Low activity	27 (27%)	3 (30%)	24 (26.7%)	
Moderate activity	19 (19%)	2 (20%)	17 (18.9%)	
High activity	13 (13%)	1 (10%)	12 (13.3%)	
Very High activity	2 (2%)	1 (10%)	1 (1.1%)	
Increased ds-DNA				
Negative	65 (65%)	7 (70%)	58 (64.44%)	0.73
Positive	35 (35%)	3 (30%)	32 (35.56%)	
Vasculitis				
Negative	95 (95%)	*8 (80%)	87 (96.67%)	<0.05
Positive	5 (5%)	*2 (20%)	3 (3.33%)	

SLEDAI-2k, Systemic lupus erythematosus disease activity index 2000.
Data are N (%) or Mean± Standard deviation or Medians (interquartile ranges)
P-value <0.05 (Student's t-test, Mann-Whitney U test, Kruskal Wallis test, or Chi-square)

Table 2. Distribution of data based on sex and qualitative serum magnesium levels

Variable						P*
Sex	Men	Men	Men	Women	Women	
Serum magnesium	Low	Normal	High	Normal	High	
Age	33 ± 6.98	27	40.73 ± 11.09	42.9 ± 13.81	53	0.16
Mean Serum magnesium(mg/dL)	2.23 ± 0.16	2.9	1.77 ± 0.16	2.21 ± 0.26	3.6	
SLE score	4 [0-6]	12	1 [0-5]	2 [0-8]	0	0.81
SLEDAI-2k						
No activity	3 (33.3%)	0 (0%)	4 (36.4%)	31 (39.7%)	1 (100%)	0.68
Low activity	3 (33.3%)	0 (0%)	5 (45.5%)	19 (24.4%)	0 (0%)	
Moderate activity	2 (22.2%)	1 (100%)	1 (9.1%)	16 (20.5%)	0 (0%)	
High activity	0 (0%)	0 (0%)	1 (9.1%)	11 (14.1%)	0 (0%)	
Very High activity	1 (11.1%)	0 (0%)	0 (0%)	1 (1.3%)	0 (0%)	
Increased ds-DNA						
Negative	6 (66.67%)	1 (100%)	9 (81.82%)	48 (61.54%)	1 (100%)	0.25
Positive	3 (33.33%)	0 (0%)	2 (18.18%)	30 (38.46%)	0 (0%)	
Vasculitis						
Negative	8 (88.89%)	1 (100%)	11 (100%)	75 (96.15%)	75 (96.15%)	<0.05
Positive	1 (11.11%)	0 (0%)	0 (0%)	3 (3.85%)	0 (0%)	
Mucosal ulcers						
Negative	9 (100%)	0 (0%)	11 (100%)	73 (93.59%)	1 (100%)	<0.05
Positive	0 (0%)	1 (100%)	0 (0%)	5 (6.41%)	0 (0%)	

SLEDAI-2k, Systemic lupus erythematosus disease activity index 2000.
Data are N (%) or Mean± Standard deviation or Medians (interquartile ranges)
* P-value <0.05 (Kruskal Wallis or Chi-square test)

statistically significant.

Finally, we compared the mean levels of magnesium based on the severity (SLEDAI-2k) and manifestations of SLE, indicating that the differences between the mean magnesium levels were statistically significant ($P < 0.05$) based on the presence or absence of vasculitis, proteinuria, rash, alopecia, mucosal ulcers, and increased Anti-dsDNA. This finding means that having any of the mentioned symptoms was associated with higher serum magnesium(But in normal range). Furthermore, serum magnesium was significantly lower in those with leukopenia than in those without ($P < 0.01$). In addition, the mean serum magnesium levels were significantly different between patients based on their SLEDAI-2k classifications ($P < 0.01$) due to the significance of the difference between the group with no disease activity and the moderate and severe activity groups ($P < 0.05$) along significance of the difference between the group with mild disease activity and those with moderate and severe activity ($P < 0.01$). We found that as the disease activity increased, so did the serum magnesium level, yet the latter never surpassed the normal upper limit (Table 3).

4. Discussion

SLE, known as a systemic autoimmune disease, involves various immune cells and inflammatory mediators that contribute to its pathogenesis, particularly dysfunctional T and B cells. Factors such as hormonal, trace elements, environmental influences, and genetic predispositions are associated with the breakdown of B- or T-cell tolerance to self-antigens, leading to the activation of both the innate and adaptive immune responses (22). Magnesium plays a crucial role in the immune system; research has demonstrated that intracellular free magnesium ions act as essential second messengers in the immune activation of T and B lymphocytes, while magnesium channels and transport proteins are vital for normal immune function (23, 24). Additionally, magnesium is implicated in cellular oxidative stress and inflammatory processes (25).

In this study, a cross-sectional analysis was applied to the SLEDAI-2k classifications to assess the association between SLE disease Activity and serum magnesium level. Out of 100 patients with lupus that we examined, only 11% had hypomagnesemia, all of them were women, and most patients had low or no disease activity). We found that as the disease activity increased, so did the serum magnesium level, yet the latter never surpassed the normal upper limit.

The prevalence of hypomagnesemia, normomagnesemia, and hypermagnesemia in new-onset SLE patients in Yang et al. study (2023) was similar our study (26). Meza-Meza et al. reported a higher magnesium consumption in SLE patients (18), which may explain the high percentage of normomagnesemia in our study and Yang et al.

Yilmaz et al. did not find a relationship between the SLEDAI score and magnesium serum level and this was while serum magnesium levels were low in lupus patients (14). In our study, most patients with hypomagnesemia were in the no or low disease activity group. The mean serum magnesium levels were significantly different between patients based on their SLEDAI-2k classifications ($P < 0.01$) due to the significance of the difference between the group with no disease activity and the moderate and severe activity groups ($P < 0.05$) along significance of the difference between the group with mild disease activity and those with moderate and severe activity ($P < 0.01$).

The detection of intracellular magnesium ions is difficult, while the detection of serum magnesium ions is simple and convenient. Clinicians also use serum or plasma magnesium to evaluate patients. so, we used serum magnesium to assess. In one study, lower serum magnesium the level had a positive association with pyuria and a negative one with hematuria, it did not correlate with higher values of inflammatory parameters (13). In our study CKD patients with stage 3 and above were part of the ex-

Table 3. Mean serum magnesium based on SLE manifestations

Variable		Mean Serum magnesium (mg/dL)	P
Seizure			
	Negative	2.19 ± 0.32	1
	Positive	-	
Psychosis			
	Negative	2.18 ± 0.32	0.30
	Positive	2.43	
Organic brain syndrome			
	Negative	2.18 ± 0.32	0.18
	Positive	2.52	
Visual disturbances			
	Negative	2.18 ± 0.32	0.86
	Positive	2.2	
Cranial nerves dysfunction			
	Negative	2.19 ± 0.31	0.71
	Positive	1.97 ± 0.59	
Lupus headache			
	Negative	2.17 ± 0.32	0.07
	Positive	2.4 ± 0.3	
Cerebrovascular accidents			
	Negative	2.19 ± 0.32	1
	Positive	-	
Vasculitis			
	Negative	2.17 ± 0.32	0.04
	Positive	2.46 ± 0.32	
Arthritis			
	Negative	2.17 ± 0.34	0.18
	Positive	2.23 ± 0.24	
Myositis			
	Negative	2.19 ± 0.32	1
	Positive	-	
Urinary casts			
	Negative	2.19 ± 0.32	1
	Positive	-	
Hematuria			
	Negative	2.19 ± 0.32	1
	Positive	-	
Proteinuria			
	Negative	2.17 ± 0.32	0.01
	Positive	2.44 ± 0.18	
Pyuria			
	Negative	2.19 ± 0.32	1
	Positive	-	
Rash			
	Negative	2.16 ± 0.32	0.03
	Positive	2.36 ± 0.3	
Alopecia			
	Negative	2.17 ± 0.33	0.04
	Positive	2.31 ± 0.17	
Mucosal ulcers			
	Negative	2.17 ± 0.31	0.04
	Positive	2.43 ± 0.33	
Pleurisy			
	Negative	2.18 ± 0.32	0.05
	Positive	2.55 ± 0.04	
Pericarditis			
	Negative	2.18 ± 0.32	0.26
	Positive	2.44 ± 0.39	
Low serum Complement levels			
	Negative	2.17 ± 0.31	0.15
	Positive	2.25 ± 0.37	
Increased Anti-dsDNA			
	Negative	2.14 ± 0.31	0.02
	Positive	2.26 ± 0.34	
Fever			
	Negative	2.19 ± 0.32	0.99
	Positive	2.12	
Thrombocytopenia			
	Negative	2.18 ± 0.32	0.79
	Positive	2.19 ± 0.39	
Leukopenia			
	Negative	2.21 ± 0.32	<0.01
	Positive	1.96 ± 0.21	
SLEDAI-2k			
	No activity	2.14 ± 0.32	<0.01
	Low activity	2.07 ± 0.26	
	Moderate activity	2.32 ± 0.3	
	High activity	2.3 ± 0.39	
	Very High activity	2.46 ± 0.08	

SLEDAI-2k, Systemic lupus erythematosus disease activity index 2000
p-value <0.05 (Student's t-test or Mann Whitney-U test)

clusion criteria due to the effect of renal failure on magnesium excretion and therefore severe renal manifestations were less in our lupus patients than in other studies.

In our study, leukopenia had an association with lower serum magnesium. Studies showed that extra- and intracellular Mg2+ concentrations are important for the activation and proliferation of neutrophils and lymphocytes, and that Mg2+ deficiency may affect the function of these cells in peripheral blood, decreasing immunoglobulin production and the number of cells that produce antibodies (27).

This study has several limitations. First, the cross-sectional design and the absence of a healthy control group as well as the small sample size prevented us from drawing conclusions about the causality of the findings.

Second, we lacked information on food and dietary habits that determine magnesium intake in patients. Third, the lack of information on drug therapy prescribed for those with normal magnesium precludes us from making comparisons when analyzing the role of proton pump inhibitors and/or diuretics on magnesium levels. Finally, the patient population in this study is of the same race. However, one of the strengths of this study, in our view, is its relatively large number of appropriately matched samples, investigating the effects on mild SLE cases along with the effects of their age and sex while also evaluating the effects of high, low, and normal levels of magnesium on SLE manifestations.

5. Conclusion

Although in recent years some studies have reported of hypomagnesemia in SLE patients, Nevertheless, magnesium is still a neglected ion in patients. We did not find a relationship between SLE score and disease activity with hypomagnesemia. Among the clinical manifestations of lupus, only serum magnesium was significantly lower in those with leukopenia than in those without leukopenia. Therefore, measuring magnesium in lupus patients with leukopenia should be considered. Of course, it should be noted that the small sample size and the absence of a healthy control group affects the generalizability of the results, and a study with a larger sample size and a control group is recommended.

Ethical Statement

The study's protocol for investigation, data collection, analysis, and reporting was approved by the Kerman University of Medical Sciences with the approval code "IR.KMU.AH.REC.1400.089"xx.

Authors' Contributions

Conception and design of the study; Najmeh Shamspour, Marjan Abasiniya, Tarlan Hassan Aghaei; Generation,

collection, assembly, analysis and/or interpretation of data; Najmeh Shamspour, Marjan Abasiniya, Tarlan Hassan Aghaei, Seyed Mohamad Hosseinian; Drafting and/or revision of the manuscript; Najmeh Shamspour, Marjan Abasiniya; and approval of the final version of the manuscript. Najmeh Shamspour, Tarlan Hassan Aghaei.

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Conflict of Interests

The authors declare that they have no competing interests.

References

1. Wallace DJ, Hahn B. Dubois' lupus erythematosus: Lippincott Williams & Wilkins; 2007.
2. Maidhof W, Hilas O. Lupus: an overview of the disease and management options. P T. 2012;37(4):240.
3. González LA, Toloza SM, Alarcón GS. Impact of race and ethnicity in the course and outcome of systemic lupus erythematosus. Rheum Dis Clin North Am. 2014;40(3):433-54.
4. Vural H, Uzun K, Uz E, Koçyigit A, Cigli A, Akyol Ö. Concentrations of copper, zinc and various elements in serum of patients with bronchial asthma. J Trace Elem Med Biol. 2000;14(2):88-91.
5. Durak I, Kavutcu M, Canbolat O, Işik A, Akyol Ö. Concentrations of some major and minor elements in larynx tissues with and without cancer. BioMetals. 1994;7:45-8.
6. Akyol Ö, Kavutçu M, Canbolat O, Işik B, Durak I. A study on the relationships between some of the trace element levels of hair, nail, serum and urine in healthy subjects. Turk J Med Res. 1993;11(2):75-81.
7. Silbergeld EK, Silva IA, Nyland JF. Mercury and autoimmunity: implications for occupational and environmental health. Toxicol Appl Pharmacol. 2005;207(2):282-92.
8. Tuncer S, Kamanli A, AkÇil E, Kavas Gö, SeÇkin B, Atay MB. Trace element and magnesium levels and superoxide dismutase activity in rheumatoid arthritis. Biol Trace Elem Res. 1999;68:137-42.
9. Honkanen V, Pelkonen P, Mussalo-Rauhamaa H, Lehto J, Westermarck T. Serum trace elements in juvenile chronic arthritis. Clin Rheumatol. 1989;8:64-70.
10. De Baaij JH, Hoenderop JG, Bindels RJ. Magnesium in man: implications for health and disease. Physiol Rev. 2015.
11. Kohli G, Bhargava A, Goel H, Yadav S, Saini A, Singh G, et al. Serum magnesium levels in patients with head and neck cancer. Magnesium. 1989;8(2):77-86.
12. Phd TJRM. Magnesium Deficiency in Systemic Lupus Erythematosus. J Nutr Environ Med. 1997;7(2):107-12.
13. Cunha L, Marcelino G, Amaral M, Alves JD. Magnesium–association with inflammation and renal disease in systemic lupus erythematosus. Portuguese Journal of Nephrology and Hypertension. 2015;29:323-31.
14. Yilmaz A, Sari RA, Gundogdu M, Kose N, Dag E. Trace elements and some extracellular antioxidant proteins levels in serum of patients with systemic lupus erythematosus. Clin Rheumatol. 2005;24(4):331-5.
15. Tóth CN, Baranyai E, Csípő I, Tarr T, Zeher M, Posta J, et al. Elemental Analysis of Whole and Protein Separated Blood Serum of Patients with Systemic Lupus Erythematosus and Sjögren's Syndrome. Biol Trace Elem Res. 2017;179(1):14-22.
16. Deekajorndech T. Fractional excretion magnesium (FE Mg) in systemic lupus erythematosus. J Med Assoc Thai. 2005;88(6):743-5.
17. Tokutake T, Baba H, Shimada Y, Takeda W, Sato K, Hiroshima Y, et al. Exogenous Magnesium Chloride Reduces the Activated Partial Thromboplastin Times of Lupus Anticoagulant-Positive Patients. PLoS One. 2016;11(6):e0157835.
18. Meza-Meza MR, Vizmanos-Lamotte B, Muñoz-Valle JF, Parra-Rojas I, Garaulet M, Campos-López B, et al. Relationship of excess weight with clinical activity and dietary intake deficiencies in systemic lupus erythematosus patients. Nutrients. 2019;11(11):2683.
19. Petri M, Orbai AM, Alarcón GS, Gordon C, Merrill JT, Fortin PR, et al. Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. Arthritis Rheum. 2012;64(8):2677-86.
20. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis. 2002;39(2 Suppl 1):S1-266.
21. Touma Z, Urowitz MB, Taghavi-Zadeh S, Ibañez D, Gladman DD. Systemic lupus erythematosus disease activity Index 2000 Responder Index 50: sensitivity to response at 6 and 12 months. Rheumatology (Oxford). 2012;51(10):1814-9.
22. Herrada AA, Escobedo N, Iruretagoyena M, Valenzuela RA, Burgos PI, Cuitino L, Llanos C. Innate Immune Cells' Contribution to Systemic Lupus Erythematosus. Front Immunol. 2019 Apr 15;10:772.
23. Nielsen FH. Magnesium deficiency and increased inflammation: current perspectives. J Inflamm Res. 2018 Jan 18;11:25-34.
24. Mazur A, Maier JA, Rock E, Gueux E, Nowacki W, Rayssiguier Y. Magnesium and the inflammatory response: potential physiopathological implications. Arch Biochem Biophys. 2007 Feb 1;458(1):48-56.
25. Wang K, Wei H, Zhang W, Li Z, Ding L, Yu T, Tan L, Liu Y, Liu T, Wang H, Fan Y, Zhang P, Shan Z, Zhu M. Severely low serum magnesium is associated with increased risks of positive anti-thyroglobulin antibody and hypothyroidism: A cross-sectional study. Sci Rep. 2018 Jul 2;8(1):9904.
26. Yang W, Lian X, Chen H. The association of serum magnesium with infection in new-onset systemic lupus erythematosus patients. Lupus. 2023 Mar;32(3):380-387.
27. Lima FDS, Fock RA. A Review of the Action of Magnesium on Several Processes Involved in the Modulation of Hematopoiesis. Int J Mol Sci. 2020 Sep 25;21(19):7084.