Uludağ Üniversitesi Tıp Fakültesi Dergisi
Journal of Uludağ University Medical Faculty
52, 1813892, 2026
DOI: https://doi.org/10.32708/uutfd.1813892

ORIGINAL RESEARCH

Interictal Serum Uric Acid, Albumin, and Uric Acid-to-Albumin Ratio in Episodic Migraine: A Case–Control Study and Association with Disease Burden

Deniz KAMACI ŞENER, Özden KAMIŞLI

Department of Neurology, Bursa City Hospital, Bursa, Türkiye.

ABSTRACT

Oxidative stress and systemic inflammation are increasingly recognized as key mechanisms in migraine pathophysiology. Uric acid (UA) and albumin (ALB) contribute to redox and inflammatory balance, and their ratio (UAR) has been proposed as a combined biomarker of these processes. This study evaluated serum UA, ALB, and UAR levels in patients with episodic migraine during the interictal period and examined their associations with clinical features and migraine-related disability. In this retrospective case–control study, 120 patients with episodic migraine and 100 age- and sex-matched healthy controls were included. Serum UA and ALB were measured, and UAR was calculated as UA (mg/dL)/ALB (g/dL). Demographic and laboratory variables were compared between groups. In the migraine group, correlations between biochemical markers and clinical variables, including pain intensity (VAS), migraine-related disability (MIDAS), attack frequency, and aura status, were analyzed. Serum UA and UAR levels were significantly lower in migraine patients than in controls (both $p<0.001$), whereas ALB levels were similar. UA, ALB, and UAR were not correlated with attack frequency, pain intensity, MIDAS scores, or aura status. Lower UA and UAR levels in episodic migraine may reflect reduced systemic antioxidant capacity. However, the absence of associations with clinical severity suggests that oxidative imbalance may not directly represent migraine burden. UAR may serve as a biochemical indicator of oxidative and inflammatory processes in migraine. Further prospective studies are needed to clarify its clinical relevance.

Keywords: Migraine. Uric acid. Albumin. Uric acid-to-albumin ratio. Oxidative stress.

Episodik Migren Hastalarında İnteriktal Serum Ürik Asit, Albümin ve Ürik Asit/Albümin Oranı: Olgu–Kontrol Çalışması ve Hastalık Yükü ile İlişkisi

ÖZET

Oksidatif stres ve sistemik inflamasyonun migren patofizyolojisinde önemli rol oynadığı giderek daha fazla kabul edilmektedir. Ürik asit (UA) ve albümin (ALB), redoks ve inflamatuvar dengeye katkıda bulunan temel bileşenlerdir. Bu iki parametrenin oranı olan ürik asit/albümin oranı (UAR), bu süreçleri birlikte yansıtan potansiyel bir biyobelirteç olarak önerilmektedir. Bu çalışmada epizodik migren hastalarında interiktal dönemde serum UA, ALB ve UAR düzeyleri değerlendirildi ve bu parametrelerin klinik özellikler ile migrenle ilişkili özürlülükle ilişkisi araştırıldı. Retrospektif olgu–kontrol tasarımındaki çalışmaya 120 epizodik migren hastası ve yaş ve cinsiyet açısından benzer 100 sağlıklı kontrol dahil edildi. Serum UA ve ALB düzeyleri ölçüldü ve UAR, UA (mg/dL) / ALB (g/dL) formülüyle hesaplandı. Gruplar arasında demografik ve laboratuvar verileri karşılaştırıldı. Migren grubunda biyokimyasal belirteçler ile ağrı şiddeti (VAS), MIDAS puanı, atak sıklığı ve aura varlığı arasındaki ilişkiler analiz edildi. Migren hastalarında serum UA ve UAR düzeyleri kontrollere göre anlamlı derecede düşük bulundu (her ikisi için $p<0,001$), ALB düzeyleri ise benzerdi. UA, ALB ve UAR düzeyleri ile atak sıklığı, ağrı şiddeti, MIDAS puanı veya aura varlığı arasında anlamlı ilişki saptanmadı. Epizodik migrende düşük UA ve UAR düzeyleri azalmış sistemik antioksidan kapasiteyi yansıtabilir. Ancak klinik şiddet ile ilişki saptanmaması, oksidatif dengesizliğin hastalık yükünü doğrudan yansıtmayabileceğini düşündürmektedir. UAR, migrenli hastalarda oksidatif ve inflamatuvar süreçlerin değerlendirilmesinde yararlı bir belirteç olabilir. Klinik öneminin netleştirilmesi için ileriye dönük çalışmalara ihtiyaç vardır.

Anahtar Kelimeler: Migren. Ürik asit. Albümin. Ürik asit/albümin oranı. Oksidatif stres.

Date Received: 6.November.2025
Date Accepted: 26.February.2026

Dr. Deniz KAMACI ŞENER
Department of Neurology, Bursa City Hospital,
Bursa, Türkiye
E-mail: drdenizkamaci@gmail.com
https://orcid.org/0000-0003-3433-6859

AUTHORS' ORCID INFORMATION
Deniz KAMACI ŞENER: 0000-0003-3433-6859
Özden KAMIŞLI: 0000-0003-1114-7860

Migraine is a common neurological disorder affecting approximately 15% of the general population and is a leading cause of disability worldwide, particularly among young and middle-aged adults[1]. The fundamental pathology of migraine is still not fully understood. Accumulating evidence suggests that oxidative stress may exacerbate migraine through mechanisms such as vascular dysfunction, cortical spreading depression, and neuronal hyperexcitability.

Emerging data also indicate that oxidative stress and systemic inflammation play important roles in migraine pathophysiology[2]. Uric acid (UA), the final product of purine metabolism, is considered one of the major endogenous antioxidants in human plasma and accounts for a substantial proportion of total antioxidant capacity. It exerts protective effects by scavenging reactive oxygen and nitrogen species and by neutralizing peroxynitrite. However, under certain conditions, elevated uric acid levels may also display pro-oxidant and pro-inflammatory properties, reflecting its dual role in redox biology[3]. Albumin (ALB), the most abundant plasma protein, also contributes to antioxidant defense and reflects systemic inflammation as a negative acute-phase reactant. Therefore, the balance between uric acid and albumin may provide insight into the oxidative and inflammatory status of patients[4,5].

Several clinical studies have examined oxidative stress parameters in migraine; however, the results have been inconsistent. While some authors found no significant differences, others reported lower serum UA levels in migraine patients compared with controls, suggesting compromised antioxidant defense[6-8]. ALB has been less frequently investigated in migraine patients. The limited number of studies evaluating albumin levels have yielded mixed results, with some reporting no differences and others demonstrating lower serum albumin levels in migraine patients[8,9].

Given these conflicting findings regarding UA and ALB in migraine, combining these two parameters into a single index may provide a more comprehensive and reliable reflection of oxidative and inflammatory balance. The uric acid-to-albumin ratio (UAR) has recently emerged as a novel biomarker reflecting redox and inflammatory status and has been associated with prognosis in cardiovascular diseases, hypertension, and acute kidney injury [5,10,11].

The aim of this case–control study was to evaluate serum UA, ALB, UAR in patients with episodic migraine during the interictal period, and to investigate their associations with clinical features and migraine-related disability. In addition, we aimed to explore the potential contribution of these parameters to oxidative stress–related mechanisms in migraine pathophysiology.

Materials and Methods

This retrospective case–control study included 120 patients with episodic migraine (EM) diagnosed according to the International Classification of Headache Disorders, 3rd edition (ICHD-3), and 100 healthy controls who were admitted to the Neurology outpatient clinic between September 2023 and June 2025. Participants were between 18 and 60 years of age. Healthy controls were selected from volunteers without a history of migraine, other primary headache disorders, or chronic systemic diseases.

Demographic and clinical data, including age, sex, body mass index (BMI), smoking status, migraine duration, attack frequency and duration, presence of aura, and associated symptoms (nausea, vomiting, photophobia, phonophobia), were recorded. Migraine-related disability was assessed using the Migraine Disability Assessment Scale (MIDAS) within the migraine group. Pain intensity was evaluated using the Visual Analog Scale (VAS).

Venous blood samples were obtained during routine outpatient visits when patients were not experiencing an acute migraine attack, as documented in medical records. Serum UA and ALB levels were measured using standard enzymatic and colorimetric methods. Albumin concentrations were originally expressed in g/L and converted to g/dL for unit consistency. The uric acid-to-albumin ratio (UAR) was calculated as UA (mg/dL) divided by ALB (g/dL).

In the comparative analyses, the EM and control groups were evaluated in terms of demographic variables (age, sex, BMI, smoking status) and laboratory parameters (UA, ALB, and UAR). Within the migraine group, correlation analyses were performed to examine the associations between biochemical parameters (UA, ALB, and UAR) and clinical variables, including MIDAS scores, VAS, attack frequency, and aura status.

Exclusion criteria included chronic systemic inflammatory disease, autoimmune disease, malignancy, hepatic or renal failure, hematological disorders, acute infection, pregnancy or lactation, chronic alcohol consumption, and use of medications that could affect oxidative or inflammatory parameters (e.g., corticosteroids, immunosuppressive agents, or antioxidant supplements). Patients receiving drugs known to influence uric acid metabolism (such as allopurinol, febuxostat, or diuretics) were also excluded. Patients with incomplete clinical or laboratory data were not included in the analysis.

Statistical Analysis

Data distribution was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± standard deviation or median (min–max), as appropriate, and categorical variables were presented as frequencies and percentages. Comparisons between migraine patients and healthy controls were performed using Student's t-test or the Mann–Whitney U test according to data distribution. The chi-square test was used for categorical variables. Correlation analyses within the migraine group were conducted using Pearson or Spearman correlation coefficients, as

appropriate. All analyses were performed using SPSS version 24.0 (SPSS Inc., Chicago, IL, USA), and a two-tailed p value <0.05 was considered statistically significant.

Results

A total of 220 individuals (120 patients with EM and 100 healthy controls) were included in the analysis. The mean age was 31.56±8.33 years in the EM group and 31.40±8.31 years in the control group, with no significant difference between the groups (p>0.05). Sex distribution, BMI, and smoking status were also similar between the groups (all p> 0.05). The demographic characteristics of the study population are presented in Table I.

Table I. Demographic characteristics of patients with episodic migraine (EM) and healthy controls

Variable	Migraine (n=120)	Control (n=100)	p-value
Age (year)	31.40 ± 8.31	31.56 ± 8.33	0.750
Gender (F/M)	100/20	82 / 18	0.935
BMI (kg/m²)	24.35 ± 4.03	24.66 ± 4.46	0.590
Smoker (yes %)	30 (30.0%)	39 (32.5%)	0.801

Values are presented as mean ± standard deviation or n (%). BMI: Body mass index.

In the EM group, the mean disease duration was 7.58±7.14 years. The mean VAS score was 7.83±1.58, and the mean MIDAS score was 8.78±6.92. Migraine with aura (MA) was detected in 15% (n=18) of patients, whereas the majority (85%, n=102) had migraine without aura (MWA). The demographic and clinical characteristics of EM patients are summarized in Table II.

Table II. Demographic and clinical characteristics of patients with EM

Variable	Migraine (n=120)
Age (years)	31.56 ± 8.33 (18–58)
Gender (F/M)	100 / 20
BMI (kg/m²)	24.66 ± 4.46 (15.06–34.77)
Smoker	39 (32.5%)
Migraine duration (years)	7.58 ± 7.14 (0.0–46.0)
VAS	8.00 (IQR 7.00–9.00)
MIDAS	7.00 (IQR 4.00–12.00)
Aura	18 (15.0%)
Nausea	106 (88.3%)
Vomiting	51 (42.5%)
Photophobia	88 (73.3%)
Phonophobia	95 (79.2%)

Values are presented as mean ± standard deviation or n (%).EM: episodic migraine; BMI: Body mass index, VAS: Visual Analog Scale, MIDAS: Migraine Disability Assessment Scale.

Serum UA levels were significantly lower in the EM group compared to controls (4.13±1.09 vs. 4.60±1.03 mg/dL, p<0.001). Serum ALB levels did not differ significantly between the two groups (4.56±0.28 vs. 4.60±0.38 g/dL, p=0.342). In addition, the UAR was significantly lower in EM patients than in controls (0.91±0.24 vs. 1.00±0.23, p<0.001). The comparison of biochemical parameters between the EM and control groups is presented in Table III.

Table III. Biochemical parameters in patients with EM and healthy controls

Parameter	Control (n=100)	Migraine (n=120)	p-value
UA (mg/dL)	4.60 ± 1.03	4.13 ± 1.09	**p<0.001**
ALB (g/dL)	4.60 ± 0.38	4.56 ± 0.28	p=0.342
UAR	1.00 ± 0.23	0.91 ± 0.24	**p<0.001**

Values are presented as mean ± standard deviation. EM: episodic migraine; UA: uric acid; ALB: albumin; UAR: uric acid-to-albumin ratio;

Correlation analyses within the migraine group showed that attack frequency was positively correlated with MIDAS (r=0.523, p<0.001), and VAS scores were also positively correlated with MIDAS (r=0.244, p=0.007). Among biochemical parameters, UAR showed a strong positive correlation with UA (r=0.968, p<0.001) and a negative correlation with ALB (r= –0.213, p=0.020). These findings are presented in Table IV.

Comparison of biochemical parameters between MA (MWA) is presented in Table V. No statistically significant differences were found between the groups in terms of serum UA, albumin, or UAR levels (all p>0.05).

Discussion and Conclusion

In this case–control study, we evaluated serum UA, ALB, and UAR in patients with episodic migraine during the interictal period. Our findings showed that UA and UAR levels were significantly lower in migraine patients compared with healthy controls, whereas ALB levels were similar between the groups. In addition, serum UA, ALB, and UAR levels were not significantly associated with MIDAS, VAS, attack frequency, or the presence of aura.

These findings may suggest reduced systemic antioxidant capacity in patients with migraine, supporting the hypothesis that oxidative stress contributes to migraine pathophysiology. Oxidative stress and systemic inflammation are known to interact and may promote neuroinflammatory processes involved in migraine development[12,13].

Table IV. Correlation analysis between clinical features and biochemical parameters in patients with EM

	Age	BMI	Migraine duration	Attack frequency	Pain duration	VAS	MIDAS	UA	ALB	UAR
Age		**r=0.192 p=0.0354**	**r=0.292 p=0.0012**	r=-0.022 p=0.8140	r=0.059 p=0.5219	r=0.024 p=0.7974	r=0.132 p=0.1518	r=-0.054 p=0.5601	**r=-0.316 p=0.0004**	r=0.003 p=0.9783
BMI			r=0.055 p=0.5519	r=-0.038 p=0.6763	r=0.171 p=0.0623	r=0.031 p=0.7397	r=-0.036 p=0.6928	**r=0.201 p=0.0276**	**r=-0.220 p=0.0158**	**r=0.245 p=0.0073**
Migraine duration				r=-0.046 p=0.6163	r=0.062 p=0.5023	r=-0.141 p=0.1242	r=0.036 p=0.6931	r=0.055 p=0.5504	r=-0.080 p=0.3847	r=0.052 p=0.5717
Attack frequency					r=0.004 p=0.9631	r=0.149 p=0.1045	**r=0.523 p<0.001**	r=-0.018 p=0.8432	r=-0.093 p=0.3140	r=-0.013 p=0.8840
Pain duration						r=0.105 p=0.2516	r=0.157 p=0.0868	r=-0.124 p=0.1758	r=0.067 p=0.4692	r=-0.139 p=0.1308
VAS							**r=0.244 p=0.0073**	r=-0.138 p=0.1338	r=-0.134 p=0.1451	r=-0.107 p=0.2454
MIDAS								r=-0.103 p=0.2618	r=-0.055 p=0.5508	r=-0.100 p=0.2785
UA									r=0.005 p=0.9542	**r=0.968 p<0.001**
ALB										**r=-0.213 p=0.0195**

Bold values indicate statistically significant correlations (p < 0.05). EM: episodic migraine; UA: uric acid; ALB: albumin; UAR: uric acid-to-albumin ratio; BMI: body mass index; VAS: visual analog scale; MIDAS: Migraine Disability Assessment Scale.

Table V. Comparison of serum UA, ALB and UAR between MA and MWA

Variable	MA (n=18) mean ± SD	MWA (n=102) mean ± SD	p-value
UA	4.03 ± 0.78	4.15 ± 1.13	0.848
ALB	4.55 ± 0.22	4.56 ± 0.29	0.936
UAR	0.90 ± 0.02	0.91 ± 0.03	0.886

Note: Data are presented as mean ± SD. MA: migraine with aura; MWA: migraine without aura; UA: uric acid; ALB: albumin; UAR: uric acid-to-albumin ratio

Uric acid is a major endogenous antioxidant and the final product of purine metabolism, capable of scavenging reactive oxygen species[14]. In addition to its antioxidant properties, UA can cross the blood–brain barrier and may exert neuroprotective effects. Reduced UA levels have been reported in several neurological disorders[15,16]. Previous studies investigating UA levels in migraine have yielded inconsistent results. While some studies found no significant difference between migraine patients and controls[6], others reported lower UA levels in migraine[7]. Consistent with the latter findings, we observed significantly lower UA levels in migraine patients compared with healthy controls. However, UA was not significantly associated with clinical features such as attack frequency, pain intensity, or MIDAS scores, suggesting that reduced UA may reflect altered antioxidant status rather than disease severity in episodic migraine.

Albumin is a major plasma protein with antioxidant properties that contribute to the neutralization of reactive oxygen and nitrogen species[4]. Some studies have reported lower serum ALB levels in migraine patients, possibly reflecting increased oxidative stress[8,17]. However, findings have been inconsistent. Yang et al. observed reduced ALB levels in migraine overall but found no significant difference between episodic and chronic migraine[8]. In our study, total ALB levels did not differ significantly between episodic migraine patients and healthy controls.

It should be noted that total ALB concentration may not fully reflect its functional antioxidant capacity. Oxidative stress can induce structural modifications of ALB, particularly at the N-terminal site, leading to the formation of ischemia-modified albumin (IMA). Previous studies have demonstrated increased IMA levels in migraine patients, suggesting that oxidative stress may alter ALB function rather than its total concentration[18,19]. Since IMA was not measured in the present study, potential functional alterations of ALB could not be evaluated. Therefore, the absence of differences in total ALB levels does not exclude structural or functional changes.

UAR has recently emerged as a composite biomarker reflecting both oxidative stress and systemic inflammation. Several studies have demonstrated the clinical significance of UAR in cardiovascular diseases, hypertension, and acute kidney injury[11,20,21].

Although UAR has not been extensively investigated in migraine, its biological rationale appears plausible. Migraine is increasingly recognized as a neurovascular disorder characterized by endothelial dysfunction, altered vascular reactivity, and low-grade systemic inflammation. While UA reflects antioxidant capacity, ALB is influenced by inflammatory and

acute-phase responses. Therefore, UAR may represent the balance between oxidative stress and inflammatory activity. A reduced UAR could indicate oxidative predominance and impaired vascular–endothelial regulation, processes that have been implicated in migraine pathophysiology. This integrated perspective may help explain why UAR differed between groups even when albumin levels alone did not show significant changes.

Since UAR reflects the balance between oxidative and inflammatory processes, we further examined its association with clinical severity and migraine-related disability. Specifically, we analyzed correlations between UAR and clinical parameters, including VAS and MIDAS scores, to evaluate whether UAR might reflect disease burden in EM.

Previous studies have reported inconsistent findings regarding the association between oxidative stress and migraine severity. While some studies suggested that higher attack frequency is associated with increased oxidative stress and reduced antioxidant defense, others did not observe significant relationships between UA, ALB, and clinical features of migraine[7,22]. In our study, UA, ALB, and UAR were not significantly correlated with migraine duration, attack frequency, VAS or MIDAS scores. These findings suggest that although UAR may reflect systemic oxidative and inflammatory balance, it does not appear to directly correspond to clinical severity in episodic migraine.

One possible explanation is that interictal measurements may not fully capture dynamic oxidative changes occurring during migraine attacks. Additionally, oxidative and inflammatory mechanisms may affect migraine through complex pathways that are not fully captured by routine clinical indices. Migraine is a multifactorial disorder influenced by genetic susceptibility, psychosocial stress, and environmental triggers, which may contribute substantially to disease expression. Future longitudinal studies incorporating both ictal and interictal assessments may provide further insight into the temporal relationship between oxidative processes and migraine activity.

Aura is thought to represent a transient cortical phenomenon related to cortical spreading depolarization, a process associated with oxidative and inflammatory activity[23,24]. Previous research comparing MA and MWA has generally not demonstrated major differences in systemic oxidative markers between the two subtypes. Yazar et al.[15] reported that both MA and MWA patients had lower serum UA levels than healthy controls, with no significant difference between migraine subgroups. Likewise, Yang et al.[8] found no significant variation in UA or ALB levels between the two forms. Consistent with these findings, our study also demonstrated comparable UA, ALB, and UAR values in patients MA and MWA. However, the relatively small number of patients with aura in our cohort may have limited the statistical power to detect subtle differences.

Although current evidence suggests that systemic oxidative–inflammatory balance does not markedly differ between migraine subtypes, it is possible that aura-related oxidative changes are localized at the cortical level and therefore not fully reflected by peripheral biochemical markers. This distinction may partly explain why systemic measurements fail to capture potential pathophysiological differences associated with aura.

In conclusion, our study demonstrated that serum UA levels and UAR were significantly lower in patients with episodic migraine compared with healthy controls, whereas ALB levels were similar between the groups. These findings may indicate a reduction in systemic antioxidant capacity in migraine. However, UA, ALB, and UAR were not significantly associated with pain intensity, migraine-related disability, or attack frequency. This suggests that although oxidative imbalance may be involved in migraine pathophysiology, it does not appear to directly reflect clinical severity in EM. Further prospective and longitudinal studies are needed to clarify the clinical relevance of these biomarkers.

This study has several limitations. First, its retrospective and cross-sectional design limits causal inference. Second, the sample size—particularly for patients with aura—was relatively small, which may have reduced the statistical power of subgroup analyses and limited the ability to detect subtle differences between migraine subtypes.

In addition, other oxidative stress markers such as total antioxidant capacity, total oxidant status, malondialdehyde, superoxide dismutase, glutathione peroxidase, and IMA were not evaluated. Assessment of these parameters might have provided a more comprehensive characterization of redox balance.

Another limitation relates to the interictal assessment. Although blood samples were obtained when patients were not experiencing an acute migraine attack, the exact duration of the interictal period could not be standardized due to the retrospective design. Since oxidative and inflammatory parameters may vary between ictal and interictal phases, this variability may have influenced the findings.

Despite these limitations, our results support a potential role of oxidative imbalance in migraine pathophysiology and provide a basis for future prospective, multicenter studies aimed at clarifying the clinical significance of these biomarkers.

Researcher Contribution Statement
Idea and design: D.K.Ş., Ö.K.; Data collection and processing: D.K.Ş.; Analysis and interpretation of data: D.K.Ş., Ö.K.; Writing of significant parts of the article: D.K.Ş.

Support and Acknowledgement Statement
No specific financial support was received for this study. The authors thank the laboratory and neurology clinic staff of Bursa City Hospital for their assistance in data collection.

Conflict of Interest Statement
The authors declare that there is no conflict of interest regarding this study.

Ethics Committee Approval Information
Approving Committee: Bursa City Hospital Ethics Committee
Approval Date: 15.10.2025
Decision No: 2025-20/9

References

1. Feigin VL, Vos T, Nichols E, et al. The global burden of neurological disorders: translating evidence into policy. Lancet Neurol. 2020;19(3):255-265. doi:10.1016/S1474-4422(19)30411-9.
2. Jiménez-Jiménez FJ, Alonso-Navarro H, García-Martín E, Espada-Rubio S, Agúndez JA. Oxidative stress and migraine. Mol Neurobiol. 2024;61(10):8344-8360. doi:10.1007/s12035-024-04114-7.
3. Sautin YY, Johnson RJ. Uric acid: the oxidant-antioxidant paradox. Nucleosides Nucleotides Nucleic Acids. 2008;27(6-7):608-619. doi:10.1080/15257770802138558
4. Roche M, Rondeau P, Singh NR, Tarnus E, Bourdon E. The antioxidant properties of serum albumin. FEBS Lett. 2008;582(13):1783-1787. doi:10.1016/j.febslet.2008.04.057
5. Yalcinkaya D, Karacali K, Ilhan BC, Yarlioglues M. Relation between serum uric acid to albumin ratio and severity of chronic coronary artery disease. Angiology. 2024;75(4):386-393. doi:10.1177/00033197231161902
6. Altunkaynak Y, Keskek A, Donmezler S, et al. A study of the relationship between serum uric acid levels and pain in patients with migraine. Medicine (Baltimore). 2023;102(5):e32810. doi: 10.1097/MD.0000000000032810.
7. Yazar T, Yazar HO, Aygün A, Karabacak V, Altunkaynak Y, Kirbaş D. Evaluation of serum uric levels in migraine. Neurol Sci. 2021;42(2):705-709. doi:10.1007/s10072-020-04598-w
8. Yang Z, Xu P, Geng C, Zhang H. Evaluation of simple antioxidant blood parameters in patients with migraine. Front Neurol. 2022;13:939363. doi:10.3389/fneur.2022.939363
9. Yasin S, Altunisik E. Decreased serum uric acid level as an indicator of altered oxidative balance in patients with migraine. Cureus. 2022;14(12). doi: 10.7759/cureus.32136.
10. Şaylık F, Çınar T, Selçuk M, Tanboğa İH. The relationship between uric acid/albumin ratio and carotid intima-media thickness in patients with hypertension. Arq Bras Cardiol. 2023;120:e20220819. doi:10.36660/abc.20220819
11. Özgür Y, Akın S, Yılmaz NG, Gücün M, Keskin Ö. Uric acid albumin ratio as a predictive marker of short-term mortality in patients with acute kidney injury. Clin Exp Emerg Med. 2021;8(2):82-87. doi:10.15441/ceem.20.024
12. Taheri M, Nicknafs F, Hesami O, et al. Differential expression of cytokine-coding genes among migraine patients with and without aura and normal subjects. J Mol Neurosci. 2021;71(6):1197-1204. doi:10.1007/s12031-020-01745-y.
13. Neri M, Frustaci A, Milic M, et al. A meta-analysis of biomarkers related to oxidative stress and nitric oxide pathway in migraine. Cephalalgia. 2015;35(10):931-937. doi:10.1177/0333102414564888.
14. Strazzullo P, Puig JG. Uric acid and oxidative stress: relative impact on cardiovascular risk. Nutr Metab Cardiovasc Dis. 2007;17(6):409-414. doi:10.1016/j.numecd.2007.02.011.
15. Yazar HO, Yazar T, Yildirim T, Keşkek A, Altunkaynak Y. Assessment of serum uric acid levels in patients with restless legs syndrome. Acta Neurol Belg. 2019;119(3):461-466. doi:10.1007/s13760-019-01177-5.
16. Brody DM, Litvan I, Warner S, et al. Relationship between uric acid levels and progressive supranuclear palsy. Mov Disord. 2016;31(5):663-667. doi:10.1002/mds.26535
17 Cheng Y-C., Chang Y-T. Serum uric acid and albumin levels in patients with migraine. J Neurol Sci. 2023;455. doi:10.1016/j.jns.2023.121625.
18. Ersoy A, Yasar H, Mertoglu C, et al. Is ischemia associated with the formation of white matter lesions in migraine? Clin Neurol Neurosurg. 2020;193:105770. doi:10.1016/j.clineuro.2020.105770.
19. Say B, Badem ND, Ergun U. Serum levels of ischemic modified albumin (IMA) and prolidase in migraine subjects. J Health Sci Med. 2020;3(1):31-35. doi:10.32322/jhsm.638466.
20. Zhang X-j, Hou A-j, Luan B, Wang C-f, Li J-j. Uric acid to albumin ratio as a novel predictor for coronary slow flow phenomenon in patients with chronic coronary syndrome and non-obstructive coronary arteries. BMC Cardiovasc Disord. 2024;24(1):358. doi:10.1186/s12872-024-04040-5.
21. Oflar E, Yildiz C, Koyuncu A, et al. Relationship between uric acid albumin ratio and peripheral artery disease complexity. Eur Rev Med Pharmacol Sci. 2023;27(23):11472-11478.
22. Togha M, Razeghi Jahromi S, Ghorbani Z, Ghaemi A, Rafiee P. An investigation of oxidant/antioxidant balance in patients with migraine: a case-control study. BMC Neurol. 2019;19(1):323. doi:10.1186/s12883-019-1555-4.
23. Ayata C, Lauritzen M. Spreading depression, spreading depolarizations, and the cerebral vasculature. Physiol Rev. 2015;95(3):953-993. doi:10.1152/physrev.00027.2014.
24. Charles A, Brennan K. Cortical spreading depression—new insights and persistent questions. Cephalalgia. 2009;29(10):1115-1124. doi:10.1111/j.1468-2982.2009.01983.x.