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Predictive Value of Uric Acid to High Density Lipoprotein Ratio and Other Factors in Obstructive Sleep Apnea Syndrome

Ürik Asit HDL Oranının ve Diğer Faktörlerin Obstrüktif Apne Sendromu Tanısındaki Değeri

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Abstract

Objective: In this study, we aimed to examine mainly the uric acid to HDL ratio (UHR) and some other factors in patients with OSAS and to see if this ratio could be used as a marker for the disease.

Materials and Methods: We reviewed the records of 113 patients who had polysomnography. We collected demographic information, OSAS risk factors, polysomnography results such as minimum oxygen saturation, REM percentage, and apnea hypopnea index, and laboratory values including UHR and the monocyte to HDL ratio. We analyzed how these factors were related to disease severity.

Results: We found that UHR was significantly associated with disease severity, showing that it may be a useful predictive marker. The monocyte to HDL ratio and the mean heart rate during sleep were also related to disease severity. These results show that several simple and accessible markers may help in understanding OSAS.

Conclusion: UHR may support earlier and more effective identification of patients with OSAS.

Keywords: Apnea Hypopnea Index, Obstructive Sleep Apnea Syndrome, Polysomnography, Uric Acid

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Öz

Amaç: Bu çalışmada, OSAS'lı hastalarda başlıca ürik asit/HDL oranını (UHR) ve bazı diğer faktörleri incelemeyi ve bu oranın hastalık için bir belirteç olarak kullanılıp kullanılamayacağını değerlendirmeyi amaçladık.

Gereç ve Yöntemler: Polisomnografi yapılan 113 hastanın kayıtlarını inceledik. Demografik bilgiler, OSAS risk faktörleri, minimum oksijen satürasyonu, REM yüzdesi ve apne-hipopne indeksi gibi polisomnografi sonuçları ile UHR ve monosit/HDL oranı dahil laboratuvar değerlerini topladık. Bu faktörlerin hastalık şiddeti ile ilişkisini analiz ettik.

Bulgular: UHR'nin hastalık şiddeti ile anlamlı şekilde ilişkili olduğunu ve bu yüzden yararlı bir öngörücü belirteç olabileceğini gördük. Monosit/HDL oranı ve uyku sırasındaki ortalama kalp hızı da hastalık şiddeti ile ilişkiliydi. Bu sonuçlar, OSAS'ı anlamada basit ve kolay ulaşılabilir birkaç belirtecin yardımcı olabileceğini göstermektedir.

Sonuç: UHR, OSAS'lı hastaların daha erken ve daha etkili şekilde belirlenmesine katkı sağlayabilir.

Anahtar Kelimeler: Apne-Hipopne İndeksi, Obstrüktif Uyku Apne Sendromu, Polisomnografi, Ürik Asit

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Introduction

Obstructive Sleep Apnea Syndrome (OSAS) is presenting by repeated episodes of partial or complete airway obstruction in sleep. It is usually defined by an apnea hypopnea index (AHI) of 5 or more events per hour, measured by polysomnography (1). OSAS affects more than 10% of the general population, and its prevalence is reported between 9% and 38% in adults. It is seen more often in men (13%–33%) than in women (6%–19%), and it becomes more common with increasing age (2). Additionally, OSAS is strongly linked to overweight and obesity, with 60% to 70% of obese individuals affected, and the prevalence surpassing 90% in those with severe obesity (2,3).

Repeated airway obstructions causes intermittent hypercapnia and hypoxia, which can lead to low oxygen levels and frequent wake-ups during sleep (4). These hypoxia–reoxygenation cycles increase the production of reactive oxygen species (ROS) and disturb cellular metabolism. When oxygen is not enough, ATP production is reduced, and ATP breaks down into ADP, AMP, and finally higher uric acid levels (5,6).

For this study, the uric acid to HDL ratio (UHR) and the monocyte to HDL ratio (MHR) were chosen as the main biomarkers because of their link with OSAS pathophysiology. Patients with OSAS often show increased inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), and these are known to be related to hypoxia and oxidative stress severity (7). UHR is indicative of oxidative stress and metabolic disturbances, both of which are exacerbated in OSAS. MHR is related to inflammation and atherosclerosis, conditions that are also common in OSAS. Since these biomarkers are easy to measure and low-cost, they are practical for clinical use.

High uric acid levels have been associated with heart failure, atherosclerosis, and hypertension, and they are considered an independent risk factor for mortality in people with high cardiovascular risk (8). Studies show that uric acid excretion increases in OSAS patients and decreases after CPAP treatment, which suggests a possible relationship between uric acid and OSAS (9,10).

Metabolic syndrome (MetS) is diagnosed based on specific criteria, one of which is low high-density lipoprotein cholesterol (HDL-C) levels Many studies have shown a strong association between OSAS and MetS (11,12,13). Which led to the idea that low HDL levels may also be a possible marker for OSAS.

Although UHR has been studied in different diseases, it has not been widely investigated in OSAS patients. In this study, we aimed to examine mainly the UHR and some other factors in patients with OSAS and to see if this ratio could be used as a marker for the disease.

Materials and Methods

The files of 113 patients who underwent polysomnography at the BAIBU EAH Neurology Clinic between January 2024 and May 2024 were retrospectively reviewed after ethics committee approval (date: 04.06.2024 and approval number: 2024/132). The study was performed in line with the ethical principles of the Declaration of Helsinki. The review included demographic data, OSAS risk factors, polysomnography findings (such as minimum oxygen saturation, sleep rapid eye movement (REM) ratio, and AHI, and laboratory results. Laboratory results examined included serum C-reactive protein (CRP), albumin, platelet count and other complete blood count data. Inflammatory parameters assessed included Neutrophil Lymphocyte Ratio (NLR), Monocyte HDL Ratio (MHR) and the UHR. Patients with OSAS were grouped by severity according to the AHI: Normal (AHI < 5), Mild (AHI 5–15), Moderate (AHI 15–30), and Severe (AHI > 30). Patients who had a history of cancer or any neurological disease were not included in the study. We also excluded people with acute infections to avoid factors that could affect the disease or the biomarkers. These criteria were used to reduce confounding effects that might change UHR levels or OSAS severity. Cancer and acute infections were excluded because they are related to systemic inflammation and metabolic changes, and neurological diseases were excluded because they can influence breathing control, sleep structure, and OSAS-related outcomes.

Statistical Analysis

Normality of continuous data was assessed using the Kolmogorov-Smirnov test, and homogeneity of variances was evaluated before applying the respective tests. Continuous data were compared using the Kruskal-Wallis test with Dunn's test for post-hoc analysis for data showing non-homogeneous distribution,

with median, minimum, and maximum values reported. For data showing homogeneous distribution, one-way ANOVA with Tukey's post-hoc test was used, with mean and standard deviation reported. Categorical data were compared using Pearson's Chi-squared test. Multinomial logistic regression models were used to evaluate the associations. To evaluate the statistical adequacy of the study, a post-hoc power analysis was conducted using effect sizes derived from relevant literature and our data. Effect sizes reported in studies by Hirotsu et al. (f=0.58), Aktas et al. (f=0.70), and Kolahi Ahari et al. (f=0.79) were considered alongside the effect size calculated from our data (f=0.82)(6,14,15). In all scenarios, the power we reached with the current sample of 113 patients was almost 100%. The minimum sample size needed to reach 80% power was between 22 and 48 patients. The minimum sample size required to achieve 80% power ranged from 22 to 48 participants. Statistical analyses were performed using SPSS version 16 (SPSS Inc., Armonk, NY), and the significance level was determined as $p<0.05$. Power analysis was conducted using the statistical software G*Power.

Results

A total of 113 patients were included, with a mean age of 48.56 ± 13.84 years (range: 19–74 years), and 37 (32.7%) of the patients were female.

Hypertension (40, 35.4%) was the most common comorbidity, followed by diabetes (29, 25.7%), hyperlipidemia (25, 22.1%), and coronary artery disease (23, 20.4%)

Regarding the severity of OSAS, 12 (10.6%) patients had normal findings, 22 (19.5%) had mild OSAS, 19 (16.8%) had moderate OSAS, and 60 (53.1%) had severe OSAS. (Table 1.).

Table 1

Demographic Characteristics

	Mean±Std	**Min-Max**
Age, Year	48.56±13.84	19-74
	n	**%**
Gender, Female	37	32.7
Hypertension	40	35.4
Diabetes	29	25.7
Hyperlipidemia	25	22.1
Coronary Diseaase	23	20.4
MI/CVD	9	8
Chronic Kidney Disease	6	5.3
Pulmonary Hypertension	1	0.9
OSAS		
Normal	12	10.6
Mild	22	19.5
Moderate	19	16.8
Severe	60	53.1

CVD: Cerebrovascular Disease, **MI**: Myocardial Infarction, **OSAS**: Obstructive Sleep Apnea Syndrome.

Age was significantly different between the groups ($p<0.001$). Patients in the Severe OSAS group were older, with a median age of 51 years (23-74), while the Normal group had a median age of 27 years (19-50). Gender distribution also differed significantly ($p = 0.001$), with the highest proportion of females in the Normal group (75%) and a noticeable decline in the percentage of female patients in the more severe OSAS groups.

The Severe OSAS group had higher instances of coronary artery disease (23.3%) and myocardial infarction/cerebrovascular disease (10%), though the difference was not statistically significant.

Laboratory results showed that white blood cell counts, hemoglobin, and mean corpuscular volume did not differ much between the groups. C-reactive protein levels were generally higher in the Severe OSAS group. UHR and MHR were also significantly higher in this group ($p<0.05$). For the sleep-related findings, minimum

SO_2 levels were lower in the Severe OSAS group, and the mean heart rate was significantly higher compared to the other groups.

In terms of sleep-related parameters, minimum SO2 levels were lower in the Severe OSAS group, and the mean heart rate was significantly higher compared to the other groups. No significant differences were observed in the percentage of REM sleep across the severity groups (p>0.05) (Table 2).

Table 2

Statistical Data

	Normal	Mild	Moderete	Severe	P
Age, Year	27(19-50)	45.5(22-64)	55(24-74)	51(23-74)	**<0.001****
Gender, Female	9(75)	10(45.5)	3(15.8)	15(25)	**0.001***
Hypertension	1(8.3)	8(36.4)	8(42.1)	23(38.3)	0.217*
Diabetes	0	5(22.7)	7(36.8)	17(28.3)	0.127*
Hyperlipidemia	0	2(9.1)	5(26.3)	18(30)	**0.047***
Coronary Diseaase	0	4(18.2)	5(26.3)	14(23.3)	0.275*
MI/CVD	0	0	3(15.8)	6(10)	0.182*
Chronic Kidney Disease	0	0	1(5.3)	5(8.3)	0.392*
Pulmonary Hypertension	0	0	0	1(1.7)	0.828*
WBC	6.33(3.50-8.40)	7.64(5.81-17.46)	6.52(4.82-13.37)	7.7(4.96-21-46)	0,067**
HGB	13(10.4-15.9)	15.1(11.5-18.1)	15.1(11.7-18)	14.9(10.5-16.6)	0,182**
MCV	84.9(78.2-95.5)	86.8(79.2-96.2)	85.2(66.5-94.6)	85.5(73.8-97.7)	0,478**
MONO	0.45(0.38-0.73)	0.61(0.41-1.37)	0.55(0.24-0.75)	0.58(0.1-1.27)	**0,028****
NEU	3.76(1.9-49.7)	4.50(2.66-14.01)	3.79(2.36-10.84)	4.5(2.38-15.81)	0,146**
CRP	1.6(0.1-4.6)[a]	4.4(0.4-33.1)[a,b]	1.9(0.1-23.8)[a,b]	4(1.1-31.8)[b]	**0,005****
ALB	48(40.5-54)	45.65(41-51)	45(38-54)	43.7(38-50)	0,213**
HDL	52.3(29-87.8)	46.8(33.6-68.7)	50(30.8-62.2)	41.9(13-78.8)	**0,02****
NLR	1.42(1.11-20.62)	2.15(0.99-5.5)	1.56(0.9-5.68)	1.8(1.04-9.45)	0,277**
UHR	0.09(0.05-0.2)	0.1(0.04-0.16)	0.11(0.07-0.26)	0.14(0.05-0.49)	**0.008****
MIN SO2	90.5(87-95)[a]	86(79-90)[a,b]	85(50-89)[a,b]	75(54-87)[b]	**<0.001****
%REM	14.5(9-22.7)	14.7(4.6-22.4)	13.9(1.3-42)	10.5(1.3-27)	0,133**
MHR	0.0097(0.005-0.02)	0.011(0.006-0.04)	0.010(0.005-0.02)	0.015(0.002-0.035)	**0.006****
PLT	252±65.8	263.2±63.8	272±48	280.5±64.7	0.366***
LYM	2.3±0.49	2.4±0.74	2.35±0.67	2.46±0.86	0.889***
Uric Acid	4.7±1.2	5±1	5.9±1.4	5.7±1.3	**0.007*****
MEAN HR	65.9±7	69.5±9	67.7±9.6	72.6±10.8	**0.05*****

*Categorical data were summarized as frequency and percentage and analyzed using Pearson's chi-squared test **: Continuous data were summarized as median (min-max) and analyzed using the Kruskal Wallis test (Bonferroni correction and Post-Hoc Dunn test). ***: Data were summarized as mean±sd and analyzed using the One-way ANOVA test (Bonfferroni correction Posthoc Tukey test). a,b: According to the results of the Post-Hoc tests, different letters indicate a significant difference between the groups, p<0.008.
ALB: Albumin, **CRP**: C-reactive Protein, **CVD**: Cerebrovascular Disease, **HDL**: High Density Lipoprotein, **HGB**: Hemoglobin, **LYM**: Lymphocyte, **MCV**: Mean Corpuscular Volume, **MEAN HR**: Mean Heart Rate, **MHR**: Monocyte to **HDL** Ratio, **MI**: Myocardial Infarction, **MIN SO2**: Minimum **O2** Saturation During Sleep, **MONO**: Monocyte, **NEU**: Neutrophil, **NLR**: Neutrophil to Lymphocyte Ratio, **OSAS**: Obstructive Sleep Apnea Syndrome, **PLT**: Platelets, **%REM**: Percentage of **REM** Sleep Duration, **UHR**: Uric Acid to **HDL** Ratio, **WBC**: White Blood Cell

In Receiver Operating Characteristic (ROC) analysis, a UHR level greater than 0.123 had 53% sensitivity and 90% specificity in predicting the presence of OSAS (AUC: 0.74, p<.001, 95% CI: 0.65–0.83) (Figure 1.).

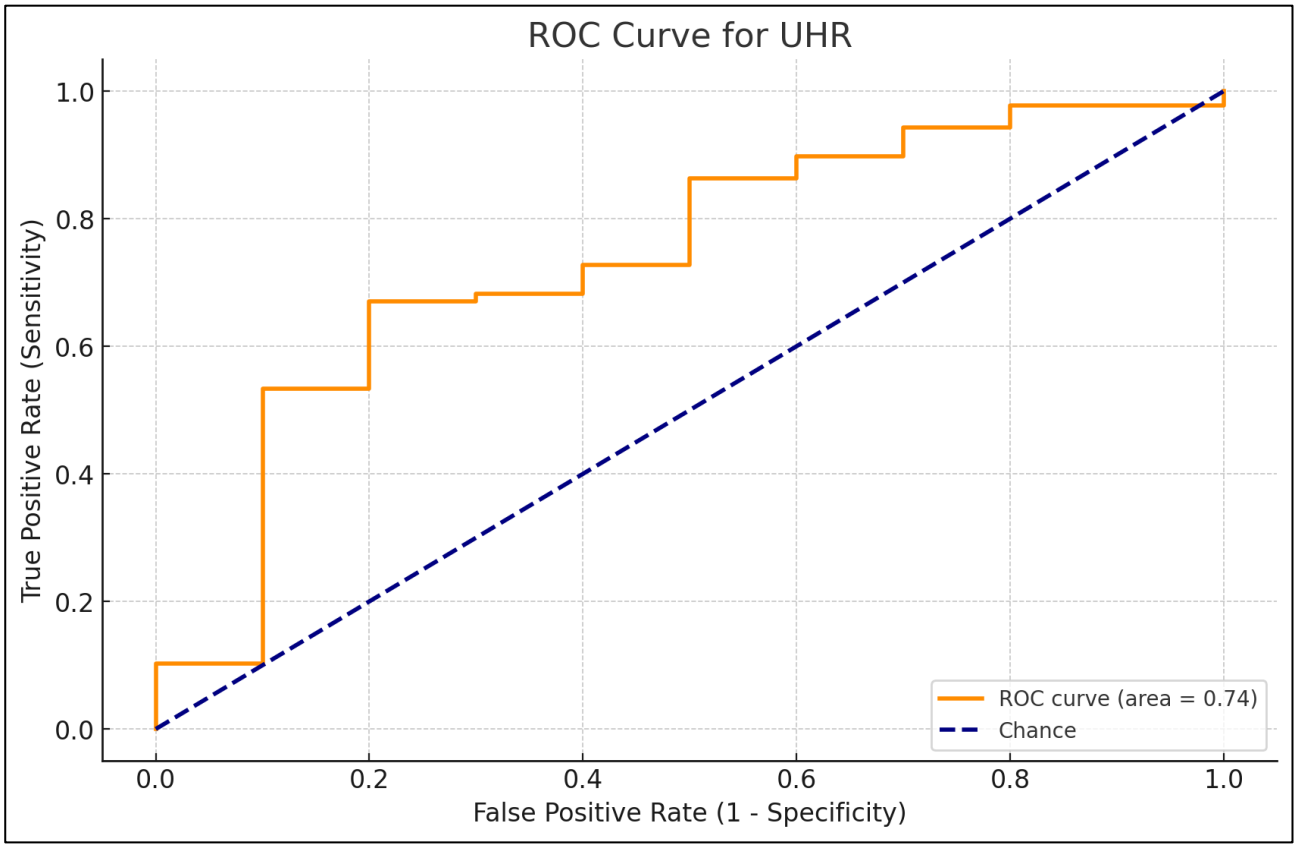

Figure 1. ROC curve of UHR in predicting the presence of Obstructive Sleep Apnea Syndrome (OSAS). The area under the curve (AUC) was 0.74 (95% CI: 0.65–0.83, $p < 0.001$).

The multinomial logistic regression analysis showed that age and gender had a significant effect on the severity of the syndrome. Older age had relation with more disease severity, and male patients were more common in the severe groups. UHR and MHR had limited predictive power in our model. However, UHR showed a borderline significant association with severe cases ($p = 0.082$), which may indicate a possible relationship.

Discussion

Our study showed a significant association between UHR and OSAS, which suggests that UHR may be a useful predictive marker. We also found that MHR and the mean heart rate during sleep were related to OSAS.

UHR has been investigated in different diseases, especially in cardiovascular conditions, and several studies have reported significant relations disease and marker (16,17). There was no study investigating the relationship between OSAS and UHR, but there were studies evaluating uric acid and lipids separately. While one study found significant changes in lipid parameters in OSAS patients, another study reported significant alterations in uric acid levels (6,18). Given the strong association of OSAS with metabolic syndrome and obesity, a relationship with lipid parameters was certain, leading us to consider that adding an additional parameter could enhance its specificity. Uric acid is known to rise in conditions like gout, kidney problems, high purine intake, too much alcohol use, increased cell breakdown, and also in intermittent hypoxia. To date, there had been no widely accepted biomarker specifically utilized for the diagnosis or severity prediction of disease. Our findings showed the potential of UHR. The fact that the UHR test showed a strong specificity of up to 90% at a cut-off value of 0.123 also increased its value. It's inexpensive and easy to measure. In the regression analysis, UHR did not show strong predictive power for disease severity, but it had a borderline significant association with severe cases. Future studies should test UHR in larger and more diverse patient groups.

Monocytes and HDL have been previously studied in patients, particularly due to their role in the pathophysiology of atherosclerosis, and a significant association between the MHR and OSAS has been demonstrated (19,20). Consistent with these findings, we also observed a meaningful link between severity and this ratio.

Mean heart rate during sleep apnea provides valuable information about cardiovascular responses. One of the significant findings of our study was the variation in mean heart rate during sleep across different OSAS severity levels. A previous study reported that heart rate variability (HRV), known as an indicator of the autonomic nervous system, may be beneficial in patients with OSAS (21). Recurrent episodes of hypoxia and awakening trigger sympathetic activation, leading to an increase in mean heart rate (22). Although mean heart rate alone is not sufficient, its evaluation together with other parameters can increase the diagnostic value.

Our study gives useful information about the diagnostic value of UHR, but it also has some limitations. While the sample size is sufficient, the results may not be consistent across different populations. Because the study is cross-sectional, we cannot establish a causal relationship between UHR and OSAS. Furthermore, we were unable to fully control for factors such as diet, medications, and other diseases that may influence UHR levels.

Data Availability Statement: The data supporting the findings of this study are available from the corresponding author, Dr. Sadettin Ersoy, upon reasonable request. Due to ethical and privacy concerns, the data are not publicly accessible.

Ethics Committee Approval: The study was approved by the Bolu Abant Izzet Baysal University Non-Interventional Clinical Research Ethics Committee Approval (date: 04.06.2024 and approval number: 2024/132).

Informed Consent: Written consent was obtained from the participants.

Conflict of Interest: Authors declared no conflict of interest.

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References

1. Soori R, Baikunje N, D'sa I, Bhushan N, Nagabhushana B, Hosmane GB. Pitfalls of AHI system of severity grading in obstructive sleep apnoea. Sleep Sci. 2022;15(Spec 1):285-8.
2. Chang JL, Goldberg AN, Alt JA, Mohammed A, Ashbrook L, Auckley D, et al. International Consensus Statement on Obstructive Sleep Apnea. Int Forum Allergy Rhinol. 2023;13(7):1061-482.
3. Imani MM, Sadeghi M, Farokhzadeh F, Khazaie H, Brand S, Dürsteler KM, et al. Evaluation of Blood Levels of C-Reactive Protein Marker in Obstructive Sleep Apnea: A Systematic Review, Meta-Analysis and Meta-Regression. Life (Basel). 2021;11(4).
4. Lavie L. Oxidative stress inflammation and endothelial dysfunction in obstructive sleep apnea. Frontiers in Bioscience-Elite. 2012;4(4):1391-403.
5. Sahebjami H. Changes in urinary uric acid excretion in obstructive sleep apnea before and after therapy with nasal continuous positive airway pressure. Chest. 1998;113(6):1604-8.
6. Hirotsu C, Tufik S, Guindalini C, Mazzotti DR, Bittencourt LR, Andersen ML. Association between uric acid levels and obstructive sleep apnea syndrome in a large epidemiological sample. PLoS One. 2013;8(6):e66891.
7. Nadeem R, Molnar J, Madbouly EM, Nida M, Aggarwal S, Sajid H, et al. Serum inflammatory markers in obstructive sleep apnea: a meta-analysis. J Clin Sleep Med. 2013;9(10):1003-12.
8. Shahin L, Patel KM, Heydari MK, Kesselman MM. Hyperuricemia and Cardiovascular Risk. Cureus. 2021;13(5):e14855.
9. Pływaczewski R, Bednarek M, Jonczak L, Górecka D, Sliwiński P. [Hyperuricaemia in males with obstructive sleep apnoea (osa)]. Pneumonol Alergol Pol. 2005;73(3):254-9.

10. García AR, Armengol ÁS, Crespo EL, Aguilar DG, Falcön AR, Bernal CC, et al. Blood uric acid levels in patients with sleep-disordered breathing. Archivos de Bronconeumología ((English Edition)). 2006;42(10):492-500.
11. Benjafield AV, Ayas NT, Eastwood PR, Heinzer R, Ip MSM, Morrell MJ, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. The Lancet Respiratory Medicine. 2019;7(8):687-98.
12. Qian Y, Xu H, Wang Y, Yi H, Guan J, Yin S. Obstructive sleep apnea predicts risk of metabolic syndrome independently of obesity: a meta-analysis. Archives of Medical Science. 2016;12(5):1077-87.
13. Xu S, Wan Y, Xu M, Ming J, Xing Y, An F, et al. The association between obstructive sleep apnea and metabolic syndrome: a systematic review and meta-analysis. BMC Pulmonary Medicine. 2015;15(1):105.
14. Aktas G, Kocak MZ, Bilgin S, Atak BM, Duman TT, Kurtkulagi O. Uric acid to HDL cholesterol ratio is a strong predictor of diabetic control in men with type 2 diabetes mellitus. Aging Male. 2020;23(5):1098-102.
15. Kolahi Ahari R, Mansoori A, Sahranavard T, Miri MS, Feizi S, Esmaily H, et al. Serum uric acid to high-density lipoprotein ratio as a novel indicator of inflammation is correlated with the presence and severity of metabolic syndrome: A large-scale study. Endocrinol Diabetes Metab. 2023;6(6):e446.
16. Yang Y, Zhang J, Jia L, Su J, Ma M, Lin X. Uric acid to high-density lipoprotein cholesterol ratio predicts adverse cardiovascular events in patients with coronary chronic total occlusion. Nutr Metab Cardiovasc Dis. 2023;33(12):2471-8.
17. Park B, Jung DH, Lee YJ. Predictive Value of Serum Uric Acid to HDL Cholesterol Ratio for Incident Ischemic Heart Disease in Non-Diabetic Koreans. Biomedicines. 2022;10(6).
18. Popadic V, Brajkovic M, Klasnja S, Milic N, Rajovic N, Lisulov DP, et al. Correlation of Dyslipidemia and Inflammation With Obstructive Sleep Apnea Severity. Front Pharmacol. 2022;13:897279.
19. Inonu Koseoglu H, Pazarli AC, Kanbay A, Demir O. Monocyte Count/HDL Cholesterol Ratio and Cardiovascular Disease in Patients With Obstructive Sleep Apnea Syndrome: A Multicenter Study. Clin Appl Thromb Hemost. 2018;24(1):139-44.
20. Atan D, Kundi FCS, Özcan KM, Dere H. A New Predictor for Obstructive Sleep Apnea Syndrome: Monocyte to HDL Ratio. Indian J Otolaryngol Head Neck Surg. 2017;69(2):142-6.
21. Ucak S, Dissanayake HU, Sutherland K, de Chazal P, Cistulli PA. Heart rate variability and obstructive sleep apnea: Current perspectives and novel technologies. J Sleep Res. 2021;30(4):e13274.
22. Naeije R. Physiological adaptation of the cardiovascular system to high altitude. Prog Cardiovasc Dis. 2010;52(6):456-66.