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The Uric Acid/HDL-Cholesterol Ratio as a Potential Marker of Obstruction in Hypertrophic Cardiomyopathy

Hipertrofik Kardiyomiyopatide Obstrüksiyonun Olası Bir Belirteci Olarak Ürik Asit/HDL-Kolesterol Oranı

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ABSTRACT

Aim: The uric acid to high-density lipoprotein cholesterol ratio (UHR) has recently emerged as a composite metabolic–inflammatory marker linked to cardiovascular risk. However, its relevance in hypertrophic cardiomyopathy (HCM), particularly in distinguishing obstructive (HOCM) from non-obstructive (HNCM) forms, remains unknown. We aimed to investigate whether UHR differs between HOCM, HNCM, and healthy controls, and to determine its association with echocardiographic and inflammatory markers, as well as its diagnostic performance for identifying obstructive HCM.

Methods: This retrospective study included 200 participants (60 HOCM, 80 HNCM, 60 controls). Clinical, biochemical, and echocardiographic data were analyzed. UHR was calculated as serum uric acid (mg/dL) divided by HDL-C (mg/dL). Correlation and receiver operating characteristic (ROC) analyses were performed.

Results: UHR was significantly higher in HOCM than in HNCM and controls ($p<0.001$). It strongly correlated with resting ($r=0.741$) and provoked ($r=0.729$) LVOT gradients, and moderately with CRP, septal thickness, and left atrial diameter. ROC analysis showed high diagnostic accuracy for detecting obstruction (AUC = 0.916, cut-off = 0.145, sensitivity 83.3%, specificity 85.0%).

Conclusions: UHR, reflecting the balance between oxidative stress and antioxidant capacity, is significantly elevated in obstructive HCM and correlates with disease severity. This easily obtainable marker may assist in phenotypic differentiation, risk stratification, and potentially in monitoring disease progression.

Keywords: hypertrophic cardiomyopathy, obstruction, uric acid, HDL cholesterol, oxidative stress, biomarker

ÖZ

Amaç: Ürik asit / yüksek yoğunluklu lipoprotein kolesterol oranı (UHR), kardiyovasküler riskle ilişkili metabolik-inflamatuvar bir belirteç olarak tanımlanmıştır. Ancak bu oranın hipertrofik kardiyomiyopati (HKM) alt tipleri arasındaki farkı yansıtıp yansıtmadığı bilinmemektedir. Bu çalışmada, UHR'nin hipertrofik obstrüktif kardiyomyopati (HOKM), hipertrofik non-obstrüktif kardiyomiyopati (HNKM) ve sağlıklı bireyler arasında farklılık gösterip göstermediğini; ekokardiyografik ve inflamatuvar parametrelerle ilişkisini ve HOKM'yi ayırt etmedeki tanısal performansını değerlendirmeyi amaçladık.

Yöntemler: Retrospektif olarak 60 HOKM, 80 HNKM ve 60 sağlıklı kontrol olmak üzere toplam 200 katılımcı incelendi. Klinik, biyokimyasal ve ekokardiyografik veriler analiz edildi. UHR, serum ürik asit (mg/dL) / HDL-kolesterol (mg/dL) oranı olarak hesaplandı. Korelasyon ve ROC analizleri yapıldı.

Bulgular: UHR, HOKM grubunda HNKM ve kontrol gruplarına göre anlamlı derecede yüksekti ($p<0.001$). UHR, istirahat ($r=0.741$) ve provokasyon ($r=0.729$) LVOT gradyanlarıyla güçlü, CRP, septal kalınlık ve sol atriyum çapı ile orta düzeyde korelasyon gösterdi. ROC analizinde UHR'nin HOKM'yi saptamadaki tanısal gücü yüksekti (AUC = 0.916, cut-off = 0.145, duyarlılık %83.3, özgüllük %85.0).

Sonuç: UHR, oksidatif stres ile antioksidan kapasite arasındaki dengeyi yansıtan bir belirteç olup, obstrüktif hipertrofik kardiyomiyopatide anlamlı olarak artmıştır ve hastalık şiddeti ile korelasyon göstermektedir. Kolayca elde edilebilen bu belirteç, fenotipik ayrımda, risk sınıflamasında ve potansiyel olarak hastalık progresyonunun izlenmesinde yardımcı olabilir.

Anahtar Kelimeler: hipertrofik kardiyomiyopati, obstrüksiyon, ürik asit, HDL kolesterol, oksidatif stres, biyobelirteç

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Introduction

Hypertrophic cardiomyopathy (HCM) is a genetic myocardial disorder characterized by left ventricular hypertrophy and variable clinical presentations, ranging from asymptomatic to severe outflow obstruction. The obstructive form (HOCM) is distinguished by dynamic left ventricular outflow tract (LVOT) pressure gradients and often presents with more aggressive symptoms and worse prognosis [1]. Identifying biomarkers that differentiate obstructive from non-obstructive phenotypes is clinically valuable for risk stratification and management.

Uric acid, a purine metabolism byproduct, has been implicated in oxidative stress, endothelial dysfunction, and inflammation. Elevated serum uric acid levels have been associated with adverse cardiovascular outcomes, left ventricular hypertrophy, and heart failure [2]. On the other hand, high-density lipoprotein cholesterol (HDL-C) exerts anti-inflammatory and antioxidant effects. The ratio of uric acid to HDL (UHR) has recently emerged as a combined marker integrating metabolic and inflammatory risk, showing promise in predicting metabolic syndrome, coronary artery disease, and other cardiovascular conditions [3–5].

Recent population-based studies have revealed associations between increased UHR and cardiovascular risk factors. Yi et al. reported that UHR was independently associated with risk of cardiovascular disease and dyslipidemia in a large cohort [4]. Similarly, Li et al. demonstrated that UHR predicted outcomes beyond serum uric acid or HDL alone [3]. Nevertheless, to date, no study has evaluated UHR in the setting of HCM or its relation to outflow tract obstruction. We hypothesized that elevated UHR is associated with increased LVOT gradient and may differentiate obstructive from non-obstructive forms of HCM.

In this study, we aimed to assess whether UHR differs between obstructive and non-obstructive HCM patients and healthy controls, and to explore the correlation of UHR with LVOT gradients, septal thickness, and inflammatory markers. Additionally, we sought to determine the discriminative performance of UHR via ROC analysis in identifying obstructive HCM.

Methods

This retrospective cross-sectional study included a total of 200 participants who were evaluated at the cardiology outpatient clinic between January 2017 and December 2024. The study population consisted of 80 patients with HNCM, 60 patients with HOCM, and 60 age- and sex-matched healthy controls. The diagnosis of HCM was established according to the current European Society of Cardiology (ESC) guidelines, defined as a left ventricular wall thickness ≥ 15 mm in the absence of another cardiac or systemic cause of hypertrophy [1]. Obstructive HCM was defined as a resting LVOT gradient ≥ 30 mmHg or a provoked gradient ≥ 50 mmHg. Patients with secondary causes of hypertrophy (e.g., hypertension, valvular disease, amyloidosis), renal failure (eGFR <60 mL/min/1.73 m²), gout, hepatic dysfunction, active infection, or inflammatory diseases were excluded.

Additionally, patients using medications known to affect serum uric acid or HDL-cholesterol levels (such as xanthine oxidase inhibitors, statins, niacin, fibrates, or diuretics) were excluded from the study.

Demographic and clinical characteristics, including age, sex, body mass index (BMI), blood pressure, and medication use, were collected from electronic medical records. Laboratory data such as fasting plasma glucose, serum uric acid, HDL-cholesterol, creatinine, and C-reactive protein (CRP) were obtained from the hospital's biochemistry database. The UHR was calculated as serum uric acid (mg/dL) divided by HDL-cholesterol (mg/dL). Estimated glomerular filtration rate (eGFR) was determined using the CKD-EPI formula.

All echocardiographic data were retrieved from digital archives of examinations performed using a Philips EPIQ7 system (Philips Medical Systems, Andover, MA, USA) with a 2–4 MHz transducer. Measurements were performed in accordance with the European Association of Cardiovascular Imaging (EACVI) recommendations [6]. Measurements included interventricular septal thickness, posterior wall thickness, left ventricular ejection fraction (LVEF), and left atrial diameter (LAD). LVOT gradients were recorded at rest and during Valsalva provocation using continuous-wave Doppler. The presence of systolic anterior

motion (SAM) of the mitral valve and LVOT obstruction were documented.

The study protocol was approved by the institutional ethics committee (Approval No: 782; 28.08.2025) and conducted in accordance with the Declaration of Helsinki. Because this was a retrospective analysis of anonymized data, the requirement for written informed consent was waived by the ethics committee.

Statistical Analysis: All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as numbers and percentages. The distribution of continuous variables was evaluated using the Shapiro–Wilk test, and homogeneity of variances was tested with Levene's test. Comparisons among the three groups (HOCM, HNCM, controls) were performed using one-way ANOVA, followed by Tukey's post-hoc test for pairwise comparisons. When normality or variance assumptions were not met, Kruskal–Wallis and Dunn–Bonferroni tests were applied. Differences in categorical variables were analyzed using the chi-square test or Fisher's exact test as appropriate. Correlations between UHR and other parameters (CRP, septal thickness, LVOT gradients, and LAD) were examined using Pearson's correlation analysis (two-tailed). The diagnostic performance of UHR for predicting obstructive HCM was evaluated using receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) and 95% confidence intervals (CI) were reported. The optimal cut-off value was determined using Youden's index. To minimize type I error due to multiple testing, Bonferroni correction was applied during post-hoc pairwise comparisons following ANOVA or Kruskal–Wallis analyses, where appropriate. A two-tailed $p < 0.05$ was considered statistically significant.

Results

A total of 200 participants were included in the study: 60 patients with HOCM, 80 with HNCM, and 60 healthy controls. Demographic and clinical characteristics are summarized in Table 1. There were no significant differences among the groups in terms of age or sex distribution (p=0.111 and p=0.470, respectively). However, the prevalence of hypertension and diabetes mellitus was significantly higher in both HCM groups compared with controls (p<0.001 for both). A family history of HCM was reported in 26.3% of the HNCM group and 41.7% of the HOCM group, but only in 3.3% of controls (p<0.001). The use of β-blocker therapy was also markedly more frequent among HCM patients, particularly in the HOCM group (93.3%) compared with HNCM (65.0%) and controls (11.7%) (p<0.001). BMI, fasting plasma glucose, and CRP levels were significantly higher in both HCM groups compared with the control group (all p<0.001). Serum uric acid levels were elevated, while HDL-cholesterol levels were lower in HOCM and HNCM groups compared with controls, resulting in a marked increase in the UHR across the study groups (p<0.001). Figure 1 illustrates the distribution of UHR values among HOCM, HNCM, and control groups. Post-hoc analysis indicated that UHR was highest in the HOCM group, intermediate in the HNCM group, and lowest in controls (p<0.001 for all pairwise comparisons).

Echocardiographic parameters are summarized in Table 2. Both HOCM and HNCM groups demonstrated significantly increased interventricular septal thickness and posterior wall thickness compared to controls (p<0.001). Septal thickness was greater in the obstructive group than in the non-obstructive group (p<0.001). Left atrial diameter was significantly enlarged in both HCM groups (p<0.001). Resting and provoked LVOT gradients were markedly higher in HOCM patients than in HNCM (55.2 ± 10.6 mmHg vs. 17.2 ± 4.7 mmHg, and 69.2 ± 11.5 mmHg vs. 23.0 ± 4.6 mmHg, respectively; both p<0.001). No significant difference was observed in LVEF between the groups (p=0.406).

Correlation coefficients between UHR and echocardiographic as well as inflammatory parameters are presented in Table 3 and illustrated in Figure 2. UHR showed a strong positive correlation with LVOT gradient at rest (r=0.741, p<0.001) and during provocation (r=0.729, p<0.001). Moderate positive correlations were also found between UHR and CRP (r=0.472, p<0.001) and septal thickness (r=0.354, p<0.001). A weak but significant correlation was noted

Table 1. Baseline demographic, clinical, and laboratory characteristics of the study population

Variable	Control (n=60)	HNCM (n=80)	HOCM (n=60)	p-value
Age (years)	53.4 ± 5.6	52.2 ± 5.9	51.1 ± 6.2	0.111
Male sex, n (%)	31 (51.7)	43 (53.8)	35 (58.3)	0.470
BMI (kg/m²)	23.8 ± 0.9	26.1 ± 2.3	27.2 ± 2.4	<0.001
FPG (mg/dL)	89.7 ± 4.6	101.1 ± 17.1	103.3 ± 17.5	<0.001
Serum uric acid (mg/dL)	4.83 ± 0.76	5.53 ± 0.97	5.99 ± 0.71	<0.001
HDL-C (mg/dL)	38.8 ± 7.6	47.2 ± 9.7	37.5 ± 6.0	<0.001
UHR	0.126 ± 0.013	0.120 ± 0.022	0.162 ± 0.017	<0.001
CRP (mg/L)	3.8 ± 1.6	4.8 ± 2.3	7.8 ± 2.9	<0.001
Creatinine (mg/dL)	0.90 ± 0.06	0.94 ± 0.13	0.91 ± 0.11	0.061
GFR (mL/min/1.73 m²)	82.1 ± 11.7	77.7 ± 14.2	81.0 ± 14.8	0.145
Hypertension, n (%)	12 (20.0)	41 (51.3)	36 (60.0)	<0.001
Diabetes mellitus, n (%)	6 (10.0)	19 (23.8)	22 (36.7)	<0.001
Family history of HCM, n (%)	2 (3.3)	21 (26.3)	25 (41.7)	<0.001
Beta-blocker therapy, n (%)	7 (11.7)	52 (65.0)	56 (93.3)	<0.001

Values are presented as mean ± standard deviation (SD) or number (percentage).

HOCM: hypertrophic obstructive cardiomyopathy; HNCM: hypertrophic non-obstructive cardiomyopathy; BMI: body mass index; FPG: fasting plasma glucose eGFR: estimated glomerular filtration rate; CRP: C-reactive protein; HDL-C: high-density lipoprotein cholesterol; UHR: uric acid/HDL-cholesterol ratio. p-values derived from one-way ANOVA or chi-square test, as appropriate.

Table 2. Echocardiographic parameters of the study groups

Variable	Control (n=60)	HNCM (n=80)	HOCM (n=60)	p-value
Septal thickness (mm)	9.6 ± 1.1	17.9 ± 2.3	18.9 ± 2.1	<0.001
Posterior wall thickness (mm)	9.6 ± 1.1	13.4 ± 1.4	14.2 ± 1.5	<0.001
Left atrial diameter (mm)	32.7 ± 1.5	35.3 ± 3.6	36.6 ± 4.5	<0.001
LVOT gradient (rest, mmHg)	—	17.2 ± 4.7	55.2 ± 10.6	<0.001
LVOT gradient (provocation, mmHg)	—	23.0 ± 4.6	69.2 ± 11.5	<0.001
LVEF (%)	65.3 ± 2.8	65.1 ± 3.4	64.6 ± 3.6	0.406

Values are presented as mean ± SD.

LVOT: left ventricular outflow tract; LVEF: left ventricular ejection fraction.

p-values calculated by one-way ANOVA; post-hoc comparisons performed using Tukey's test.

Table 3. Correlation between UHR and echocardiographic and inflammatory parameters

Variable	Pearson correlation coefficient (r)	p-value
LVOT gradient (rest)	0.741	<0.001
LVOT gradient (provocation)	0.729	<0.001
CRP	0.472	<0.001
Septal thickness	0.354	<0.001
Left atrial diameter	0.294	<0.001
Left ventricular ejection fraction	0.056	0.406

Pearson's correlation analysis (two-tailed).

LVOT: left ventricular outflow tract; CRP: C-reactive protein; UHR: uric acid HDL-cholesterol ratio.

between UHR and left atrial diameter (r=0.294, p<0.001), while no significant association was found with LVEF (p>0.05).

ROC analysis demonstrated that UHR had high diagnostic accuracy for identifying HOCM (Figure 3). The area under the curve (AUC) was 0.916 (95% CI 0.871–0.962, p<0.001). The optimal cut-off value of 0.145 yielded a sensitivity of 83.3% and a specificity of 85.0% for predicting the obstructive phenotype.

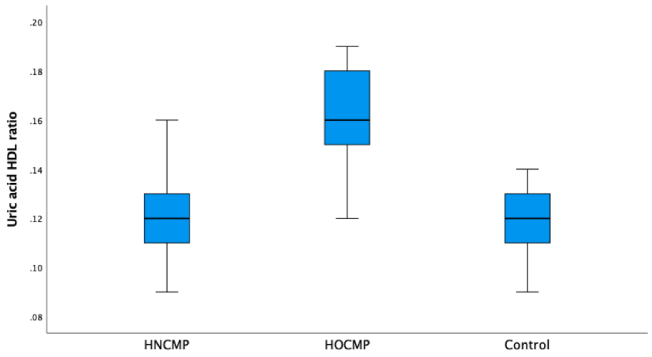

Figure 1. Distribution of UHR levels among study groups. Boxplot illustrating the distribution of UHR values in patients with obstructive HOCM, HNCM, and healthy controls. Mean UHR levels were significantly higher in the HOCM group compared to both HNCM and controls (p<0.001, one-way ANOVA with Tukey post-hoc analysis).

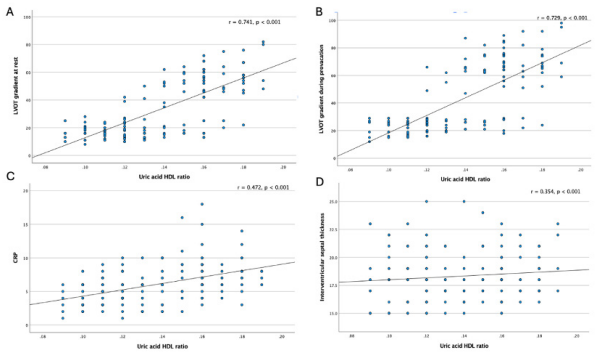

Figure 2. Correlation analyses between UHR and echocardiographic and inflammatory parameters. Scatter plots with regression lines show the relationships between UHR and: (A) LVOT gradient at rest (r=0.741, p<0.001), (B) LVOT gradient during provocation (r=0.729, p<0.001), (C) CRP (r=0.472, p<0.001), and (D) interventricular septal thickness (r=0.354, p<0.001). All correlations were positive and statistically significant (Pearson's correlation analysis, two-tailed).

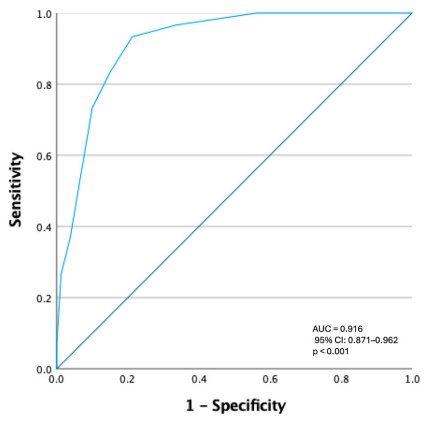

Figure 3: ROC curve of UHR for identifying obstructive hypertrophic cardiomyopathy. The ROC analysis demonstrated excellent diagnostic accuracy of UHR in differentiating obstructive from non-obstructive HCM, with an AUC of 0.916 (95% CI: 0.871–0.962, p<0.001). The optimal cut-off value of 0.145 yielded 83.3% sensitivity and 85.0% specificity.

Discussion

In this study, we demonstrated that patients with obstructive HOCM exhibit significantly higher UHR compared with both HNCM patients and healthy controls. Moreover, UHR was strongly correlated with resting and provoked LVOT gradients, and moderately correlated with septal wall thickness, CRP, and left atrial diameter. ROC analysis showed that UHR had high discriminative ability (AUC = 0.916) for identifying obstructive HCM, with a cut-off of 0.145 yielding high sensitivity and specificity.

Elevated uric acid and reduced HDL levels are known to contribute to endothelial dysfunction, inflammation, and oxidative stress. The UHR has recently emerged as a combined metabolic–inflammatory index, reflecting increased oxidative burden and diminished antioxidant defense. Our findings extend this concept to HCM patients, suggesting that metabolic and inflammatory changes may be linked to LVOT obstruction severity. Previous population-based studies have shown that higher UHR levels are associated with increased cardiovascular risk and dyslipidemia [4]. Similarly, studies in hypertensive cohorts reported elevated UHR in patients with poor blood pressure control and correlations with metabolic parameters such as BMI and eGFR [7].

Recent data also support broader vascular implications of UHR. Jiang et al. reported that higher UHR levels in an elderly population were associated with increased stroke risk, and that UHR outperformed uric acid or HDL-C alone in discriminating cerebrovascular risk [8]. This suggests that UHR reflects systemic vascular stress and may relate to LVOT obstruction through oxidative and endothelial dysregulation.

Yang et al. showed in patients with acute myocardial infarction that high uric acid combined with low HDL-C was independently associated with increased mortality and major adverse cardiovascular events, and UHR served as a reliable prognostic marker (AUC = 0.716) [9]. This interaction supports the idea that UHR may

actively reflect oxidative stress and reduced antioxidant protection. The higher discriminative performance of UHR in our HCM cohort (AUC = 0.916) further supports its potential diagnostic and prognostic value in identifying patients with more severe obstruction.

In HCM, previous research has indicated that higher serum uric acid predicts adverse outcomes. Gao et al. reported that elevated uric acid predicted cardiac death in patients with obstructive HCM managed conservatively [10]. Mechanistically, uric acid acts both as a marker and mediator of oxidative stress via xanthine oxidase activity, generating reactive oxygen species, consuming nitric oxide, and promoting endothelial dysfunction. Meanwhile, HDL exerts protective effects through antioxidant, anti-inflammatory, and reverse cholesterol transport mechanisms. Thus, UHR may reflect the balance between pro-oxidant and antioxidant forces, explaining its association with LVOT obstruction and myocardial remodeling.

Evidence from long-term cohort studies further highlights UHR's prognostic relevance beyond cardiac disease. Elevated UHR has been linked to higher cardiovascular mortality [12] and increased incidence of ischemic heart disease [13]. This suggests that UHR integrates chronic metabolic and vascular stress with clinical outcomes.

A major strength of our study is the use of complementary analytical approaches group comparisons, correlation matrices, and ROC analysis to provide a comprehensive evaluation of UHR in HCM. The consistent associations, especially with LVOT gradients, support that UHR is more than a coincidental marker.

Limitations: Several limitations should be acknowledged. First, the cross-sectional and retrospective design may introduce selection bias, and causality cannot be established. Second, our single-center sample, although adequately powered, may limit generalizability to other populations. Third, we did not measure additional metabolic or inflammatory biomarkers such as interleukins, NT-proBNP, or oxidative stress enzymes, which could provide deeper mechanistic insights. Fourth, the ROC-derived cut-off value (0.145) requires validation in larger, prospective HCM cohorts before clinical application. Finally, while Pearson correlations were used, potential deviations from normality may have slightly influenced correlation estimates.

Conclusion

Our findings indicate that UHR may serve as a cost-effective and easily obtainable biomarker for identifying patients with severe, obstructive HCM. Given its strong correlation with LVOT gradients, UHR could be useful for monitoring disease severity, disease progression or response to therapy. Future prospective studies with larger cohorts are needed to validate these findings and to determine whether modifying uric acid or HDL metabolism can influence HCM outcomes. Exploring the mechanistic interplay between UHR, oxidative stress, microvascular dysfunction, and myocardial fibrosis may further clarify its role in HCM pathophysiology.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Ethics Committee Approval: This research complies with all the relevant national regulations, institutional policies and is in accordance with the tenets of the Helsinki Declaration, and has been approved by the Medical Faculty Ethical Committee, Akdeniz University (Approval Number: TBAEK-782, Date: 28.08.2025).

Informed Consent: Because the analysis was retrospective and patient data were anonymized, the requirement for written informed consent was waived.

Author Contributions: A.Ü.K. (0000-0002-8930-3651) and **T.K. (0000-0002-0838-2037) :** All authors contributed to all stages. All authors read and approved the final manuscript.

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REFERENCES

1. Arbelo E, Protonotarios A, Gimeno JR, Arbustini E, Barriales-Villa R, Basso C, et al. 2023 ESC Guidelines for the management of cardiomyopathies. Eur Heart J. 2023;44(37):3503–626. doi:10.1093/eurheartj/ehad194.
2. Ndrepepa G. Uric acid and cardiovascular disease. Clin Chim Acta. 2018;484:150–63. doi: 10.1016/j.cca.2018.05.046.
3. Li F, Zhao D, Li Q, Lin X, Sun H, Fan Q. Uric Acid to High-Density Lipoprotein Cholesterol Ratio is a Novel Marker to Predict Functionally Significant Coronary Artery Stenosis. J Interv Cardiol. 2022;2022:9057832. doi: 10.1155/2022/9057832.
4. Yi Y, Luo Q, Chen J, Chen Z, Aydemir HA, Chen P, et al. Association between the uric acid-to-HDL-cholesterol ratio (UHR) and the risk of cardiovascular disease

and dyslipidemia: a population-based study. Lipids Health Dis.2025;24(1):143. doi: 10.1186/s12944-025-02551-4.

5. Li Z, Liu Q, Yao Z. The serum uric acid-to-high-density lipoprotein cholesterol ratio is a predictor for all-cause and cardiovascular disease mortality: a cross-sectional study. Front Endocrinol (Lausanne). 2024;15:1417485. doi: 10.3389/fendo.2024.1417485.
6. Galderisi M, Cosyns B, Edvardsen T, Cardim N, Delgado V, Di Salvo G, et al. Standardization of adult transthoracic echocardiography reporting in agreement with recent chamber quantification, diastolic function, and heart valve disease recommendations: an expert consensus document of the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2017;18(12):1301–10. doi: 10.1093/ehjci/jex244.
7. 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. doi: 10.1002/edm2.446.
8. Jiang Q, Zhan G, Liu Y, Jiang C, Wang K, Zheng G, et al. Serum uric acid to high-density lipoprotein cholesterol ratio is associated with stroke in the elderly: a population based study. Front Neurol. 2025;16:1594080. doi: 10.3389/fneur.2025.1594080.
9. Yang Y, Zhang J, Jia L, Su J, Ma M, Lin X. The interaction between uric acid and high-density lipoprotein cholesterol on the prognosis of patients with acute myocardial infarction. Front Cardiovasc Med. 2023;10:1226108. doi: 10.3389/fcvm.2023.1226108.
10. Gao J, Shao CL, Meng XB, Wang WY, Zhang K, Wang JJ, et al. Uric acid is associated with cardiac death in patients with hypertrophic obstructive cardiomyopathy. J Geriatr Cardiol. 2021;18(4):281–8. doi: 10.11909/j.issn.1671-5411.2021.04.006.
11. Packer M. Uric acid is a biomarker of oxidative stress in the failing heart: Lessons learned from trials with allopurinol and SGLT2 inhibitors. J Card Fail. 2020;26(11):977–84. doi: 10.1016/j.cardfail.2020.08.015.
12. Cui Y, Zhang W. Long-term cardiovascular risk and mortality associated with uric acid to HDL-C ratio: a 20-year cohort study in adults over 40. Sci Rep. 2025;15(1):14242. doi: 10.1038/s41598-025-99205-3.
13. Ou Y, Qin Z, Wang P, Zou F. Serum uric acid to high-density lipoprotein cholesterol ratio predicts all-cause mortality in adults with metabolic dysfunction-associated steatotic liver disease. Sci Rep. 2025;15(1):11278. doi: 10.1038/s41598-025-94651-5.