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Composite Uric Acid Index for Prediction of Significant Coronary Stenosis on Computed Tomography Angiography

ABSTRACT

Objective: A recently proposed uric acid index combining fasting glucose, triglycerides, and uric acid has shown promise in cardiovascular risk stratification. It was assessed whether the index could predict significant coronary artery stenosis (≥50%) on coronary computed tomography (CT) angiography and compared its diagnostic performance with serum uric acid.

Methods: In this retrospective, single-center study, 258 adults who underwent coronary CT angiography between January 2023 and December 2024 were included. Coronary calcification was scored using the Agatston method by 2 cardiologists. Clinical, demographic, and laboratory data were collected. The uric acid index was calculated as Ln [triglycerides × uric acid × glucose/2]. Univariable and hierarchical multiple logistic regression analyses were performed.

Results: Seventy-one patients (27.5%) showed ≥50% stenosis. Those with stenosis were older and had higher Agatston scores, creatinine, uric acid, glucose, HbA1c, and lower high-density lipoprotein cholestrol (HDL-C). In the final multiple models adjusted for age, sex, creatinine and Agatston score, the uric acid index remained a strong independent predictor of ≥50% stenosis (odds ratio [OR] = 1.988, $P = .019$, 95% confidence interval [CI] = 1.117-3.538). The index demonstrated significant incremental predictive value over the base model, with a Net Reclassification Improvement (NRI) of 0.500 ($P < .05$) and an Integrated Discrimination Improvement (IDI) of 0.029. The area under the curve for the uric acid index (0.688) exceeded that of uric acid (0.664).

Conclusion: In a real-world coronary CT angiography cohort, the composite uric acid index independently predicted significant coronary stenosis. Its incremental predictive value requires validation in larger, prospective studies.

Keywords: Biomarkers, coronary stenosis, CT angiography, uric acid

INTRODUCTION

Ischemic heart disease remains a leading global cause of mortality, posing a substantial public health challenge. Obesity, diabetes, and hypertension—interconnected metabolic and genetic trends—are progressively driving the rise in prevalence.[1] As these upstream risk factors continue to grow, future trends in ischemic disease may worsen, emphasizing the necessity for more accurate predictive metabolic biomarkers.[2] Recent global data indicate an increasing prevalence of cardiovascular disease in individuals who appear healthy and asymptomatic and may harbor risk, thereby emphasizing the necessity for early risk assessment.[3]

Serum levels of triglycerides, glucose, and uric acid, which are interlinked through shared metabolic pathways, have been shown to predict cardiovascular risk more accurately when integrated into composite indices rather than assessed individually.[4,5] A novel uric acid index incorporating glucose, triglycerides, and uric acid demonstrates enhanced predictive capability for cardiovascular risk, exceeding the predictive value of its individual components.[6] However, it remains unclear whether this combined index offers superior predictive value over serum uric acid alone in identifying clinically significant coronary artery lesions. The present study therefore aims to assess the independent predictive power of the

ORIGINAL INVESTIGATION

Volkan Çamkıran[1]
Gediz Doğay Us[2,3]
Eray Aksoy[4]
Şahhan Kılıç[5]
Gülsüm Bingöl[6]
Leyla Bulut[7]
Haşim Tüner[8]
Elif Ayduk Gövdeli[9]
Ümit Acar[10]
Batool Achmar[11]
Barış Ökçün[10]
Melih Us[3]

[1]*Department of Cardiology, Faculty of Medicine, İstinye University, İstanbul, Türkiye*
[2]*Department of Cardiovascular Disease, School of Nutrition and Translational Research in Metabolism, Maastricht University, Maastricht, The Netherlands*
[3]*Department of Cardiovascular Disease, Pax Clinic, İstanbul, Türkiye*
[4]*Department of Cardiovascular Surgery, Vehbi Koç Foundation American Hospital, İstanbul, Türkiye*
[5]*Department of Cardiology, Ministry of Health Çorlu State Hospital, Çorlu, Türkiye*
[6]*Department of Cardiology, Faculty of Medicine, Arel University, İstanbul, Türkiye*
[7]*Department of Biochemistry, Prof. Dr. Süleyman Yalçın City Hospital, İstanbul, Türkiye*
[8]*Department of Cardiology, İstanbul Arel University, Memorial Bahçelievler Hospital, İstanbul, Türkiye*
[9]*Department of Cardiology, Royal Brompton Hospital, Chelsea, London, United Kingdom*
[10]*Department of Cardiology, İstanbul Üniversitesi – Cerrahpaşa Faculty of Medicine, İstanbul, Türkiye*
[11]*Bahçeşehir University Faculty of Medicine, İstanbul, Türkiye*

Corresponding author:
Volkan Çamkıran
✉ wolfkan36@gmail.com

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uric acid index for detecting ≥50% coronary artery stenosis as determined by coronary computed tomography (CT) angiography.

METHODS

This retrospective observational study was conducted in the Department of Cardiology of a tertiary-care center. The study protocol was approved by a Local Institutional Ethics Committee at its meeting on January 5, 2023, with decision number 86. All participants provided informed consent. The study was carried out in accordance with the principles of the Declaration of Helsinki.

All consecutive adult patients (≥18 years) who underwent coronary computed tomography (CT) angiography between January 2023 and December 2024 were screened for eligibility. The cohort included both outpatients and inpatients referred for diverse indications, such as routine health check-ups of asymptomatic individuals, evaluation of atypical or typical angina (including intermittent or persistent chest pain), assessment of dyspnea or reduced exercise tolerance, and preoperative cardiac evaluation before noncardiac surgery. Patients with complete demographic and laboratory data were included. Patients were excluded if they had pregnancy, incomplete or missing digital records, known coronary artery disease or prior coronary revascularization (PCI or coronary artery bypass grafting (CABG)), active infection or inflammatory disease, chronic kidney disease (eGFR < 60 mL/min/1.73 m^2), hepatic dysfunction (ALT or AST > 3× upper limit of normal), gout or urate-lowering therapy, malignancy, or use of lipid-lowering or antidiabetic drugs that could influence metabolic parameters.

Coronary calcium scoring was performed using the standard Agatston method[7] by 2 independent cardiologists, and results were recorded in a computerized database. Coronary stenosis severity was graded according to the Coronary Artery Disease—Reporting and Data System (CAD-RADS) classification, and significant stenosis was defined as CAD-RADS ≥3 (≥50% luminal narrowing). Baseline demographic, clinical, and laboratory variables were retrieved from the hospital's electronic medical records and entered into a dedicated study dataset. The uric acid index was calculated as previously described[6] using the formula Ln [fasting triglycerides (mg/dL) × fasting uric acid (mg/dL) × fasting glucose (mg/dL)/2].

HIGHLIGHTS

- A composite uric acid score that combines fasting glucose, triglycerides, and uric acid was tested for predicting substantial coronary stenosis on coronary computed tomography angiography.
- In a retrospective real-world cohort, patients with ≥50% coronary artery stenosis had significantly higher uric acid levels.
- These results provide credence to the possible use of a metabolic composite score in coronary risk assessment, which calls for further investigation.

The normality of continuous variables was tested with the Kolmogorov–Smirnov test. Normally distributed data are presented as mean ± SD and compared using the Student's *t*-test, whereas non-normally distributed data are presented as median [interquartile range] and compared using the Mann–Whitney *U*-test. Categorical variables are expressed as counts and percentages and compared with the chi-square test. Correlations between the uric acid index and metabolic or imaging parameters were assessed using Pearson's (for normally distributed continuous data) or Spearman's (for non-normally distributed continuous data) correlation coefficients as appropriate. To assess the independent predictive value of the uric acid index, a hierarchical multiple logistic regression analysis that is strongly correlated with HbA1c were performed. To avoid multicollinearity, HbA1c was strictly excluded from the multiple models, as the uric acid index formula inherently incorporates fasting glucose levels. The incremental value of the uric acid index was assessed by adding it to a baseline model (containing age, sex, creatinine and Agatston score) and calculating the Net Reclassification Improvement (NRI) and Integrated Discrimination Improvement (IDI). Receiver-operating characteristic (ROC) curves were compared using DeLong's test to evaluate the superiority of the index over serum uric acid alone. Hierarchical logistic regression models were constructed: Model 1 (unadjusted), Model 2 (adjusted for age, sex), and Model 3 (adjusted for age, sex, creatinine, and Agatston score). Receiver-operating characteristic curves were constructed to compare the discriminatory performance of the uric acid index versus uric acid alone, and area under the curve (AUC) values, sensitivities, and specificities were reported. A 2-sided $P < .05$ was considered statistically significant. All statistical analyses were performed using IBM SPSS Statistics, version 19 (IBM Corp., Armonk, NY, USA).

RESULTS

Among the 258 participants, 71 (27.5%) exhibited ≥ 50% stenosis at coronary CT angiography, while 187 (72.5%) had < 50% stenosis. Patients with significant stenosis were older (median 61 [50-70] vs. 51 [43-60] years, $P < .001$), had higher Agatston scores (54 [3-325] vs. 0 [0-13], $P < .001$), and larger ascending aortic diameters (34 [32-38] vs. 33 [29-35] mm, $P = .002$) compared with those without stenosis. Sex distribution did not differ significantly between groups (male 69% vs. 58.3%, $P = .114$) (Table 1).

Laboratory analysis revealed that the ≥ 50% stenosis group had higher serum creatinine (1.0 ± 0.7 vs. 0.8 ± 0.2 mg/dL, $P = .003$), higher uric acid (6.2 [5.1-6.8] vs. 5.1 [3.8-6.2] mg/dL, $P < .001$), higher fasting glucose (97 [84-111] vs. 91 [83-100] mg/dL, $P = .030$), higher HbA1c (6.1 ± 1.4 vs. 5.6 ± 0.7 %, $P = .006$), and lower HDL-C (43 [35-59] vs. 53 [41-64] mg/dL, $P = .012$). The uric acid index was also significantly higher among the participants with ≥ 50% stenosis (10.8 ± 0.7 vs. 10.3 ± 0.8, $P < .001$) (Table 2).

In univariable logistic regression analyses (Table 3), age (OR = 1.056, $P < .001$, 95% CI: 1.025-1.088), creatinine (OR = 11.710; $P = .005$, 95% CI: 2.132-64.302), HbA1c (OR = 1.776; $P = .003$, 95%

Table 1. Comparison of Clinical Data and Demographics Between Patients and Controls

Demographic and Clinical Variables	Patients CAD–RADS ≥III ≥50% Stenosis (n = 71) (%)	Controls CAD–RADS <III <50% Stenosis (n = 187) (%)	*P*
Male gender, n (%)	49 (69)	109 (58.3)	.114
Age (years)	61 (50-70)	51 (43-60)	<.001
Agatston score*	54 (3-325)	0 (0-13)	<.001
Ascending aorta (mm)*	34 (32-38)	33 (29-35)	.002

Expressed as median (interquartile range).

CI: 1.217-2.591), and the uric acid index (OR = 2.176; *P* = .001, 95% CI: 1.392-3.401) were all significantly associated with the presence of ≥ 50% stenosis. Additionally, Agatston score and ascending aorta diameter were also significant predictors.

A hierarchical multiple logistic regression analysis were performed to assess the robustness of the uric acid index as a predictor of ≥50% coronary stenosis (Table 3). In the unadjusted model (Model 1), the uric acid index was strongly associated with significant stenosis (OR = 2.176, *P* = .001, 95% CI: 1.392-3.401). When adjusted for demographic factors including age and male sex (Model 2), the association remained significant (OR = 1.861, *P* = .018, 95% CI: 1.112-3.114). Finally, in the fully adjusted model (Model 3), which further included renal function (creatinine) and Agatston score, the uric acid index retained its independent association with significant stenosis (OR = 1.988, *P*p = .019, 95% CI: 1.117-3.538). The addition of UAI to the base model (age, sex, creatinine, and Agatston score) significantly improved the model's performance (NRI: 0.500; IDI: 0.029). In this final model (Model 3), the Agatston score (OR = 1.005, *P* = .011, 95% CI: 1.001-1.009) and age (OR = 1.057, *P* = .021, 95% CI: 1.008-1.107) were identified as independent predictors, while male sex (*P* = .132) and creatinine (*P* = .297) did not reach statistical significance. HbA1c was excluded from this final model to avoid multicollinearity. This stepwise analysis demonstrates that the predictive value of the uric acid index is robust and independent of key demographic and metabolic confounders.

Receiver-operating characteristic analysis demonstrated that the uric acid index provided slightly superior discrimination for ≥50% stenosis compared with serum uric acid alone (AUC 0.688 vs. 0.664; sensitivity 67% vs. 63%; specificity 70% vs. 65%). According to the DeLong test, this difference in diagnostic performance was statistically significant (*P* = .032). (Table 4).

Table 2. Comparison of Laboratory Characteristics Between Patients and Controls

Laboratory Variables	CT Coronary Angiography ≥50% Stenosis (n = 71)	CT Coronary Angiography <50% Stenosis (n = 187)	*P*
WBC (10^3/μL)	8.1 (6.9-9.5)	7.9 (6.8-9.4)	.422
Hemoglobin (g/dL)	12.9±2.7	13.3±3.1	.224
Creatinine (mg/dL)	1±0.7	0.8±0.2	.003
Urea (mg/dL)	33±13	30±7.6	.387
Sodium (mmol/L)	142±3.9	140±3.4	.651
Potassium (mmol/L)	4.4±0.4	4.3±0.4	.239
Calcium (mg/dL)	9.6±0.5	9.4±1.2	.913
Uric acid (mg/dL)	6.2 (5.1-6.8)	5.1 (3.8-6.2)	<.001
Glucose(mg/dL)	97 (84-111)	91 (83-100)	.030
Albumin (g/L)	4.5±0.4	4.5±0.3	.233
AST (U/L)	20 (16-269)	19 (16-24)	.798
ALT (U/L)	22 (17-30)	21 (16-31)	.473
Total cholesterol (mg/dL)	206 (188-239)	212 (174-246)	.832
LDL-C (mg/dL)	133 (105-155)	127 (90-158)	.447
HDL-C (mg/dL)	43 (35-59)	53 (41-64)	.012
Triglyceride (mg/dL)	161 (108-215)	134 (85-203)	.062
Hb A1c (%)	6.1±1.4	5.6±0.7	.006
Homocysteine (μmol/L)	12 (9.5-15.3)	11.3 (9.7-14.5)	.533
NT-pro BNP (pg/mL)	55 (29-339)	51 (33-74)	.711
Uric acid index	10.8±0.7	10.3±0.8	<.001

Values are presented as mean ± SD or median (interquartile range) depending on normal distribution.
ALT, alanine aminotransferase; AST, aspartate aminotransferase; HBA1c, glycated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; NT-pro BNP, N-terminal natriuretic peptide; WBC, white blood cell.

Table 3. Univariate and Multiple Predictors of 50% Stenosis on Coronary CT Angiography

	Univariate Predictors			Multiple Predictors		
	OR	95% CI	*P*	OR	95% CI	*P*
Age (years)	1.056	1.025-1.088	<.001	1.057	1.008-1.107	.021
Male sex	1.583	0.887-2.825	.114	0.417	0.133-1.302	.132
Agatston score	1.007	1.003-1.011	.001	1.005	1.001-1.009	.011
Ascending aorta	1.158	1.063-1.261	.001			
Creatinine	11.710	2.132-64.302	.005	1.755	0.610-5.047	.297
Uric acid	1.503	1.177-1.918	.001			
Glucose	1.013	1.002-1.023	.016			
HDL-C	0.974	0.952-0.996	.023			
Triglyceride	1.002	0.999-1.004	.165			
Hb A1c	1.776	1.217-2.591	.003			
Uric acid index	2.176	1.392-3.401	.001	1.988	1.117-3.538	.019
Hierarchical Models						
Model 1				2.176	1.392-3.401	.001
Model 2				1.861	1.112-3.114	.018
Model 3				1.988	1.117-3.538	.019

Model 1: Unadjusted Uric Acid Model.
Model 2: Model 1 adjusted for Age and Male Sex.
Model 3: Model 2 adjusted for Creatinine and Agatston Score (Fully Adjusted Final Model).
CI, confidence interval; CT, computed tomography; HbA1C, glycated hemoglobin; HDL-C, high-density lipoprotein cholestrol; OR, odds ratio.

Scatter plots illustrating the relationships between the uric acid index and key metabolic and imaging parameters are presented in Figure 1. The uric acid index showed a significant negative correlation with HDL-C (panel a: $r = -.477$, $P < .001$, $r^2 = .228$) and significant positive correlations with ascending aortic diameter (panel b: $r = .365$, $P < .001$, $r^2 = .133$), non-HDL-C (panel c: $r = 0.477$, $P < .001$, $r^2 = 0.228$), triglycerides (panel d: $r = .795$, $P < .001$, $r^2 = .632$), serum uric acid (panel e: $r = .463$, $P < .001$, $r^2 = .214$), and glucose (panel f: $r = .499$, $P < .001$, $r^2 = .249$).

DISCUSSION

This single-center study evaluated whether a composite uric acid index combining fasting triglyceride, glucose, and uric acid values could predict the presence of significant coronary artery stenosis detected by CT angiography. The index was significantly elevated in individuals with ≥50% stenosis and remained an independent predictor even after adjustment for conventional cardiovascular risk factors. Although its discriminatory capacity was modest, it slightly exceeded that of serum uric acid alone. To the best of the knowledge, this study is the first to examine the standalone predictive performance of the uric acid index in cardiovascular risk stratification.

Table 4. ROC Analysis for Prediction of 50% Stenosis in Coronary CT Angiography

	Threshold	Specificity	Sensitivity	AUC (95% CI)
Uric acid	>5.6	65	63	0.664
UA index	>10.7	70	67	0.688

AUC, area under the curve; CT, computed tomography; ROC, receiver operating characteristic; UA, uric acid.

While many studies have examined serum uric acid levels in relation to coronary atherosclerosis, compelling evidence in asymptomatic populations supports its value as a screening biomarker. In a community cohort, Kiss et al[8] found that higher serum uric acid was independently associated with coronary artery calcium after adjusting for conventional risk factors. More recently, a CT angiography study showed elevated uric acid to be linked with greater plaque burden and stenosis severity,[9] while another work demonstrated associations with noncalcified plaque beyond calcified lesions.[10] Interestingly, in patients undergoing CABG, Wu et al[11] reported that adding serum uric acid to the TyG index produced a significant improvement in prognostic prediction—change in C-statistic 0.038, continuous NRI 0.336, IDI 0.031 ($P < .001$)—indicating a synergistic effect in patients undergoing surgical revascularization. The findings are consistent with this, showing that in a CT angiography–based cohort, a composite uric acid index retains independent predictive power for ≥50% stenosis, suggesting that integrating uric acid into metabolic indices might modestly improve discrimination compared to uric acid alone. Taken together, these findings support the rationale for further evaluating simple, routinely available metabolic parameters in combination as potential tools for early risk stratification, particularly in populations undergoing noninvasive coronary imaging.

The diagnostic superiority of the uric acid index over serum uric acid alone has been rarely investigated in the literature. Most longitudinal cohorts have focused exclusively on serum uric acid. A large Korean cohort of approximately 92,000 adults without gout reported that each 1 mg/dL increment in uric acid was associated with a 6% higher risk of incident cardiovascular disease or mortality (HR ~1.06).[12] Similarly, the

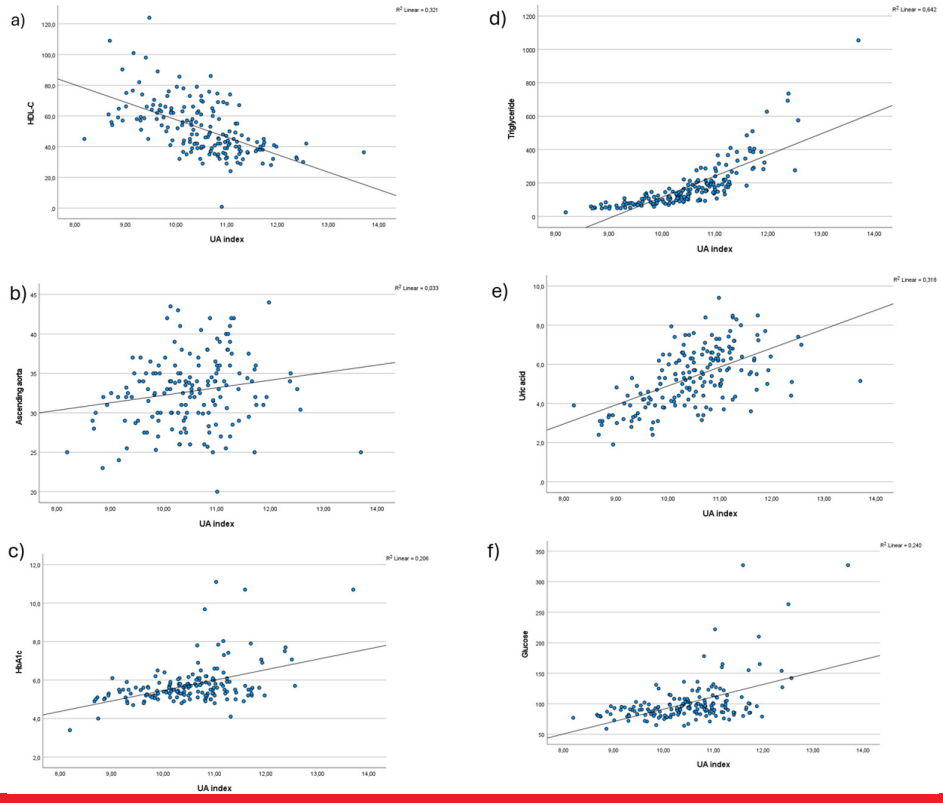

Figure 1. Scatter plots demonstrating linear correlations between the uric acid index and cardiometabolic/imaging parameters. (A) HDL-C ($r = -.477$, $P < .001$, $r^2 = .228$); (B) ascending aortic diameter ($r = .365$, $P < .001$, $r^2 = .133$); (C) non-HDL-C ($r = .477$, $P < .001$, $r^2 = .228$); (D) triglycerides ($r = .795$, $P < .001$, $r^2 = .632$); (E) serum uric acid ($r = .463$, $P < .001$, $r^2 = .214$); (F) fasting glucose ($r = .499$, $P < .001$, $r^2 = .249$). *r*, correlation coefficient; r^2, coefficient of determination; HDL-C, high-density lipoprotein cholesterol; non-HDL-C, non-high-density lipoprotein cholesterol.

URRAH study demonstrated a graded association between uric acid levels and all-cause or cardiovascular mortality, independent of triglyceride levels (adjusted HR 1.53 for all-cause, 2.08 for CVD per 1 mg/dL increase).[13] These studies highlight the prognostic relevance of uric acid as a cardiometabolic biomarker, yet they do not address whether its predictive value improves when incorporated into composite indices.[14] In another study, however, uric acid index showed a stronger correlation with cardiovascular risk ($R^2 = .31$) than uric acid alone ($R^2 = .19$), and remained independently associated with high cardiovascular risk after adjustment (PRa = 1.58, 95% CI 1.11-2.24).[10] The results align with these findings, demonstrating that a composite uric acid index is elevated among individuals with significant coronary stenosis and retains independent predictive value after controlling for conventional risk factors.

The hierarchical analysis showed that the uric acid index remained a significant predictor even after adjusting for age, sex, renal function and Agatston score. Although the odds ratio decreased slightly from 2.176 to 1.988 after adjustments, the persistence of statistical significance suggests that the index captures a metabolic risk profile not fully explained by traditional risk factors alone. This supports its potential utility as a practical, low-cost screening marker in asymptomatic individuals.

Few studies explored combinations of uric acid with other biomarkers to improve cardiovascular risk prediction. Yi et al[15] showed that the uric acid-to-HDL cholesterol ratio (UHR) was strongly associated with cardiovascular disease incidence, with participants in higher UHR quartiles showing markedly greater CVD rates (quartile 4: 11%) and a non-linear relationship between UHR and cardiovascular risk.[15] Similarly, Zhang et al[16] demonstrated that the uric acid-to-albumin ratio predicted coronary slow flow even among individuals without significant stenosis, suggesting its potential utility in detecting early vascular dysfunction.[16] These observations parallel the findings in a CT angiography cohort, in which a composite uric acid index independently predicted ≥ 50% stenosis. Collectively, the evidence indicates that integrating uric acid with metabolic parameters may modestly enhance cardiovascular risk discrimination compared with single-marker approaches.

The strengths of this study include the use of coronary CT angiography, which allows direct visualization and quantification of coronary artery stenosis, providing a more accurate assessment of subclinical atherosclerosis compared with serum biomarkers or clinical scores alone. In addition, the relatively large sample size enhances the statistical robustness and generalizability of the findings. The study also utilized routinely available biochemical parameters,

making the uric acid index a practical and easily applicable tool in clinical settings.

Limitations of the Study

The study was conducted in a single tertiary-care center and relied on a retrospective review of consecutive cases, which may introduce selection bias and limit the generalizability of the findings. Although all available data were extracted from the digital archive, some potentially relevant variables could not be captured. Laboratory values were obtained in routine clinical practice rather than within a standardized research protocol, introducing the possibility of measurement variability. Due to the retrospective nature of the study, complete laboratory data (specifically HbA1c and creatinine) were not available for all patients, which necessitated the exclusion of some participants from the multiple analysis. Furthermore, to prevent multicollinearity with the glucose component of the index, HbA1c was not included in the final regression model. Additionally, while the Agatston score is a potent predictor of stenosis, its inclusion in the fully adjusted hierarchical model (Model 3) did not attenuate the independent predictive power of the uric acid index. This confirms that the index provides significant incremental value beyond traditional risk factors and coronary calcification. However, larger prospective studies are still warranted to further validate its long-term prognostic utility across diverse populations. Finally, as an observational study, causality between the uric acid index and the presence of significant coronary artery stenosis on CT angiography cannot be established. Larger, prospective studies in different settings are warranted to validate the findings and to define practical cut-off values for the uric acid index in cardiovascular risk assessment.

CONCLUSION

In a real-life cardiology practice, the composite uric acid index—combining triglyceride, glucose, and uric acid—was independently associated with the presence of ≥ 50% stenosis on coronary CT angiography, even after hierarchical adjustment for age, sex, renal function and Agatston score. Although its discriminatory performance was only modestly superior to that of uric acid alone, the findings suggest that combining simple metabolic biomarkers may provide incremental value in cardiovascular risk evaluation. Prospective validation in larger, multi-center, and ethnically diverse cohorts is warranted before considering the routine clinical implementation of this index.

Ethics Committee Approval: This study was approved by the Ethics Committee of Memorial Bahcelievler Hospital (Approval No: 86, Date: 05.01.2023).

Informed Consent: All participants provided informed consent.

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