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ORIGINAL ARTICLE

Metabolic and renal predictors of coronary artery calcification: the independent role of the uric acid/estimated glomerular filtration rate (UA/eGFR) ratio and Castelli indices

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ABSTRACT

Aim To examine the association between metabolic parameters and novel cardiometabolic indices with the coronary artery calcium score (CACS).

Methods This retrospective cross-sectional study included 130 patients who underwent coronary computed tomography angiography (CCTA) at the Radiology Clinic of the Clinical Centre of the University of Sarajevo between January and June 2024. Patients were classified into two groups: those with CACS ≤100 and those with CACS >100. Platelet count, mean platelet volume (MPV), estimated glomerular filtration rate (eGFR), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), uric acid (UA), and novel cardiometabolic indices, including Castelli risk index I and II (CRI-I and CRI-II), non-high-density lipoprotein cholesterol (non-HDL-C), were compared between the groups.

Results Patients with CACS >100 had significantly higher MPV, TC, LDL-C, UA, non-HDL-C, CRI-I, CRI-II, and the UA/eGFR ratio. Older age, increased platelet activity, dyslipidaemia, hyperuricemia, and the higher UA/eGFR ratio correlated positively with CACS, whereas eGFR correlated negatively. In multivariate regression analysis, the UA/eGFR ratio emerged as an independent predictor of higher CACS (OR=2.37; 95% CI 1.18–4.78; p=0.017).

Conclusion Elevated UA level and adverse cardiometabolic indices were associated with greater coronary artery calcification. The UA/eGFR ratio independently predicts higher CACS, highlighting its potential prognostic value.

Keywords: coronary angiography, glomerular filtration rate, uric acid, vascular calcification

INTRODUCTION

Coronary artery calcification (CAC) is a well-established marker of atherosclerosis that reflects calcium deposition within the coronary arterial wall. Although it does not identify soft, non-calcified plaques, the overall calcium burden remains a strong predictor of future cardiovascular events (1). The coronary artery calcium score (CACS) (Agatston score), is the most widely used measure of CAC. It is traditionally derived from non-contrast cardiac CT scans; however, with advances in imaging technology, contrast-enhanced coronary computed tomography angiography (CCTA) can also provide reliable calcium quantification and simultaneous visualisation of both calcified and non-calcified plaques (2).

CACS helps clinicians stratify cardiovascular risk; a score above 100 suggests an intermediate to high risk of CAD. It is often used to guide preventive strategies such as starting statins or aspirin. The primary limitation of CACS is that it is not particularly useful in symptomatic patients with established CAD; instead, it is primarily applied in asymptomatic patients or those with uncertain risk profiles (3). Since CACS does not capture non-calcified plaques and may not fully reflect the metabolic activity of atherosclerosis, incorporating metabolic parameters can enhance its reliability and improve early detection and risk stratification in cardiovascular disease. Metabolic parameters such as total cholesterol (TC), low-den-

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sity lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), and serum uric acid (UA) levels are significantly associated with the development of atherosclerosis and cardiovascular risk. LDL-C is a significant factor in plaque formation, whereas HDL-C exerts a protective effect through its involvement in reverse cholesterol transport (4). To complement conventional lipid measurements, several cardiometabolic indices have been explored to provide a more integrated assessment of lipid-related cardiovascular risk.

The atherogenic index of plasma (AIP) indicates the balance between protective and atherogenic lipids, with higher values reflecting greater cardiovascular risk. Non-HDL cholesterol (non-HDL-C) reflects the total concentration of atherogenic lipoproteins and is considered a stronger predictor of atherosclerotic risk than LDL-C alone. The Castelli indices, defined as the ratio of TC and HDL-C (Castelli I) and LDL-C to HDL-C (Castelli II), provide additional insight into lipid balance, with increased ratios reflecting a more atherogenic profile (5).

Elevated UA is associated with increased CAC, most likely due to pro-inflammatory and pro-oxidative mechanisms that promote endothelial dysfunction and vascular injury (6). Similarly, a reduced estimated glomerular filtration rate (eGFR), reflecting impaired kidney function, is associated with increased CAC risk due to disruptions in mineral metabolism and shared cardiovascular risk factors. The UA to eGFR ratio (UA/eGFR) has recently emerged as a composite biomarker that integrates metabolic and renal components. Higher values of this ratio correlate with greater atherosclerotic burden and CAC, even among patients with normal UA levels, suggesting superior predictive potential for cardiovascular risk (7-9). This interplay between UA metabolism, renal function, and vascular calcifications highlights the importance of managing metabolic and renal health to lower the cardiovascular risk (10).

Although previous studies have examined the impact of individual metabolic parameters on cardiovascular risk, data regarding their combined indices and direct links to CAC remain limited. Moreover, evidence from Bosnia and Herzegovina is particularly scarce, and no prior studies have specifically addressed this topic, underscoring the importance of filling this research gap.

The aim of this study was to investigate the role of metabolic and renal markers, including UA, lipid indices, and the UA/eGFR ratio, in severity of CAC and to identify metabolic determinants of coronary calcification to improve cardiovascular risk prediction.

PATIENTS AND METHODS

Patients and study design

A retrospective cross-sectional study included 130 patients who underwent CCTA at the Clinic for Radiology of the Clinical Centre of the University of Sarajevo between January 2024 and June 2024. Due to the retrospective nature of the study, the sample size was defined by the number of eligible patients available during the study period, and a formal sample size or power calculation was not performed. The inclusion criteria encompassed patients aged between 18 and 75 with complete medical history data, laboratory findings before CCTA, and a CCTA report with a recorded CACS. Patients who did not meet age criteria, patients with incomplete medical records, active oncological or haematological disease, previously known CAD, including prior myocardial infarction, percutaneous or surgical intervention, were not included in the study. Patients receiving medications that could significantly affect lipid profile or uric acid levels (statins, fibrates, allopurinol) were also excluded.

The Ethics Committee of the Clinical Centre of the University of Sarajevo approved the study.

Methods

Calcium score values were obtained from CCTA results performed on the Aquilion Prime CT scanner (Canon Medical Systems Corporation, Tochigi, Japan), using an iobitridol contrast agent. The area of each calcification was measured in mm^2 and multiplied by the calcium density within the calcification. Individual results for all arterial segments were summed to obtain the CACS (11). A CACS threshold of 100 Agatston units was used to stratify patients into groups, as initially proposed by Agatston et al. (12) and subsequently validated in extensive population studies such as the Multi-Ethnic Study of Atherosclerosis (MESA) (13).

The first group included patients with measured CACS≤100, while the second group comprised patients with CACS>100. Patients with CACS≤100 have a low to moderate risk of CAD, whereas those with CACS>100 have a high risk of CAD (11,3). Demographic and relevant clinical data were collected through a detailed analysis of medical records. The following parameters were extracted: age, sex, platelet count (PTL), mean platelet volume (MPV), serum creatinine, TC, LDL-C, HDL-C, TG, and UA. eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula, which considers serum creatinine levels, sex, age, and race (14).

Using the collected laboratory parameters, cardiometabolic indices were calculated according to established formulas, including the atherogenic index of plasma ($AIP=\log_{10}[TG/HDL\text{-}C]$), non-HDL cholesterol (non-HDL-C = TC – HDL-C), Castelli risk index I (CRI-I = TC/HDL-C), Castelli risk index II (CRI-II = LDL-C/HDL-C), and the uric acid to estimated glomerular filtration rate ratio (UA/eGFR = UA/eGFR).

Laboratory parameter values and cardiometabolic indices were compared between the two patient groups.

Statistical analysis

The results are presented in tabular form using the number of cases, arithmetic mean (M) with standard deviation (SD), or median and interquartile range (IQR), depending on the data type. The normality of continuous variables was tested using the Shapiro-Wilk test. Variables with non-normal distribution were analysed using nonparametric tests (Mann-Whitney and Spearman correlation); log transformation was not applied. Differences between the groups were assessed using the Student's t-test (t score) or Mann-Whitney U test (Z score), as appropriate. The association between CACS and individual laboratory parameters and cardiometabolic indices was analysed using Spearman's rank correlation coefficient (r). To identify independent predictors of higher CACS, a model using bivariate logistic regression analysis, incorporating all predefined predictors from the study framework. The results of all tests were considered statistically significant at a confidence level of 95% (<0.05).

RESULTS

The study included 130 patients, with a significantly higher prevalence of women, 90 (60%) (p=0.029). Patients with CACS>100 were older than those with CACS≤100, with a median age of 69 years (p=0.001).

Regarding haematological parameters, the platelet count (PTL) did not differ significantly between the groups (p=0.351). However, the mean platelet volume (MPV) was considerably higher in the CACS >100 group, with a median value of 8.80 fL (p=0.022).

In terms of kidney function and renal parameters, patients in the CACS>100 group had lower estimated glomerular filtration rates (eGFR) (p=0.038) and higher level of uric acid (UA) with a median of 334.5 µmol/L (p=0.0001).

The analysis of lipid profiles demonstrated that total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) levels were significantly higher in the CACS>100 group, with median values of 5.3 mmol/L (p=0.0001) and 3.7 mmol/L (p=0.0001), respectively. However, no significant differences were found in high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) between the groups (p=0.830, p=0.639, respectively).

Significant differences in cardiometabolic indices were observed between the groups. The non-HDL-C (p=0.0001), Castelli risk indices I (CRI-I) (p=0.0001), and II (CRI-II) (p=0.001) were significantly higher in the CACS>100 group. Furthermore, the UA/eGFR ratio was notably higher in this group (p=0.0001) (Table 1).

Table 1. Comparison od atherogenic and renal indices between patients with CACS≤100 and CACS>100

Variable	CACS≤100	CACS > 100	Test statistic*	p
AIP (Mean±SD)	0.13 ± 0.23	0.15 ± 0.60	t = -0.503	0.613
		Median (IQR)		
Non-HDL-C (mmol/L)	2.99 (2.70–3.64)	4.20 (3.70–5.00)	Z= -5.353	<0.001
CRI-I	3.72 (3.20–4.12)	4.64 (3.53–5.98)	Z= -3.902	<0.001
CRI-II	2.15 (1.55–2.48)	3.17 (2.10–3.88)	Z= -4.551	0.001
UA/eGFR ratio	3.49 (3.01–4.30)	4.71 (3.80–6.19)	Z= -5.189	<0.001

*t=test statistics for independent samples t-test; Z=test statistics for Mann-Whitney U test.
HDL-C, high-density lipoprotein; AIP, atherogenic index of plasma; CRI-I, Castelli risk index I; CRI-II, Castelli risk index II; UA/eGFR ratio, uric acid/estimated glomerular filtration rate ratio; IQR, interquartile range; SD, standard deviation; CACS, coronary artery calcium score

Correlation analysis revealed several parameters that were significantly associated with CACS. Age (r=0.405; p=0.0001), uric acid (r=0.439; p=0.0001), total cholesterol (r=0.449; p=0.001), and LDL-C (r=0.413; p=0.001) had moderate to strong positive correlations with CACS, indicating that older patients and those with higher levels of uric acid, TC, and LDL-C were more likely to have higher CACS. In contrast, eGFR showed a weak negative correlation with CACS (r=0.238; p=0.008), suggesting that patients with higher eGFR had a lower likelihood of having significant coronary artery calcification.

Further analysis of cardiometabolic indices revealed a strong positive correlation between the UA/eGFR ratio (r=0.471; p=0.0001) and CACS, suggesting that a higher UA/eGFR ratio was associated with a higher probability of increased coronary artery calcification. Additionally, non-HDL-C (r=0.425; p=0.0001) and Castelli risk indices (CRI-I and CRI-II) showed moderate positive correlations with CACS, further supporting their role as predictors (Table 2).

Table 2. Correlation analysis of the impact of parameters on coronary artery calcium score (CACS)

Variable	r*	p
Sex	-0.123	0.163
Age	0.405	<0.001
eGFR	-0.238	0.008
PTL	-0.024	0.786
MPV	0.221	0.012
TC	0.449	<0.001
HDL-C	0.102	0.250
LDL-C	0.431	<0.001
TG	-0.013	0.881
UA	0.439	<0.001
AIP	-0.055	0.534
Non-HDL-C	0.425	<0.001
CRI-I	0.247	0.005
CRI-II	0.319	<0.001
UA/eGFR ratio	0.471	<0.001

* r=Pearsons correlation coefficient.
eGFR, estimated glomerular filtration rate; PTL, platelet count; MPV, mean platelet volume; TC, total cholesterol; HDL-C, high-density lipoprotein; LDL-C, low-density lipoprotein; TG, triglycerides; UA, uric acid; AIP, atherogenic index of plasma; CRI-I, Castelli risk index I; CRI-II, Castelli risk index II; UA/eGFR ratio, uric acid/estimated glomerular filtration rate ratio.

Table 3. Logistic regression analysis of predictors of higher coronary artery calcium score (CACS) (N=130)

Variable	Coefficient (β)	Wald (t)	p	OR (95% CI)*
PTL	-0.073	-0.802	0.424	–
MPV	0.130	1.440	0.153	–
HDL-C	-0.078	-0.329	0.743	–
LDL-C	-0.371	-0.539	0.591	–
TG	0.092	0.321	0.749	–
UA	-0.160	-1.065	0.289	–
AIP	-0.275	-0.795	0.428	–
Non-HDL-C	0.338	0.564	0.574	–
CRI-I	-0.444	-0.602	0.548	–
CRI-II	0.543	0.705	0.482	–
UA/eGFR ratio	0.864	2.413	0.017	2.37 (95% CI 1.18–4.78)
Sex	-0.077	-0.839	0.403	–
Age	0.122	1.252	0.213	–

Model fit: Nagelkerke pseudo-R^2 = 0.470
*Odds ratio (OR) with 95% CI is shown only for the significant predictor (UA/eGFR ratio).
* Coefficient (β) - regression coefficient; a positive β indicates an increase, while a negative β indicates a decrease in the likelihood of higher CACS.
* Wald (t) - test of the significance of an individual regression coefficient (β); higher values indicate a greater contribution to the model
PTL, platelet count; MPV, mean platelet volume; HDL-C, high-density lipoprotein; LDL-C, low-density lipoprotein; TG, triglycerides; UA, uric acid; AIP, atherogenic index of plasma; CRI-I, Castelli risk index I; CRI-II, Castelli risk index II; UA/eGFR ratio, uric acid/estimated glomerular filtration rate ratio; N, number;

Regression analysis identified the UA/eGFR ratio as the only independent predictor of having CACS>100 (OR 2.37, 95% CI 1.18–4.78; p=0.017) (Table 3). The overall model explained 47.0% of the variance in CACS classification (pseudo-R^2, Nagelkerke = 0.470).

DISCUSSION

In our study, the UA/eGFR ratio emerged as an independent predictor of CACS>100, remaining significant after adjustment for traditional cardiovascular and metabolic risk factors. Secondary findings suggest that dyslipidaemia and increased platelet activity are strongly associated with a higher CACS score. Renal function is inversely related to coronary calcification.

Older age and the predominance of women in the high CACS group may be explained by the fact that women generally live longer than men, resulting in a higher proportion of elderly females in the study population. In addition, coronary calcium scores in women tend to rise sharply after menopause due to the loss of estrogen-mediated vascular protection (15,16). Consequently, the combination of greater longevity and postmenopausal acceleration of calcification likely accounts for the predominance of older women with higher CACS values observed in our cohort.

Patients with higher CACS values also demonstrated increased MPV, which correlated positively with the severity of coronary calcification. An elevated MPV indicates enhanced platelet reactivity, potentially leading to an increased risk of thrombosis. This effect, in addition to plaque instability, is particularly significant in atherosclerosis and cardiovascular disease. It may be a potential predictor of coronary heart disease, with higher MPV values associated with an elevated risk of cardiovascular events (17,18).

Patients with higher CACS values had lower eGFR and higher UA concentrations, suggesting that impaired kidney function and elevated uric acid jointly contribute to the progression of coronary calcification. These factors promote atherosclerosis through endothelial dysfunction, oxidative stress, systemic inflammation, and disturbances in mineral-bone metabolism (19-21). Moreover, elevated serum uric acid may accumulate in the arterial wall, increasing vascular rigidity and reducing elasticity (22).

In our study, neither UA nor eGFR independently predicted higher CACS, whereas the UA/eGFR ratio demonstrated a consistent association. This supports the concept that integrating metabolic and renal components provides a more sensitive indicator of subclinical atherosclerosis and cardiovascular risk (23). Similar results have been reported in Japanese and European cohorts, where higher UA/eGFR ratios were associated with increased coronary calcification and vascular stiffness, even among patients with normal uric acid levels (24,25).

Patients with CACS >100 showed markedly higher levels of TC, LDL-C, non-HDL-C, CRI-I, and CRI-II, confirming a strong relationship between dyslipidaemia and coronary calcification (26–29). In contrast, HDL-C, triglycerides, and AIP did not differ significantly between the groups, suggesting that these parameters may be less relevant in later stages of atherosclerosis (30-32). Moderate positive correlations between TC and LDL-C with CACS further underscore the contribution of atherogenic lipid fractions to plaque calcification. These findings align with previous reports demonstrating that non-HDL-C and Castelli indices are useful markers of overall lipid-related cardiovascular risk and may enhance risk stratification beyond conventional lipid parameters (27–29).

Study limitations include a relatively small sample size and a retrospective, single-centre, cross-sectional design, which may introduce selection bias and limit causal interpretation. The absence of a power analysis may limit the detection of smaller effects. Data on all medications were not available, which could have influenced results. Renal markers may fluctuate with hydration or metabolic changes, and there is a residual confounding by unmeasured comorbidities such as hypertension or obesity that cannot be excluded. The lack of longitudinal follow-up also limits the assessment of temporal associations. Nevertheless, our results offer essential insight into how metabolic and renal factors relate to coronary calcification and may have clinical implications for simple, cheap, and widely accessible cardiovascular risk evaluation.

In conclusion, this study demonstrates that the UA/eGFR ratio is independently associated with the extent of coronary artery calcification, highlighting the interplay between metabolic, renal, and lipid pathways in the continuum of vascular injury leading to calcification. Future multicentric, prospective studies with larger and more diverse populations are needed to validate these results and to explore whether integrating UA/eGFR into existing CAC-based risk models can enhance predictive accuracy and guide preventive strategies.

AUTHOR CONTRIBUTIONS

Statement: Conceptualization, Z.B.J. and M.B.; Methodology, Z.B.J., S.P. and M.B.; Software, M.B., and A.B.; Validation, E.B., M.B., S.P. and F.Z.; Formal analysis, Z.B.J. and M.B.; Investigation, Z.B.J., M.B., E.B., S.P., F.Z., A.B.; Resources, Z.B.J and M.B.; Data curation Z.B.J. and M.B.; Writing—original draft preparation, Z.B.J., M.B. and M.B.; Writing— review and editing, E.B., M.B., S.P., F.Z., and A.B.; Visualization, M.B.; Supervision, S.P., and F.Z.; Project administration, funding acquisition, Z.B.J and M.B.. All authors have read and agreed to the published version of the manuscript.

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Conflicts of interest: None to declare.

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