RESEARCH ARTICLE

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Uric Acid/Albumin Ratio For Predicting Contrast-Induced Nephropathy in Patients With Acute Coronary Syndrome Undergoing Coronary Angiography

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Objective: One of the common complications of coronary angiography and percutaneous coronary intervention in patients with acute coronary syndrome is contrast-induced nephropathy (CIN). The objective was to determine the predictive value of the uric acid-to-albumin ratio (UAR) in identifying the risk of CIN.

Materials and Methods: A total of 1,437 patients were enrolled in the study. Patients were divided into two groups: those with and those without CIN. Demographic characteristics and laboratory parameters were compared between the groups.

Results: Contrast-induced nephropathy was observed in 242 patients. Diabetes mellitus, hypertension, and atrial fibrillation were more prevalent in this group. Serum uric acid, albumin, and UAR levels differed significantly between the groups. In multivariate analysis, UAR was identified as an independent predictor of CIN (OR= 4.671, p = 0.005). The area under the curve (AUC) of UAR for predicting CIN was 0.930 (95% CI: 0.914–0.947). The optimal cut-off value was 0.164, with a sensitivity of 85.1% and specificity of 87.6% (p < 0.001).

Conclusion: UAR was identified as a significant independent predictor of CIN. Its use in routine practice may be beneficial for early identification of CIN risk before coronary angiography.

Key Words: *Serum uric acid, serum albumin, contrast-induced nephropathy, coronary angiography*

Akut Koroner Sendromlu Hastalarda Koroner Anjiyografi Sonrası Gelişen Kontrast Nefropatisini Öngörmede Ürik Asit/Albümin Oranı

Amaç: Akut sendromlar nedeniyle uygulanan primer perkütan girişimlerin en sık görülen komplikasyonlarından biri kontrast kaynaklı nefropati (CIN) gelişimidir. Bu çalışma, nefropati patofizyolojisinde rol oynayan mekanizmalar üzerindeki etkilerinden dolayı, ürik asit ve albüminin birleşik bir belirteç olarak değerlendirilmesini amaçlamıştır. Çalışmanın temel hedefi, Ürik Asit/Albümin Oranı'nın (UAR) CIN riskini öngörmedeki değerini belirlemektir.

Gereç ve Yöntem: Çalışmaya toplam 1.437 hasta dahil edildi. Hastalar CIN gelişimine göre iki gruba ayrıldı. Gruplar arasında demografik özellikler ve laboratuvar parametreleri karşılaştırıldı.

Bulgular: Kontrast kaynaklı nefropati 242 hastada gözlendi. Bu grupta diyabetes mellitus, hipertansiyon ve atriyal fibrilasyon daha yaygındı. Serum ürik asit, albümin ve UAR düzeyleri gruplar arasında anlamlı farklılık gösterdi. Çok değişkenli analizde, UAR bağımsız bir CIN öngördürücüsü olarak belirlendi (OR: 4,671; p = 0,005). UAR'ın CIN öngörüsündeki eğri altındaki alanı (AUC) 0,930 idi (%95 GA: 0,914–0,947). En uygun eşik değeri 0,164 olarak belirlendi; bu değer için duyarlılık %85,1 ve özgüllük %87,6 olarak hesaplandı (p < 0,001).

Sonuç: UAR, kontrast kaynaklı nefropati (CIN) için anlamlı ve bağımsız bir öngördürücü olarak belirlenmiştir. Koroner anjiyografi öncesinde CIN riskinin erken belirlenmesinde UAR'ın rutin klinik uygulamalarda kullanımı faydalı olabilir.

Anahtar Kelimeler: *Serum ürik asit, serum albümin, kontrast kaynaklı nefropati, koroner anjiyografi*

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Introduction

Contrast-induced nephropathy (CIN) is one of the most concerning complications of selective angiography, particularly in patients undergoing percutaneous coronary intervention (PCI) for acute or chronic coronary syndromes. The incidence of acute kidney injury (AKI) following angiography ranges from approximately 7% to 22%, with higher rates observed in patients undergoing primary PCI (1). Although various prophylactic strategies-such as statins and N-acetylcysteine-have been investigated to reduce this risk (2, 3) an effective preventive approach remains elusive due to the multifactorial pathogenesis of CIN (4). Therefore, early detection of CIN is of critical importance in this high-risk population. Uric acid (UA), the final product of purine catabolism, has been associated with endothelial dysfunction and inflammatory responses (5). Owing to its pro-inflammatory properties, elevated UA levels have been shown to be an independent predictor of CIN after coronary angiography (6). Conversely, inflammation can lead to a reduction in serum albumin levels (7). Serum albumin is also closely associated with renal function and has been linked to the severity of renal impairment (8). The uric acid-to-albumin ratio (UAR) is a novel index proposed as a marker of oxidative stress and inflammation. Recent evidence has demonstrated its prognostic utility in predicting adverse clinical outcomes in patients with ST-elevation myocardial infarction (STEMI) (9).

The aim of this study was to evaluate the utility of UAR—a simple and readily accessible laboratory parameter—as a predictor of CIN development in patients with acute coronary syndrome (ACS) undergoing coronary angiography.

Materials and Methods

Research and Publication Ethics: This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethics approval was obtained from the Atatürk University Faculty of Medicine Ethics Committee on 26.01.2023 (decision no: B.30.2.ATA.0.01.00/270; reference no: 01-69).

Patients Selection: This study was designed as a retrospective analysis. Patients who presented to our tertiary care hospital between January 2023 and April 2023, were diagnosed with acute coronary syndrome (ACS), and underwent coronary angiography were included. A total of 1,628 patient records were reviewed. After excluding patients with end-stage renal disease or those requiring hemodialysis, severe proteinuria, active malignancies, rheumatologic diseases, or those receiving uric acid–lowering therapy, data from the remaining 1,437 patients were included in the final analysis. Patient data were obtained from the hospital's electronic medical record system and emergency department admission forms. The diagnosis of ACS was established according to the current clinical guidelines available at the time(10) Coronary angiography or PCI was performed via either femoral or radial access. A non-ionic, low-osmolar contrast agent was used in all procedures. The volume of contrast administered was determined manually by the interventional cardiologist and was recorded for each patient.

Laboratory Measurements: Blood samples were collected from the brachial vein upon admission to the emergency department and prior to the initiation of reperfusion therapy. Biochemical analyses were performed using a standard automated analyzer (Beckman Coulter Inc., Brea, CA, USA). The reference range for serum uric acid (UA) was 3.5–7.2 mg/dL, and for serum albumin, it was 35–52 g/L. The uric acid-to-albumin ratio (UAR) was calculated by dividing the serum uric acid level by the albumin level. Biochemical and complete blood count (CBC) tests were repeated daily at 06:00 during hospitalization, and the peak values observed during the hospital stay were recorded for analysis.

Follow-up: The Kidney Disease: Improving Global Outcomes (KDIGO) guideline was used to diagnose contrast-induced nephropathy (CIN) during post-procedural follow-up. According to this guideline, acute kidney injury (AKI) is defined by any of the following criteria:

- An increase in serum creatinine by ≥0.3 mg/dL within 48 hours,
- An increase to ≥1.5 times the baseline value within the previous 7 days, or
- A urine output of <0.5 mL/kg/hour for more than 6 hours (11).

Data on the need for dialysis, length of hospital stay, and in-hospital mortality were also recorded.

Statistical Analysis: Statistical analyses were performed using the SPSS software program (IBM SPSS Inc., Chicago, IL, USA). The distribution of variables was assessed using the Shapiro-Wilk test. Depending on the distribution characteristics, either the Student's t-test or the Mann–Whitney U test was applied for comparisons between groups.Bivariate regression analysis was used to identify predictors of CIN. Receiver operating characteristic (ROC) curve analysis was conducted to determine the optimal cut-off value for predicting CIN. The area under the curve (AUC) and the corresponding 95% confidence intervals (CI) were reported. A p-value of <0.05 was considered statistically significant.

Results

A total of 1,437 patients were included in the study. Patients were categorized based on the development of contrast-induced nephropathy (CIN), which was observed in 242 individuals. Upon evaluation of demographic characteristics, male sex, diabetes mellitus (DM), hypertension (HT), and atrial fibrillation (AF) were found to be significantly more common in the CIN group. Among laboratory parameters, hemoglobin, glucose, uric acid, peak creatinine, peak troponin, NT-proBNP, serum albumin, and the uric acid-to-albumin ratio (UAR) differed significantly between the two groups. Detailed comparisons are provided in Table 1. Univariate and multivariate logistic regression analyses were performed to identify potential predictors of CIN. In the multivariate analysis, which included parameters that were statistically significant in the univariate model, AF (OR: 2.658), DM (OR: 3.100), peak creatine kinase–myocardial band (CK-MB) level (OR: 1.001), and UAR (OR: 4.671) were identified as independent predictors of CIN ($p < 0.001$, < 0.001, 0.045, and 0.005, respectively) (Table 2). Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive performance of UAR for CIN. The area under the curve (AUC) was 0.930 (95% CI: 0.914–0.947), with an optimal cut-off value of 0.164, yielding a sensitivity of 85.1% and specificity of 87.6% ($p < 0.001$) (Table 3 and Figure 1).

Table 1. Baseline characteristics, laboratory results of all study patients, and patients with and without CIN

	Total Study Population, n = 1437	Non-CIN group,n = 1195	CIN group, n = 242	p
Demographics				
Age, years	65.4±14.2	64.4±12.9	66.5±15.4	.571
Male gender, n (%)	1042(72.5)	890(74.5)	152(62.8)	**.001**
Atrial Fibrillation, n(%)	167(11.6)	102(8.5)	65(26.9)	**<.001**
Diabetes mellitus, n (%)	525(36.5)	384(32.2)	141(58.5)	**<.001**
Hypertension, n (%)	726(50.6)	584(49.0)	142(58.7)	**.004**
Hyperlipidemia, n (%)	878(61.1)	721(60.3)	157(64.9)	.105
CAD, n(%)	561(39.0)	467(39.1)	94(39.3)	.506
Heart Failure, n (%)	1093(76.6)	891(74.6)	202(83.5)	**.002**
Smoking, n (%)	629(43.8)	524(43.9)	105(43.3)	.086
BMI, kg/m2	27.6±5.4	28.4±5.1	26.7±5.6	.226
On admission, clinical characteristics				
Systolic blood pressure, mm/Hg	135.9±36.1	136.3±40.0	135.4±32.2	.999
Heart rate per minute	81.4±20.2	80.3±19.4	82.4±20.9	.129
Left-ventricular ejection fraction (%)	41.5±10.2	42.8±10.4	40.1±10.2	**<.001**
MI type				**<.001**
STEMI	742(51.6)	574(48.0)	168(69.4)	
NSTEMI/USAP	695(48.4)	621(52.0)	74(30.6)	
Laboratory results				
Hemoglobin, g/dL	13.6±2.6	14.0±2.3	13.2±2.9	**<.001**
White blood cell count, cells/μL	10.6±5.3	10.5±4.9	10.7±5.7	.792
Platelet count, cells/μL	257±84	261±82	253±86	.171
Admission blood glucose, mg/dL	173.8±92.4	165.2±88.2	182.4±96.5	**.007**
Uric acid, mg/dL	6.1±0.5	5.8±0.6	6.3±0.4	**<.001**
Baseline creatinine, mg/dL	1.25±0.97	1.21±0.91	1.32±1.14	.198
Peak creatinine, mg/dL	1.64±1.28	1.43±1.21	1.85±1.36	**<.001**
Peak creatine kinase–myocardial band, ng/mL	79.9(4-4356)	72.1(4-1732)	118.3(45-4356)	**.001**
Peak troponin, ng/l	9803(31-24615)	8486(31-15302)	11629(4203-24615)	**.003**
NT-proBNP, pg/dL	2110(123-12345)	1984(123-2044)	2729(547-12345)	**.004**
Albumin, g/L	36.7±3.6	40.2±3.5	33.2±3.7	**<.001**
Total cholesterol, mg/dL	179.1±50.8	185.9±57.3	172.2±44.2	.192
TG, mg/dL	166.8±82.3	178.4±77.5	155.1±87.1	.082
HDL, mg/dL	39.3±13.8	39.4±12.5	39.1±15.1	.813
LDL, mg/dL	124.9±36.0	126.5±37.7	123.4±34.2	.240
Uric Acid-Albumin Ratio	0.17±0.02	0.14±0.02	0.19±0.01	**<.001**
Angiographic and clinical data				
Multi-vessel stenosis (> 50%), n (%)	404(28.1)	346(29.0)	58(24.0)	.066
LAD of the infarct-related artery, n (%)	752(52.3)	588(49.2)	164(67.8)	**<.001**
Contrast volume, mL	276.2±162.4	274.8±161.1	277.5±163.8	.814
Need for dialysis, n(%)	28(1.9)	0	28(11.6)	**<0.001**
Length of hospital stay, days	6(2-44)	6.3(2-13)	8.6(3-44)	**<.001**
In-hospital mortality, n(%)	59(4.1)	36(3)	23(9.5)	**<.001**

CAD: coronary artery disease, HF: heart failure, BMI: body mass index, STEMI: ST-elevation myocardial infarction, NSTEMI: non-ST-elevation myocardial infarction, NT-proBNP: N-terminal prohormone brain natriuretic peptide, TG: triglycerides, HDL: high-density lipoprotein, LDL: low-density lipoprotein, LAD: left anterior descending artery, USAP: Unstable angina pectoris

Table 2. Univariate and multivariate analyses for the predictor of CIN

	Univariate Analysis	p	Multivariate Analysis	p
	OR (95% CI)		OR (95% CI)	
Male	1.728(1.291-2.312)	**<.001**	0.790(0.464-1.347)	.387
Atrial fibrillation	1.579(1.405-1.774)	**<.001**	2.658(1.836-5.502)	**<.001**
Diabetes mellitus	2.967(2.234-3.940)	**<.001**	3.100(1.795-5.353)	**<.001**
Hypertension	1.482(1.119-1.959)	**.006**	1.236(0.736-2.077)	.424
Heart Failure	1.723(1.198-2.478)	**.003**	1.182(0.547-2.554)	.671
Left-ventricular ejection fraction	0.975(0.962-0.988)	**<.001**	0.984(0.954-1.016)	.331
Hemoglobin	0.866(0.815-0.920)	**<.001**	0.980(0.894-1.075)	.674
Admission blood glucose	1.002(1.001-1.003)	**.007**	0.999(0.996-1.002)	.414
Uric acid	2.361(1.579-3.532)	**<.001**	0.037(0.001-1.175)	.062
Peak creatine kinase–myocardial band	1.001(1.000-1.001)	**.009**	1.001(1.000-1.001)	**.045**
Peak troponin	1.001(1.001-1.002)	**.004**	1.001(0.998-1.003)	.260
NT-proBNP	1.001(1.001-1.002)	**.001**	1.001(0.947-1.081)	.201
Albumin	0.585(0.548-0.625)	**<.001**	1.279(0.728-2.245)	.392
LAD of the infarct-related artery	2.171(1.620-2.908)	**<.001**	1.452(0.897-2.351)	**.129**
Uric Acid-Albumin Ratio	3.511(2.076-4.451)	**<.001**	4.671(3.906-5.582)	**.005**

Table 3. Receiver operating characteristics analysis results for acid-to-albumin ratio

Risk Factor	The area under the curve (95%)	Cut Off	P	Sensitivity (%)	Specificity (%)
UAR	0.930(0.914-0.947)	0.164	<0.001	85.1	87.6

UAR: Uric acid- albumin ratio

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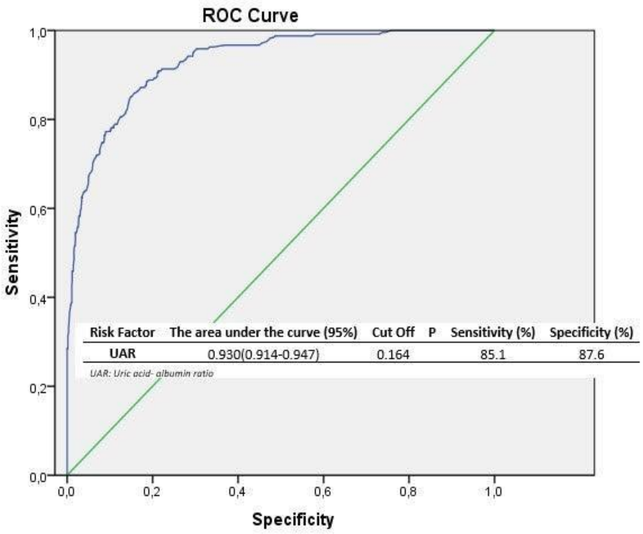

Figure 1. ROC curve analysis of the uric acid-to-albumin ratio for predicting contrast-induced nephropathy.

Discussion

The primary focus of this study was the association between the uric acid-to-albumin ratio (UAR) and the development of contrast-induced nephropathy (CIN). A higher UAR was found to be associated with a higher likelihood of CIN, suggesting that it may serve as an indicator of risk. To the best of our knowledge, this is one of the few studies including all acute coronary syndrome (ACS) subtypes in this context. Contrast media (CM) are widely used in various imaging procedures, particularly in coronary angiography. They are eliminated exclusively via glomerular filtration. In individuals with normal renal function, CMs are typically cleared within 24 hours (12).

The pathophysiology of CIN involves three primary mechanisms: medullary ischemia, the generation of reactive oxygen species, and direct tubular toxicity. However, the exact contribution of each mechanism remains uncertain (13). CIN has been reported to account for approximately 11% of hospital-acquired acute kidney injury in previous studies (14). Once CIN develops, it is associated with increased morbidity and mortality over the subsequent two years (15). Established risk factors for CIN include hypertension, diabetes mellitus, atrial fibrillation, and chronic heart failure (CHF), all of which have been shown to increase susceptibility to acute kidney injury after cardiac catheterization (16). In the subgroup analysis of ACS types, the incidence of CIN was noticeably higher in patients presenting with STEMI compared with those with NSTEMI. This finding may be explained by the greater inflammatory burden, more pronounced ischemia–reperfusion injury, and increased hemodynamic instability typically associated with STEMI, all of which may exacerbate renal vulnerability to contrast exposure. Furthermore, STEMI procedures often require urgent and more extensive angiographic evaluation, which can lead to higher contrast volume and additional procedural stress. Previous studies have similarly reported higher rates of CIN in STEMI populations (1), supporting the notion that this subgroup carries an intrinsically elevated risk profile. The alignment of our results with existing evidence reinforces the importance of careful renal risk assessment in STEMI patients undergoing coronary angiography. CIN remains a major clinical concern in interventional cardiology due to its impact on long-term outcomes.

Serum uric acid (UA) may have prognostic value in ST-elevation myocardial infarction (STEMI), as elevated levels have been linked with adverse in-hospital events (17). Several studies have demonstrated a significant relationship between hyperuricemia and increased risk of CIN following coronary angiography (18). Experimentally, UA has been shown to precipitate as monosodium urate crystals, inducing local inflammation. Moreover, UA contributes to oxidative stress, inflammation, and endothelial dysfunction (19). Sanchez-Lozada et al. reported that hyperuricemia is an independent risk factor for AKI in critically ill patients, even after adjusting for hypertension, chronic kidney disease, and CHF (20). Hyperuricemia may lead to AKI via both crystalline and non-crystalline pathways, including renal vasoconstriction, inflammation, and apoptosis. In a long-term cohort study involving nearly 50,000 participants, hyperuricemia was significantly associated with all-cause mortality and progression to renal failure (21).

Albumin is known to play important roles in various physiological and pathological processes. It is the main determinant of intravascular oncotic pressure and serves as a carrier protein for various bioactive molecules, including hormones, drugs, and free fatty acids (22). Additionally, albumin has demonstrated anti-inflammatory, anti-oxidative, and anti-apoptotic properties (23). Hypoalbuminemia has been linked to coronary artery disease and increased all-cause mortality (24). Oxidative stress and inflammation, both of which are central to the pathogenesis of CIN, are exacerbated by hypoalbuminemia (25). It leads to increased blood viscosity, oxidative stress, and endothelial dysfunction (26). Additionally, decreased synthesis and increased serum albumin catabolism have been associated with an increased inflammatory response (7). In patients with ACS, elevated chemokine levels trigger systemic inflammation, reduce antioxidant capacity, increase lipid peroxidation, and promote oxidative stress (27). A meta-analysis confirmed that hypoalbuminemia is a significant predictor of AKI (28).

Considering the shared pathophysiological pathways of uric acid and albumin in inflammation and oxidative stress, their combined evaluation through UAR has shown superior predictive value compared to individual assessment. One study demonstrated that UAR >1.7 was significantly associated with AKI in critically ill patients (29). Recently, Faisal et al. reported that UAR >1.62 provided better predictive accuracy for CIN (30). It is important to note that the UAR values in those studies were calculated based on albumin levels measured in g/dL. The findings of our study appear to be consistent with these results.

Several findings of our study are consistent with previous reports evaluating predictors of contrast-induced nephropathy. Similar to earlier studies, elevated uric acid levels and reduced serum albumin-both indicators of heightened oxidative stress and systemic inflammation-were associated with a higher risk of CIN. The strong predictive performance of the uric acid-to-albumin ratio in our cohort also aligns with recent evidence demonstrating its prognostic value in patients with acute coronary syndromes and in those undergoing primary percutaneous coronary intervention. Nevertheless, the magnitude of the predictive strength observed in our study appears to be higher than that reported in earlier literature, which may be explained by differences in patient selection, inclusion of all ACS subtypes, and the larger sample size. These similarities and discrepancies suggest that UAR may be a robust biomarker across different clinical contexts, although further multicenter prospective studies are warranted to clarify its generalizability.

This study has several limitations. First, it was conducted at a single center and designed retrospectively, which may introduce selection bias. Second, the multifactorial nature of CIN pathogenesis limits the ability of our findings to explain all contributing mechanisms. Lastly, although mortality data were collected, the absence of a clear linear relationship with UAR limits further interpretation; therefore, mortality was only presented descriptively in the table. Future prospective and multicenter studies will be needed to clarify the extent to which UAR can be integrated into routine clinical practice.

In conclusion, combined evaluation of serum uric acid and albumin as UAR revealed significant results in predicting CIN following ACS. Given its accessibility and simplicity, UAR may serve as a useful marker and should be considered for routine laboratory assessment following coronary angiography.

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