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Association Between Uric Acid-to-Albumin Ratio and Contrast-Induced Nephropathy and Mortality: An Evaluation in Chronic Total Occlusion Cases

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Abstract

Objectives: Among individuals undergoing coronary angiography (CAG), chronic coronary total occlusion (CTO) represents a prevalent lesion type that often requires treatment with percutaneous coronary intervention (PCI). Following PCI, contrast-induced nephropathy (CIN) represents a frequent complication that contributes to elevated morbidity and mortality. The uric acid-to-albumin ratio (UAR) has recently been identified as a novel biomarker linked to unfavorable clinical outcomes. This investigation sought to determine the prognostic significance of UAR for CIN and long-term mortality in CTO patients.

Methods: A total of 169 patients managed with PCI for one or more CTO lesions were retrospectively evaluated. Patients were then categorized according to the development of CIN into two groups: CIN-positive (n = 27) and CIN-negative (n = 142).

Results: The CIN (+) group demonstrated significantly elevated serum uric acid levels, higher UAR values, and increased mortality rates compared with the CIN (−) group (all $p < 0.001$). Further multivariate regression analysis established UAR as an autonomous prognostic indicator of CIN ($p = 0.012$). A UAR cut-off value of 1.77 predicted CIN with 66.7% sensitivity and 62% specificity, while a cut-off of 1.90 predicted long-term mortality with 64.5% sensitivity and 73.9% specificity. According to Kaplan–Meier survival curves, individuals in the CIN-positive group exhibited markedly lower long-term survival and a higher frequency of all-cause death (log-rank, $p < 0.001$).

Conclusion: An increased UAR independently predicted both CIN and long-term mortality in CTO patients, underscoring its prognostic significance in this high-risk population.

Keywords: Chronic total occlusion (CTO), Contrast-Induced Nephropathy (CIN), Uric Acid-to-Albumin Ratio (UAR), mortality

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Ürik Asit-Albumin Oranı ile Kontrast Kaynaklı Nefropati ve Mortalite Arasındaki İlişki: Kronik Total Oklüzyon Olgularında Bir Değerlendirme

Öz

Amaç: Koroner anjiyografi (CAG) uygulanan bireyler arasında, kronik koroner total oklüzyon (CTO) sık görülen bir lezyon tipini temsil etmekte olup genellikle perkütan koroner girişim (PCI) ile tedavi gerektirmektedir. PCI sonrasında, kontrast kaynaklı nefropati (CIN) sık rastlanan bir komplikasyon olup artmış morbidite ve mortaliteye katkıda bulunmaktadır. Ürik asit-albumin oranı (UAR), son dönemde olumsuz klinik sonuçlarla ilişkili yeni bir biyobelirteç olarak tanımlanmıştır. Bu çalışma, CTO hastalarında UAR'ın CIN ve uzun dönem mortalite açısından prognostik önemini belirlemeyi amaçlamıştır.

Yöntemler: Bir veya daha fazla CTO lezyonu nedeniyle PCI uygulanan toplam 169 hasta retrospektif olarak değerlendirildi. Hastalar, CIN gelişimine göre iki gruba ayrıldı: CIN-pozitif (n = 27) ve CIN-negatif (n = 142).

Bulgular: CIN (+) grubu, CIN (−) grupla karşılaştırıldığında anlamlı şekilde daha yüksek serum ürik asit düzeyleri, daha yüksek UAR değerleri ve artmış mortalite oranları gösterdi (tümü p < 0,001). Ayrıca, çok değişkenli regresyon analizi UAR'ı CIN için bağımsız bir prognostik gösterge olarak ortaya koydu (p = 0,012). UAR için 1,77 kesme değeri CIN'i %66,7 duyarlılık ve %62 özgüllük ile öngörürken, 1,90 kesme değeri uzun dönem mortaliteyi %64,5 duyarlılık ve %73,9 özgüllük ile tahmin etti. Kaplan–Meier sağkalım eğrilerine göre, CIN-pozitif gruptaki bireyler uzun dönem sağkalım açısından belirgin şekilde daha düşük ve tüm nedenlere bağlı ölüm sıklığı açısından daha yüksek bulundu (log-rank, p < 0,001).

Sonuç: Artmış UAR, CTO hastalarında hem CIN'i hem de uzun dönem mortaliteyi bağımsız olarak öngörmüş olup, bu yüksek riskli popülasyonda prognostik önemini vurgulamaktadır.

Anahtar kelimeler: Kronik total oklüzyon (CTO), Kontrast Kaynaklı Nefropati (CIN), Ürik Asit-Albumin Oranı (UAR), mortalite.

INTRODUCTION

The term chronic total occlusion (CTO) denotes a persistent, complete coronary artery blockage with a duration greater than three months. Angiographically, it is characterized by TIMI 0 flow in the affected vessel segment[1]. The prevalance of CTO among patients undergoing coronary angiography (CAG) ranges from 15% to 30%[2,3]. CTO has been linked to unfavorable clinical clinical results in the short as well as the long term[4].

Among the complications of percutaneous coronary intervention (PCI), contrast-induced nephropathy (CIN) is observed with notable frequency. It has been associated with acute kidney injury, longer hospital stays, and higher rates in relation to both cardiovascular and overall mortality[5]. Identifying high-risk patients before contrast exposure is critical. The most widely used risk scoring system in this context was defined by Mehran et al. and is based on eight factors:[6] age >75 years, anemia, diabetes mellitus (DM), congestive heart failure (CHF), chronic kidney disease (CKD), contrast volume, hypotension, and use of an intra-aortic balloon pump (IABP). However, because this model incorporates procedural characteristics, it cannot be used to identify at-risk patients in the preprocedural setting. This limitation prompted us to investigate whether alternative preprocedural markers could guide risk stratification for CIN.

Serum uric acid has been shown to independently predict CIN in previous studies[7]. Albumin, conversely, exerts anti-inflammatory and antioxidant properties. Lower serum albumin levels have been observed in patients who develop CIN[8,9].

Recent studies have identified the serum uric acid-to-albumin ratio (UAR) as an innovative biomarker[10]. It is easy to calculate using routine biochemical tests and may serve as a practical clinical tool.

The main goal of this investigate was to explore the prognostic role of UAR for CIN and long-term death in patients with CTO treated with

PCI. We also examined whether UAR could serve as a risk stratification marker in this population.

METHODS

Patients were enrolled in this single-center, retrospective observational study between 18 and 95 years of age who underwent CAG. The study included 169 consecutive patients detected with CTO and admitted at our center between January 1, 2016, and December 31, 2021. Demographic characteristics, laboratory results, echocardiographic findings, and follow-up data were collected from hospital and social security records. Creatinine measurements were retrospectively recorded at hospital admission, prior to PCI, and during the first 72 hours after the procedure. Pre-procedural serum uric acid and albumin levels were utilized to compute the UAR.

Inclusion Criteria

Patients undergoing PCI for at least one coronary artery CTO lesion were recruited for the study.

Exclusion Criteria

Exclusion criteria were: age under 18 years, presentation with acute coronary syndrome (ACS), end-stage renal disease (GFR <15 mL/min/1.73 m^2), history of renal transplantation, severe heart failure (New York Heart Association Class IV), malignancy, hematological disorders, and exposure to contrast media within the last 7 days prior to PCI.

Data Collection

Demographic features (age, sex) and comorbidities such as DM, hypertension (HT), hyperlipidemia (HL), smoking, coronary artery disease (CAD), chronic kidney disease (CKD), peripheral artery disease (PAD), cerebrovascular disease (CVD), and CHF were retrieved from medical records. Clinical presentations, routine laboratory findings, and procedural data (contrast volume, duration) were recorded. Blood samples were obtained from the antecubital vein in the early morning following a 12-hour fast—prior to PCI and again at 24 and 72 hours post-procedure. Biochemical analyses were performed using an automated analyzer (Roche Diagnostic Modular Systems, Tokyo, Japan). All patients underwent transthoracic echocardiography (TTE) using a Vivid S5 system (GE Medical Systems, Horten, Norway). The Simpson's technique was used to evaluate left ventricular ejection fraction (LVEF). The PCI procedure employed a non-ionic, low-osmolar contrast medium (iohexol, Omnipaque 350 mg/mL). To mitigate CIN risk, intravenous isotonic saline (0.9%) was infused at 1 mL/kg/hour starting 12 hours before PCI and maintained for 12 hours after. In patients with significant LV dysfunction (LVEF <40%) or overt heart failure, the infusion rate was modified to 0.5 mL/kg/hour.

Definitions

For this study, CIN was diagnosed when serum creatinine rose by ≥0.5 mg/dL or ≥25% relative to baseline within 48–72 hours of contrast administration.11 Heart failure was defined as moderate-to-severe systolic dysfunction of the left ventricle with an LVEF <40%. HT was diagnosed as systolic BP ≥140 mmHg, diastolic BP ≥90 mmHg, or present use of antihypertensives. DM was described as fasting plasma glucose ≥126 mg/dL or ongoing treatment with insulin or oral antidiabetic drugs.

Study Endpoints

The main outcome measure in this study was the occurrence of CIN, while all-cause mortality was designated as the secondary endpoint.

Ethical Considerations

The protocol of this investigation received authorization from the local ethics committee (Approval No: 2025/4-38; Date: April 22, 2025). The conduct of this study strictly followed the ethical principles established by the Declaration of Helsinki (2024 version).

Statistical Analysis

Statistical analyses were conducted out using SPSS version 26.0. Data normality was evaluated with histograms, probability plots, and the Kolmogorov–Smirnov test. Continuous variables

were presented as mean ± SD or median (IQR), and categorical variables as percentages. The Student's t-test or Mann–Whitney U test was applied for continuous variables, and the chi-square or Fisher's exact test for categorical variables. Predictors of CIN were identified by univariate and multivariate logistic regression analyses, reported as ORs with 95% CIs. Survival was analyzed with Kaplan–Meier curves and contrasted by the log-rank test. A p-value <0.05 was deemed statistically important.

RESULTS

The participants of the study comprised 169 patients, with a mean age of 61.9 ± 11.1 years. Among them, 127 (75.1%) were male. Based on CIN development, the study population was classified into two groups: CIN-positive (CIN [+], n=27, 16%) and CIN-negative (CIN [−], n=142, 84%). An overview of the groups' demographic, clinical, and laboratory characteristics is provided in Table I.

Table I: Baseline Demographic, Clinical, and Laboratory Characteristics of the Study Population

Variables	*All patient (n=169)*	*CIN (-) (n=142)*	*CIN (+) (n=27)*	*P value*
Demographics				
Age (years)	61.9 ± 11.1	61.4 ± 10.9	64.7 ± 11.7	0.155
Gender, male, n (%)	127 (75.1)	105 (73.9)	22 (81.5)	0.406
Comorbidities, n(%)				
Ejection fraction (%)	48.4 ± 11.2	49.5 ± 10.7	42.4 ± 12	**0.002**
Hypertension, n (%)	56 (33.1)	44 (31)	12 (44.4)	0.173
Diabetes mellitus, n (%)	48 (28.4)	38 (26.8)	10 (37)	0.278
Smoking, n (%)	43 (25.4)	34 (23.9)	9 (33)	0.304
Chronic kidney disease, n (%)	9 (5.3)	5 (3.5)	4 (14.8)	**0.037**
Congestive heart failure, n (%)	18 (10.7)	13 (9.2)	5 (18.5)	0.148
Stroke history, n (%)	2 (1.2)	1 (0.7)	1 (3.7)	0.295
Peripheral arterial disease	7 (4.1)	4 (2.8)	3 (11.1)	0.082
History of coronary artery disease, n (%)	77 (45.6)	66 (46.5)	11 (40.7)	0.583
Laboratory Parameters, mean ± SD				
White blood cell count (×10^3/μL)	9.3 ± 3.5	9.4 ± 3.5	10 ± 3.8	0.439
Hemoglobin (g/dL)	13.7 ± 1.9	13.8 ± 1.9	13.7 ± 1.9	0.892
Platelets (×10^3/μL)	245.9 ± 75.2	243.7 ± 70.8	257.4 ± 95.7	0.388
Lymphocytes (×10^3/μL)	2.2 ± 0.9	2.2 ± 0.87	2 ± 0.92	0.266
Neutrophils (×10^3/μL)	6.5 ± 3.4	6.4 ± 3.3	6.9 ± 3.5	0.450
Creatinine (mg/dL)	0.88(0.79-1.1)	0.88 (0.77-1.05)	0.92 (0.82-1.3)	0.136
Glucose (mg/dL)	157.3 ± 86.8	152.2 ± 80.9	184.3 ± 110.7	**0.078**
Total cholesterol (mg/dL)	180.5 ± 50.8	178.8 ± 52.1	189.6 ± 42.9	0.310
LDL cholesterol (mg/dL)	103.1 ± 38.3	101.2 ± 38.9	113 ± 33.6	0.144
HDL cholesterol (mg/dL)	40.1 ± 10.5	40.5 ± 10.9	38.3 ± 8.5	0.315
Triglycerides (mg/dL)	156 (104-220)	156.5 (100.3- 221.5)	156 (118-209)	0.803
Uric acid (mg/dL)	6.3 ± 2.1	6 ± 1.8	8.1 ± 2.6	**<0.001**
Albumin (g/dL)	3.63 ± 0.44	3.65 ± 0.40	3.53 ± 0.59	0.200
Uric Acid/Albumin ratio	1.76 ± 0.68	1.66 ± 0.55	2.28 ± 0.99	**<0.001**
Outcomes, n(%)				
Follow-up duration (months)	32 (20-51.5)	32 (20-48.3)	32 (18-55)	0.702
Mortality, n (%)	31 (18.3)	16 (11.3)	15 (55.6)	**<0.001**

Data are presented as mean ± standard deviation, median (interquartile range), or number (percentage).

Abbreviations: CIN, contrast-induced nephropathy; LDL, low-density lipoprotein; HDL, high-density lipoprotein. Statistical significance is considered at a p-value of less than 0.05.

The two groups demonstrated no marked differences regarding the majority of demographic and laboratory parameters. However, serum uric acid levels ($p < 0.001$), UAR ($p < 0.001$), and total mortality rate (55.6% vs. 11.3%, $p < 0.001$) were considerable higher in the CIN (+) group. In the CIN (+) group, LVEF was importantly lower ($p = 0.002$), and the prevalence

of CKD was substantially higher contrasted with the CIN (−) group (14.8% vs. 3.5%, p = 0.037) (Table I).

The operational features of each group are presented in Table II. Among the angiographic parameters, Patients in the CIN-positive group received a markedly higher amount of contrast medium (p = 0.040). Furthermore, patients in the CIN (+) group had a higher frequency of multiple CTOs (22.2% vs. 4.9%, p = 0.002). While the frequency of CTO in the right coronary artery (RCA) and left anterior descending (LAD) was comparable between groups, the frequency of CTO in the circumflex artery (CX) was importantly higher in the CIN (+) group (33.3% vs. 12.7%, p = 0.007). The groups did not differ importantly with respect to other angiographic characteristics (Table II).

Table II: The operational characteristics of the study population

Variables	*All patient (n=169)*	*CIN (-) (n=142)*	*CIN (+) (n=27)*	*P value*
Angiographic and Procedural Characteristics				
Total stent length (mm)	48.4 ± 19.8	48.9 ± 19.1	45.3 ± 23.7	0.451
Total stent diameter (mm)	2.87 ± 0.33	2.87 ± 0.33	2.83 ± 0.33	0.870
SYNTAX score	16.3 ± 6.2	16.3 ± 6.3	16.5 ± 5.8	0.883
Contrast volume (mL)	198 ± 78.4	192.6 ± 75	226.3 ± 90.4	**0.040**
Procedure duration (minutes)	62.6 ± 35.5	63.1 ± 36.4	60.1 ± 30.5	0.690
Procedural success, n (%)				
Successful	131 (77.5)	108 (76.1)	23 (85.2)	0.450
Failed	38 (22.5)	34 (23.9)	4 (14.8)	
Lesion and Vessel Characteristics, n(%)				
One vessel	156 (92.3)	135 (95.1)	21 (77.8)	**0.002**
Two vessels	13 (7.7)	7 (4.9)	6 (22.2)	
Vessel distribution, n (%)				
LAD	74 (43.8)	62 (43.7)	112 (44.4)	0.940
CX	27 (16)	18 (12.7)	9 (33.3)	**0.007**
RCA	81 (47.9)	69 (48.6)	12 (44.4)	0.693
Total lesion length, n (%)				
<20 mm	65 (38.5)	56 (39.4)	86 (60.6)	0.550
>20 mm	104 (61.5)	9 (33.3)	18 (66.7)	
Number of diseased vessels, n (%)				
1	59 (34.9)	54 (38)	5 (18.5)	
2	61 (36.1)	49 (34.5)	12 (44.4)	0.149
3	49 (29)	39 (27.5)	10 (37)	
Diseased vessel, n (%)				
LAD	130 (76.9)	107 (75.4)	23 (85.2)	0.266
CX	84 (49.7)	66 (46.5)	18 (66.7)	0.054
RCA	114 (67.5)	96 (67.6)	18 (66.7)	0.924
Lesion Complexity and Calcification				
Calcification, n (%)				
Present	44 (26)	37 (26.1)	7 (25.9)	0.989
Absent	125 (74)	105 (73.9)	20 (74.1)	
JCTO skor				
0 (easy)	15 (8.9)	11 (7.7)	4 (14.8)	
1 (intermediate)	30 (17.8)	24 (16.9)	6 (22.2)	0.432
2 (difficult)	47 (27.8)	43 (30.3)	4 (14.8)	
3 (very difficult)	77 (45.6)	64 (45.1)	13 (48.1)	
Post-procedure TIMI flow, n (%)				
TIMI 1	1 (0.7)			
TIMI 2	5 (3.7)			
TIMI 3	128 (95.5)			

Data are presented as mean ± standard deviation, median (interquartile range), or number (percentage).

Abbreviations: CIN, contrast-induced nephropathy; SYNTAX, Synergy Between PCI With Taxus and Cardiac Surgery; LAD, left anterior descending artery; CX, circumflex artery; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction; J-CTO, Japanese Chronic Total Occlusion score. Statistical significance is considered at a p-value of less than 0.05.

To explore independent determinants of CIN, univariate and multivariate logistic regression analyses were applied. UAR remained a important independent predictor of CIN development (OR: 2.322; 95% CI: 1.202–4.485; p = 0.012) (Table III).

Table III: Independent Predictors of Contrast-Induced Nephropathy (CIN) by Univariate and Multivariate Logistic Regression Analysis

Variable	*Univariate analysis*		*Multivariate analysis*	
	OR (95% CI)	*p-value*	*OR (95% CI)*	*p-value*
Chronic Kidney Disease (CKD)	*4.765(1.190-19.074)*	***0.027***	*2.139(0.436-10.498)*	*0.349*
Ejection Fraction (EF)	*0.946(0.911-0.981)*	***0.003***	*0.965(0.924-1.007)*	*0.097*
Uric Acid/Albumin Ratio	*3.218(1.763-5.871)*	***<0.001***	*2.322(1.202-4.485)*	***0.012***
Contrast Volume (ml)	*1.005(1.000-1.010)*	***0.045***	*1.004(0.999-1.010)*	*0.098*
Total Number of Vessels	*5.510(1.687-17.993)*	***0.005***	*3.092(0.796-12.022)*	*0.103*

Statistical significance is considered at a p-value of less than 0.05.

The ability of UAR to predict CIN was analyzed by ROC curve analysis (Figure 1). ROC analysis demonstrated an AUC of 0.688 (95% CI: 0.577–0.800; p = 0.002). A UAR cut-off point of 1.77 predicted CIN with 66.7% sensitivity and 62% specificity (Central Illustration). In addition, ROC curve analysis was used to evaluate the ability of UAR to predict long-term mortality. The AUC was 0.706 (95% CI: 0.599–0.813; p < 0.001). A cut-off value of 1.90 demonstrated 64.5% sensitivity and 73.9% specificity for predicting all-cause long-term mortality (Figure 2) (Central Illustration).

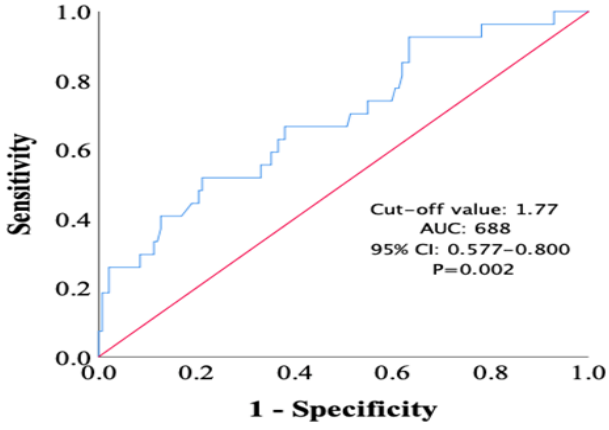


Figure 1. Receiver Operating Characteristic (ROC) Curve Analysis of Uric Acid/Albumin Ratio Predicting Contrast-Induced Nephropathy (CIN) with a Cut-off Value of 1.767, Sensitivity of 66.7%, and Specificity of 62%.

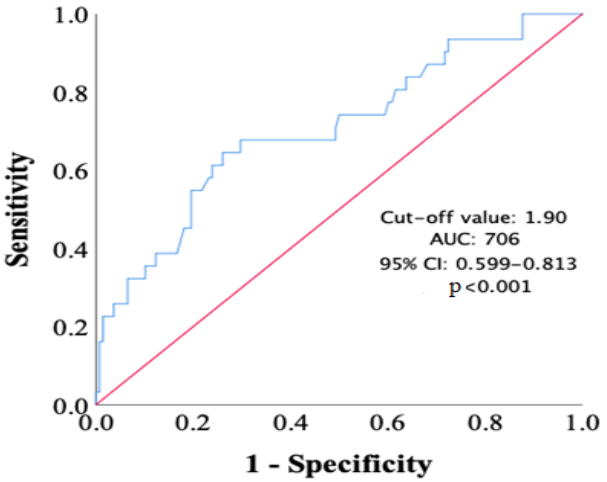
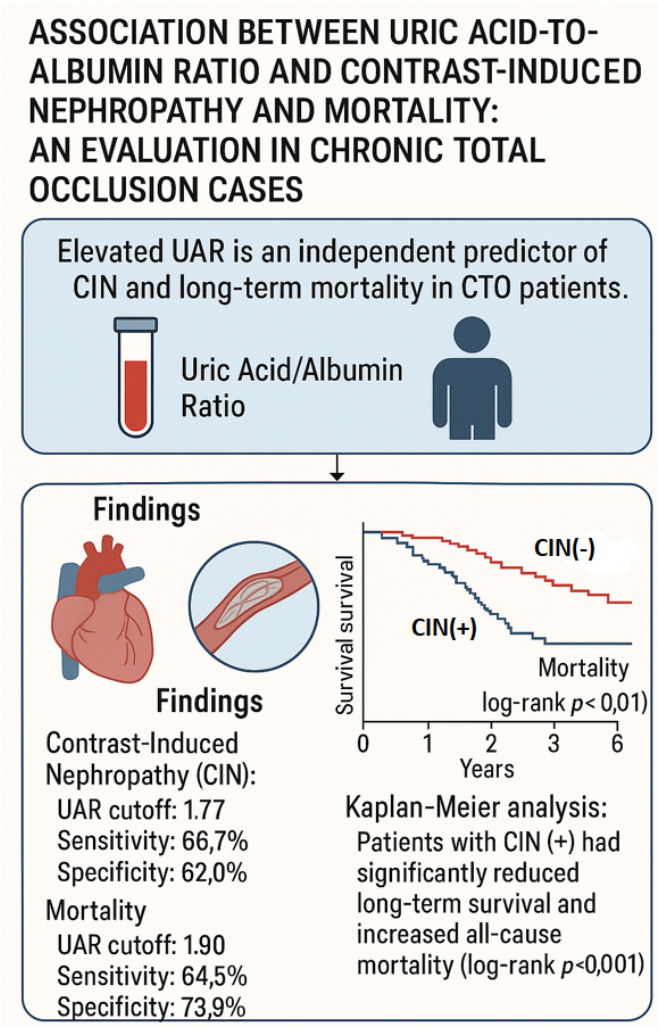


Figure 2. Receiver Operating Characteristic (ROC) Curve Analysis of Uric Acid/Albumin Ratio Predicting Mortality with a Cut-off Value of 1.90, Sensitivity of 64.5%, and Specificity of 73.9%.



Central Illustration: Association Between Uric Acid-to-Albumin Ratio and Contrast-Induced Nephropathy and Mortality: An Evaluation in Chronic Total Occlusion Cases

According to Kaplan–Meier survival curves, the CIN-positive cohort experienced significantly worse long-term survival and greater all-cause death risk (log-rank, p < 0.001) (Central Illustration). Likewise, patients with higher UAR values showed significantly lower survival rates and increased mortality over the follow-up period (log-rank, p = 0.001) (Figure 3).

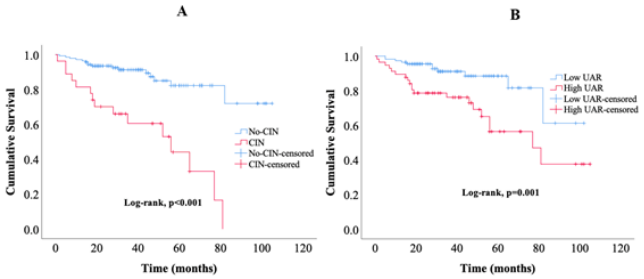


Figure 3. Kaplan–Meier Survival Curves Comparing Long-Term Survival and All-Cause Mortality Between CIN (+) and CIN (–) Groups(A) (Log-Rank Test, p < 0.001) and Between Patients With High Versus Low Uric Acid/Albumin Ratio (UAR) Values (B)(Log-Rank Test, p = 0.001).

DISCUSSION

This study introduces the novel application of the serum UAR for predicting occlusion (CTO) undergoing PCI. The principal findings can be summarized as follows: 1) UAR was identified as an distinct predictor of CIN onset following PCI in patients with CTO; 2) patients who develop CIN demonstrate reduced long-term survival; 3) UAR not only predicts CIN occurrence but also prognosis, with increasing UAR levels connected to an increased risk of long-term all-cause mortality.

Serum uric acid (UA), the final metabolite of purine catabolism, has a marked role in the initiation and progression of CAD, and increased UA levels have been documented in patients with CTO[12]. By enhancing oxidative stress and stimulating inflammation, hyperuricemia contributes to endothelial impairment, microvascular damage, and atherosclerosis, potentially facilitating CTO formation[12,13]. Additionally, elevated serum UA has been recognized as an independent determinant of CIN development[7]. Increased UA levels are also associated with various pathological mechanisms, including nitric oxide system inhibition and stimulation of the renin-

angiotensin-aldosterone system, alongside the oxidative stress, inflammation, and endothelial dysfunction described above[14]. Considering that CIN pathogenesis involves renal vasoconstriction, impairment of endothelial function, cellular damage, and resulting medullary hypoxia[15,16] elevated serum UA appears to share common pathogenic mechanisms with CIN. Moreover, it should be acknowledged that serum uric acid levels are influenced by hydration status. Previous studies have demonstrated that volume depletion reduces uric acid excretion independently of urine flow, thereby increasing serum UA concentrations[17]. In addition, another study including children aged 7–18 years with acute gastroenteritis reported higher serum UA levels in patients with moderate to severe dehydration, suggesting that UA may serve as an adjunct marker for assessing fluid deficit[18]. In this context, elevated UAR values in our CTO cohort could partially reflect dehydration, which is a known risk factor for CIN. However, given that no patients under 18 years of age were included in our study and that all patients underwent PCI only after an adequate assessment of hydration status, this potential confounder is unlikely to have influenced our findings.

As a negative acute-phase protein, albumin has antioxidant capacity and is recognized as a potent eliminator of free oxygen radicals[19]. Albumin levels decrease in inflammatory states[20]. Given that inflammation increases the likelihood of CIN development[21], it is expected that patients who develop CIN after interventional procedures have lower serum albumin levels[9]. Since both high uric acid and low albumin levels have been shown to predict CIN[7,9], the predictive value of UAR for CIN has been evaluated in various cardiovascular disease populations. For example, Şaylık et al. reported that STEMI patients who developed CIN after primary PCI had significantly higher UAR values compared to those who did not, and they identified a cutoff value of 1.62[22]. Similarly, Demirci et al. found elevated UAR levels in patients with PAD who underwent endovascular intervention and developed CIN, and reported a cutoff value of 1.19[23]. Yeter et al. reported that in intensive care unit patients, an association was observed between UAR, AKI, and mortality, and a threshold of 1.7 was determined[24]. Our study assessed the relationship of UAR with the occurrence of CIN among CTO patients, and concluded that a UAR cutoff value of 1.77 was predictive of CIN occurrence.

Beyond predicting CIN, the relationship between UAR and mortality has also been studied in some cardiovascular disease groups. Kalkan et al. demonstrated that UAR independently predicts mortality in STEMI patients[25], while Wang et al. demonstrated UAR as a prognostic indicator in aortic dissection[26]. Özgür et al. reported that in patients with AKI, higher UAR levels were significantly linked to increased short-term mortality[27]. In line with these findings, our study demonstrated that elevated UAR levels were associated with increased long-term mortality.

In lesions requiring complex interventions such as CTO, it has been shown that the risk of developing CIN increases proportionally with the prolongation of the procedure time and the increase in the volume of contrast administered[28]. Furthermore, patients who develop CIN exhibit higher mortality rates compared to those without CIN.28 Our findings align with existing literature showing a strong relationship between CIN and mortality[29].

Contrast volume (CV) represents an important risk factor for CIN. In patients with renal impairment and reduced glomerular filtration rate (GFR), minimizing contrast dose is crucial to lower the incidence of CIN[30]. The study by Lee et al. demonstrated that utilizing safe threshold

levels for contrast volume may effectively reduce the risk of renal injury[31]. Although contrast volume was significantly different between CIN (+) and CIN (–) groups in our study, it did not emerge as an independent predictor of CIN in multivariable regression analyses. Moreover, the use of low-osmolar contrast agents—selected in this study—has been associated with a reduced incidence of CIN when contrasted to high-osmolar agents[32].

Among the limitations of this study are its retrospective nature, single-center scope, the relatively small number of participants, and the absence of evaluation of certain CIN risk factors, particularly proteinuria and nephrotoxic drug exposure. Additionally, UAR values were limited to pre-procedural measurements, and possible dynamic changes during follow-up were not evaluated. Assessment of peri- and post-procedural variations in the UAR could provide valuable insights for both the staging of acute kidney injury and the improvement of prognostic prediction. Future large-scale, multicenter studies may focus on establishing threshold values for UAR, assessing its dynamic changes during prospective follow-up, and comparing its prognostic utility with other established biomarkers.

CONCLUSIONS

In conclusion, our study demonstrated that the uric acid-to-albumin ratio is markedly relationship with CIN and mortality in patients with chronic total occlusion undergoing PCI. Implementing prophylactic strategies before the procedure and close post-procedural monitoring of these patients may help reduce adverse events.

Ethical approval: The protocol of this investigation received authorization from the local ethics committee (Approval No: 2025/4-38; Date: April 22, 2025). The conduct of this study strictly followed the ethical principles established by the Declaration of Helsinki (2024 version).

Conflict of Interest: The authors declared no conflicts of interest.

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