

Evaluation of The Relationship Between Uric Acid Level and Uric Acide/Albumin Ratio With Stent Thrombosis In Patients Presenting Acute Coronary Syndrome

Ahmet Ferhat Kaya^{1*}, Adem Aktan², Remzi Sarıkaya¹

¹Van Bölge Eğitim Araştırma Hastanesi

²Mardin Artuklu University

ABSTRACT

This study aimed to evaluate the relationship between uric acid level and uric acide/albumin ratio (UAR) and stent thrombosis in patients with acute coronary syndrome (ACS). This single-center retrospective cohort study included 142 patients who underwent interventional treatment with stent implantation for ACS. The patients were divided into two groups: those who developed stent thrombosis within one year (n=67) and those who did not (n=75). Demographic, clinical, and laboratory data were collected from the hospital records. The uric acid/albumin ratio was calculated. Stent thrombosis is significantly associated with diabetes and hyperlipidemia.(respectively p=0,001, p=0,002) Uric acid levels were significantly higher and UAR was significantly lower in the stent thrombosis group. (respectively p= 0,001, p=0,001) The development of no reflow and post-dilatation was more frequent in the stent thrombosis group. ROC analysis revealed that a uric acid cut-off value of 5.55 had a sensitivity of 59.7% and a specificity of 64% for predicting stent thrombosis. The relationship between uric acid levels, UAR, and stent thrombosis likely involves complex processes including inflammation and oxidative stress. Understanding these relationships may contribute to the development of targeted interventions to reduce the risk of stent thrombosis and improve the outcomes of ACS patients undergoing stent implantation.

Keywords: Stent thrombosis, Acute coronary syndrome, Uric acid levels

Introduction

High serum uric acid levels have been associated with many diseases, particularly cardiovascular disease (1). Uric acid increases oxidative stress and inflammation, leading to structural deterioration of the vascular wall and proliferation of vascular smooth muscle cells (2). These effects are closely associated with the development, severity, and progression of atherosclerosis.

Interventional methods play a significant role in treating of diseases (1). Stent thrombosis, which occurs after stent placement in patients undergoing percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS), is a significant complication that can result in substantial morbidity and mortality. Despite advances in stent technology, stent thrombosis occurs in 5–20% of cases and remains clinically significant (3, 4). This condition increases healthcare costs and negatively affects patient prognoses. Many factors contribute to the

development of stent thrombosis, including resistance to antiplatelet therapy, stent type and placement, treatment duration, and postprocedural care (5, 6).

In addition to uric acid, serum albumin levels are also important biomarkers of cardiovascular processes. Albumin constitutes a large proportion of serum proteins and exhibits antioxidant, anti-inflammatory, anticoagulant, and anti-aggregant properties (7). As a negative acute-phase reactant, albumin plays a protective role against thrombotic events by inhibiting platelet aggregation and activation (8, 9). Low albumin levels are associated with increased blood viscosity and impaired endothelial function (10).

In recent years, the uric acid-to-albumin ratio (UAR), which combines uric acid and albumin levels, has been proposed as an indicator of oxidative stress and associated with various cardiovascular conditions. UAR is considered a potential marker for predicting adverse

*Corresponding Author: Ahmet Ferhat Kaya, Department of Cardiology, Van Regional Education and Research Hospital, Van, Turkey
Mail: kayaferhatahmet@gmail.com, Phone: +90 (507) 123 64 74

ORCID ID: Ahmet Ferhat Kaya: 0000-0003-0544-0657, Adem Aktan: 0000-0003-0505-9784, Remzi Sarıkaya: 0000-0003-4774-3103

Received: 05.12.2024, Accepted: 29.09.2025

cardiovascular events and determining the severity of coronary artery disease (11–14).

This study aimed to evaluate the relationship between serum uric acid levels and UAR in patients with stent thrombosis and to investigate the possible underlying mechanisms.

Material and Method

Study Population: This study was conducted using a single-center retrospective cohort design. The study included 67 patients who underwent interventional treatment for acute coronary syndrome, followed by re-interventional treatment for stent thrombosis within one year, as well as 75 patients who were managed with medical therapy for one year and did not require re-intervention.

Patients' clinical and demographic data were obtained through a hospital registry search. The following data were collected: age, sex, body mass index (BMI), heart rate, hypertension (HTN), diabetes mellitus (DM), alcohol use, hyperlipidemia (HLP), smoking, chronic obstructive pulmonary disease (COPD), ejection fraction (EF), length of hospital stay, and type of myocardial infarction.

Patients with advanced heart failure, severe liver or kidney failure, malignancy, hematological diseases, inflammatory or autoimmune diseases, pregnancy, gout, uric acid-lowering medication use, insufficient clinical data, angiography for acute coronary syndrome, or in-stent restenosis were excluded from the study.

Pre-angiographic data from all the patients were considered. This study was approved by the Institutional Ethics Committee. This study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Laboratory Tests: Laboratory data were obtained by scanning the hospital information system. White Blood Cell (WBC), urea, creatinine, C-Reactive Protein (CRP), Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), sodium, potassium, (high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglyceride, thyroid stimulating hormone (TSH), albumin, uric acid, hemoglobin (Hgb), platelet (Plt), Mean Corpuscular Volume (MCV), Red Cell Distribution (RDW), Mean Platelet Volume (MPV), and Platelet Distribution Width (PDW) were measured in blood collected from patients at the hospital. Uric acid/albumin ratio (UAR): The serum uric acid value was calculated by dividing

the value by the serum albumin value ($\mu\text{mol/g}$) (UAR).

Results

A priori power analysis was performed using G*Power (version 3.1.9.7) with an effect size of 0.5, alpha error of 0.05, and desired power of 0.80. The minimum required sample size was 128, indicating that at least 64 patients were required per group. Each group included more than 64 patients. The power of this study was 0.801. Patients who underwent coronary angiography for acute coronary syndrome and received interventional treatment (2nd generation generation stents available at our hospital) were included in this investigation.

This study included 142 patients. Of these, 95 (66.9%) were male. The mean patient age was 60.9 ± 12.6 years, and the follow-up period was 12 ± 6 months. The mean age of the men was 58.3 ± 1.3 years, and the mean age was 66.3 ± 1.5 years. During follow-up, stent thrombosis developed in 67 patients but did not develop in 75 patients. The patients were divided into two groups. There were no differences between the groups in terms of sex ($p=0.065$), age, BMI, heart rate, hypertension, smoking status, COPD, ejection fraction, length of hospital stay, or myocardial infarction type ($p>0.05$). Diabetes ($p=0.001$) and hyperlipidemia ($p=0.002$) were significantly higher in patients with stent thrombosis. Alcohol use was present only in the thrombosis group, but the difference was not significant ($p=0.159$). The demographic and clinical data are summarized in Table 1.

The laboratory findings of patients who did and did not develop stent thrombosis are shown in Table 2. There were no significant differences in WBC, urea, creatinine, CRP, ALT, AST, sodium, potassium, HDL, LDL, triglyceride, TSH, albumin, hemoglobin, or other hematological parameters between the two groups ($p>0.05$). Uric acid levels were considerably higher in the group that developed stent thrombosis ($(5.1 (2.3-7.1) \text{ vs } 5.8 (3.6-10.4), p = 0.001)$). The UAR was observed to be significantly lower in the stent thrombosis group ($(0.8(0.57-1.7) \text{ vs } 0.7(0.3-1.2), p = 0.001)$). The platelet distribution width (PDW) was also significantly different between the groups ($p=0.044$). No association was found between other parameters and stent thrombosis. These results imply that uric acid, UAR, and PDW may be involved in the development of stent thrombosis.

Table 1: Demographic data of Patients Who Developed and Did Not Develop Stent Thrombosis

	Without Stent Thrombosis (n=75)	Stent Thrombosis (n=67)	p value
Gender (Female/Male), n(%)	30(%40)/45(%60)	17(%25,4)/50(%74,6)	0,065
Age, (years) mean±SD	60,59(±12,85)	62,45(±9,9)	0,189
BMI, (kg/m2) median(IQR)	25,3(20,8-33,5)	25,5(20,5-33,3)	0,594
HR, (minute) median (IQR)	73,8(54-105)	75,8(53-106)	0,345
HT, n(%)	45(%60)	46 (%68,7)	0,286
DM, n(%)	26(34,7)	44(%65,7)	0,001
Alcohol , n(%)	0(%0)	2(%3)	0,159
HPL, n(%)	35(%46,7)	48(%71,6)	0,002
SMOKING, n(%)	28(%37,3)	21(%31,3)	0,537
COPD, n(%)	4(%5,3)	2(%3)	0,502
EF, % median (IQR)	51,6(30-60)	48,7(25-65)	0,085
Hospitalization day, median (IQR)	3,2(2-5)	3,3(1-8)	0,574

BMI: Body mass index; HR: Heart Rate; HT: Hypertension, DM: Diabetes Mellitus, HPL: Hyperlipidemia, COPD: Chronic Obstructive Lung Disease, EF: Ejection Fraction

Table 3 provides a comparative analysis of the angiographic findings observed among patients who did and did not develop stent thrombosis. There were no statistically significant differences between the groups in terms of radial and femoral access rates, angiography duration, radiation dose (dose-area product and effective dose), and the amount of contrast material used ($p > 0.05$). However, the development of no reflow was significantly higher in the stent thrombosis group (34.3% vs. 5.3%; $p=0.001$). The present study found a significant increase in the prevalence of left anterior descending artery (LAD) involvement within the specified vascular distribution in subjects who did not experience stent thrombosis ($p = 0.004$). The post-dilatation rate was significantly higher in the stent thrombosis group (58.2% vs. 30.7%; $p=0.001$). No statistically significant differences were observed in terms of predilatation or PTCA alone ($P > 0.05$). These findings indicate that no reflow and postdilatation may be effective factors in the development of stent thrombosis.

In addition, the Receiver Operating Characteristic (ROC) analysis of uric acid and the uric acid/albumin ratio is presented in patients with and without stent thrombosis. The area under the curve (AUC) of uric acid was 0.715 (95% confidence interval [CI]: 0.632-0.798), with a cut-off value of 5.55 milligrams per deciliter (mg/dL) ($p=0.0001$). At this value, the sensitivity and specificity were 59.7% and 64%, respectively. The area under the curve (AUC) of the uric acid/albumin ratio was 0.685 (95% confidence

interval [CI]: 0.598-0.771), with a cutoff value of 1.34 ($p < 0.001$). The sensitivity and specificity were 62.7% and 62.7%, respectively. These two parameters demonstrated a moderate capacity to differentiate between patients with and without the risk of stent thrombosis (Table 4, Figure 1).

Statistical Analysis: The data were analyzed using SPSS version 25.0 for Windows (IBM Corp., Armonk, NY, USA). In this study, stent thrombosis was the primary outcome variable in the patients with ACS. Subjective and objective methods, specifically the Lilliefors and Shapiro-Wilk tests, were used to assess the normal distribution of continuous variables. Continuous variables are expressed as mean $\pm$ standard deviation (SD) or median (interquartile range), and categorical variables are expressed as percentages. Categorical variables were compared using the chi-squared test or Fisher's exact test, as appropriate. Numerical variables with a normal distribution were evaluated using the parametric Student's t-test, whereas those without a normal distribution were evaluated using the non-parametric Mann-Whitney U test. To estimate the cutoff value for uric acid, the specificity and sensitivity values were calculated using the receiver operating characteristic (ROC) curve. Statistical significance was set at $P < 0.05$.

Discussion

The present study demonstrated that elevated serum uric acid levels and an augmented uric acid/albumin ratio were significantly

Table 2: Laboratory Findings of Patients Who Developed and Did Not Develop Stent Thrombosis

	Without Stent Thrombosis (n=67)	Stent Thrombosis (n=75)	p value
WBC(x103/uL), median (IQR)	10,7(3,4-22)	10,5(4,8-17,2)	0,672
Urea , (mg/dL) , median (IQR)	38,7(17-139)	37(20-94)	0,522
Creatinine, (mg/dL) , median (IQR)	0,94(0,46-2,24)	0,9(0,36-2,46)	0,628
CRP(mg/dL) , median (IQR)	1,1(0,06-8,4)	1(0,4-11,9)	0,697
ALT(U/L), median (IQR)	26,1(5-200)	24,1(6,5-82)	0,605
AST(U/L) , median (IQR)	64,4(7-949)	57,8(5-563)	0,749
Sodium(mEq/L) , median (IQR)	137(129-144)	137(128-144)	0,683
Potassium(mEq/L) , median (IQR)	4,1(3,2-5,5)	4,1(2,9-5,1)	0,335
HDL cholesterol (mg/dl) , median (IQR)	39,8(23-75)	40(21-71)	0,942
LDL cholesterol (mg/dl) , median (IQR)	106(26-203)	103(28-207)	0,596
Triglyceride(mg/dl) , median (IQR)	183,5(54-1010)	149,5(44-656)	0,147
TSH(gr/L) , median (IQR)	2,9(0-68)	1,8(0,02-6,0)	0,289
Albumin (gr/L) , median (IQR)	4,0(2,7-5,3)	4,0(2,4-5,0)	0,677
Uric Acide(mg/dl) , median (IQR)	5,1(2,3-7,1)	5,8(3,6-10,4)	0,001
Uric Acid Ratio /Albumin, median (IQR)	01,2(0,58-1,75)	1,4(0,78-2,81)	0,001
Hgb(gr/L) , median (IQR)	14,5(10,1-18,0)	14,3(11,6-17,2)	0,572
Monocyte (103/mm3) , median (IQR)	0,7(0,1-1,6)	0,7(0,06-1,6)	0,824
Neutrophils (103/mm3) , median (IQR)	7,4(1,7-17)	7,4(2,4-15)	0,932
Lymphocyte (103/mm3) , median (IQR)	2,2(0,5-4,5)	2,0(0,4-6,8)	0,312
Plt (103/mm3) , median (IQR)	252(74-414)	236(113-376)	0,253
Mcv (fL) , median (IQR)	88(77-99)	89(79-127)	0,383
RDW(%),median (IQR)	11,9(10,3-20,6)	11,9(10,5-15,9)	0,677
MPV(fL) , median (IQR)	8,1(5,7-13,1)	8,2(6,2-13,9)	0,431
PDW(%),median (IQR)	19,4(17,3-23,2)	19,8(17,7-22,6)	0,044

WBC: White Blood Cell, CRP= C-Reactive Protein, ALT =Alanine Aminotransferase, AST =Aspartate Aminotransferase,HDL =High Density Lipoproteine, LDL =Low Density Lipoproteine,TSH = Thyroid Stimulating Hormone , Hgb= Hemoglobin, Plt= Platelet, MCV =Mean Corpuscular Volüm,RDW = Red Cell Distribution,MPV= Mean Platelet Volüme,PDW= Platelet Distribution Width

associated with the development of stent thrombosis in patients undergoing percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS). Despite advancements in stent design and implementation of dual antiplatelet therapy, stent thrombosis remains a serious complication, with a significant increase in mortality and morbidity (15,16). Consequently, there remains a compelling need for reliable biomarkers that can effectively stratify risk.

Hyperuricemia is not merely a metabolic byproduct; it triggers endothelial dysfunction,

increased oxidative stress, and vascular smooth muscle cell proliferation, creating a prothrombotic environment (17-19). Increased uric acid, in turn, facilitates endothelial damage and platelet aggregation by increasing reactive oxygen species (19, 20). This mechanism is consistent with the elevated UA levels observed in the present study, and supports the hypothesis that UA can serve as a potential indicator of thrombotic risk.

The SYNTAX score, a tool used to evaluate the integrity of coronary artery disease, has been demonstrated to be associated with inflammatory

Table 3: Angiographic Findings of Patients With and Without Stent Thrombosis

	Without Stent Thrombosis (n=75)	Stent Thrombosis (n=67)	p value
Radial acces/Femoral Access, n(%)	67(%89,3)/8(%10,7)	62(%92,5)/5(%7,5)	0,512
Angio Duration , min , median (IQR)	38,2(10-120)	40,8(10-120)	0,492
Radiation (Dose-area product)(Gy cm2) min , median (IQR)	2456,8(182-6035)	2407,3(331-6035)	0,807
Radiation (Effective Dose) (mSv) min, median (IQR)	15235,9(2844,0-88088,0)	13166,1(193,2-56511,0)	0,293
Amount of Contrast material,mL min, median (IQR)	211,2(50-450)	196,3(15-400)	0,303
Noreflow , n(%)	4 (%5,3)	23(%34,3)	0,001
	LAD	34(%45,3)	21(%31,3)
	CX	8(%10,7)	7(%10,4)
	RCA	18(%24)	11(%16,4)
Which Vessel ?, n(%)	LAD+CX	6(%8)	9(%13,4)
	LAD+RCA	7(%9,3)	8(%11,9)
	CX+RCA	1(%1,3)	4(%6)
	LAD+CX+RCA	1(%1,3)	7(%10,4)
Just Ptca, n(%)	2(%2,7)	5(%7,5)	0,202
Predilatation, n(%)	62(%82,7)	55(%82,1)	0,929
Postdilatation, n(%)	23(%30,7)	39(%58,2)	0,001

LAD: Left anterior descending artery; CX: Circumflex artery; RCA: Right Coronary Artery; PTCA; Percutaneous Transluminal Coronary Angioplasty

Table 4: ROC analysis table for uric acid and uric acid albumin ratio in patients with and without stent thrombosis

Risk Factor	AUC(%95)	Cut off	P value	Sensitivity (%)	Spesifity (%)
Uric Acide	0,715(0,632-0,798)	5,55	< 0.001	59,7	64
Uric Acide/Albumine Ratio	0,685(0,598-0,771)	1,34	0,001	62,7	62,7

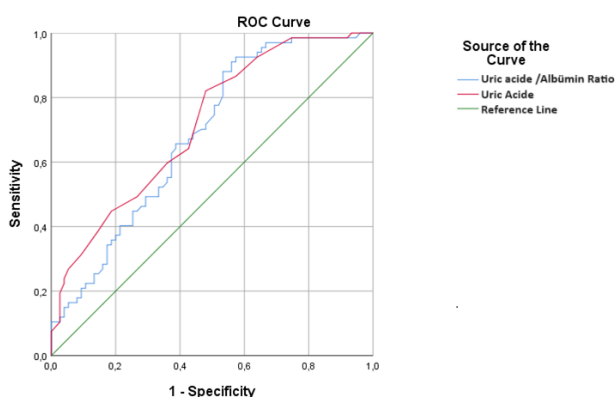


Image: ROC analysis image showing uric acid and uric acid albumin ratio in patients with and without stent thrombosis

markers, including uric acid and CRP (15,16). The validity of these relationships has been corroborated by studies conducted by Xing et al.

(16), whereas Wang et al. (17) demonstrated an augmented risk of vascular complications due to hyperuricemia in patients with diabetes. The relationship between diabetes and stent thrombosis in our study aligns with these previous findings.

The roles of oxidative stress and inflammation in stent thrombosis have been substantiated by delayed re-endothelialization and neointimal hyperplasia processes (18,19). As indicated in literature, uric acid has been reported to increase proinflammatory cytokine release and facilitate thrombus development (20). Moreover, hyperuricemia has been identified as an independent predictor of mortality in obstructive cerebrovascular disease (21).

Hyperalbuminemia provides vascular protection and is a potent antioxidant and anti-inflammatory agent (22-27). Conversely,

albumin deficiency reflects chronic inflammation is associated with cardiovascular risk (24-26). Therefore, the evaluation of uric acid and albumin provides a better understanding of oxidative stress and antioxidant balance (28).

In our study, cutoff values of 5.55 mg/dL for uric acid and 1.34 for the uric acid-to-albumin ratio predicted stent thrombosis with moderate sensitivity and specificity in the ROC analysis. These findings demonstrate that these parameters can be used for clinical risk assessments.

Postdilatation and multivessel intervention were more common in patients with stent thrombosis, while local endothelial damage combined with systemic risk factors increased the risk of thrombosis. Therefore, combining the evaluation of biomarkers and intervention characteristics is important for risk stratification.

Limitations: Our study had several limitations. First, the single-center, retrospective design and relatively small sample size limited the study's general validity. Therefore, multicenter studies with larger patient groups are required. Additionally, advanced imaging methods such as IVUS and OCT have not been used to evaluate stent thrombosis, which prevents comparisons between stent generations. Blood parameters were assessed only from blood samples obtained at presentation, and serial measurements were not performed.

Serum uric acid levels and uric acid-to-albumin ratio have been identified as significant factors in the development of stent thrombosis. It has been demonstrated that procedural factors, with a particular emphasis on post-dilatation and multivessel interventions, have the potential to elevate the risk of thrombosis. It has been demonstrated that uric acid and UAR possess the capacity to serve as predictors of stent thrombosis, exhibiting moderate sensitivity and specificity in this regard. These findings offer a promising basis for the development of novel approaches to clinical risk stratification. To enhance the generalizability of the findings, it is imperative to conduct comprehensive multicenter studies.

Declaration of Generative Ai and Ai-Enabled Technologies In The Writing Process: The authors declare that they did not use generative artificial intelligence or artificial intelligence-supported technologies during the writing process.

Ethics Committee Approval: The study was approved by the Local Ethics Committee with the

decision number 2024/11-2 ethics committee date: 5/11/2024

Conflict of Interest: The authors declare that they have no conflicts of interest.

Funding: This study was not financially supported by any organization.

Authors' Contributions

AFK: Conceptualization, Data curation, Formal Analysis, Writing the original draft, writing the review, and editing. **AFK:** Conceptualization, Data curation, Formal Analysis, Writing of the original drafts, writing the review, and editing. **AFK and AA:** Formal Analysis, Writing the original drafts. **AA,RS,** Conceptualization, Data curation, Writing- original draft.

References

- 1) Bhatt DL. Percutaneous coronary intervention in 2018. *JAMA* 2018; 319: 2127-2128.
- 2) Giacoppo D, Alfonso F, Xu B, Claessen BEPM, Adriaenssens T, Jensen C, *et al.* Paclitaxel-coated balloon angioplasty vs. drug-eluting stenting for the treatment of coronary in-stent restenosis: a comprehensive, collaborative, individual patient data meta-analysis of 10 randomized clinical trials (DAEDALUS study). *Eur Heart J* 2020; 41: 3715-3728.
- 3) Moussa ID, Mohanane D, Saucedo J, Stone GW, Yeh RW, Kennedy KF, *et al.* Trends and outcomes of restenosis after coronary stent implantation in the United States. *J Am Coll Cardiol* 2020; 76: 1521-1531.
- 4) Sakaguchi M, Hasegawa T, Ehara S, Matsumoto K, Mizutani K, Iguchi T, *et al.* New insights into spotty calcification and plaque rupture in acute coronary syndrome: an optical coherence tomography study. *Heart Vessels* 2016; 31: 1915-1922.
- 5) Yalçın, A. A., Topuz, M., Bıyık, İ., Aktürk, İ. F., Çelik, Ö., Işıksağan, N., ... & Uzun, F. (2014). Role of insulin-like growth factor 1 in stent thrombosis under effective dual antiplatelet therapy. *Advances in Interventional Cardiology*, 4, 242-249.
- 6) Tăpoi, L., Şalaru, D. L., Sascău, R. A., & Stătescu, C. (2021). Uric acid—an emergent risk marker for thrombosis?. *Journal of Clinical Medicine*, 10(10), 2062.
- 7) Arques S. Serum albumin and cardiovascular disease: state-of-the-art review. *Ann Cardiol Angeiol (Paris)* 2020; 69: 192-200.

- 8) Don BR, Kaysen G. Serum albumin: relationship to inflammation and nutrition. *Semin Dial* 2004; 17: 432-437.
- 9) Mikhailidis DP, Ganotakis ES. Plasma albumin and platelet function: relevance to atherogenesis and thrombosis. *Platelets* 1996; 7: 125-137.
- 10) Zhang WJ, Frei B. Albumin selectively inhibits TNF alpha-induced expression of vascular cell adhesion molecule-1 in human aortic endothelial cells. *Cardiovasc Res* 2002; 55: 820-829.
- 11) Yalcinkaya D, Karacali K, Ilhan BC, Yarlioglues M. Relation between serum uric acid to albumin ratio and severity of chronic coronary artery disease. *Angiology* 2023; 33197231161902.
- 12) Zhang Y, Xu Z, He W, Lin Z, Liu Y, Dai Y, *et al.* Elevated serum uric acid/ albumin ratio as a predictor of post-contrast acute kidney injury after percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction. *J Inflamm Res* 2022; 15: 5361-5371.
- 13) Selçuk M, Çınar T, Şaylık F, Akbulut T, Asal S, Çiçek V, *et al.* Predictive value of uric acid/albumin ratio for the prediction of new-onset atrial fibrillation in patients with ST-Elevation myocardial infarction. *Rev Invest Clin* 2022; 74: 156-164.
- 14) Çakmak EO, Bayam E, Çelik M, Kahyaoglu M, Eren K, Imanov E, *et al.* Uric acid-to-albumin ratio: a novel marker for the extent of coronary artery disease in patients with non-ST-elevated myocardial infarction. *Pulse (Basel)* 2021; 8: 99-107.
- 15) Yu W, Cheng JD. Uric acid and cardiovascular disease: an update from molecular mechanism to clinical perspective. *Front Pharmacol* 2020; 11: 582680.
- 16) Xing Y, Guo JT, Gai LY, Liu B, Luo DL. Association of Uric Acid and C-reactive Protein with the Severity of Coronary Artery Disease Using SYNTAX Score and Clinical SYNTAX Score. *Heart Surg Forum* 2019; 22: E247-252.
- 17) Wang JL, Qin Z, Wang ZJ, Shi DM, Liu YY, Zhao YX, *et al.* New predictors of in-stent restenosis in patients with diabetes mellitus undergoing percutaneous coronary intervention with drug-eluting stent. *J Geriatr Cardiol* 2018; 15: 137-145.
- 18) Zhu HY, Zhao SZ, Zhang ML, Wang Y, Pan ZM, Cheng HR, *et al.* Elevated serum uric acid increases the risk of ischemic stroke recurrence and its inflammatory mechanism in older adults. *Front Aging Neurosci* 2022; 14: 822350.
- 19) Jukema JW, Verschuren JJ, Ahmed TA, Quax PH. Restenosis after PCI. Part 1: pathophysiology and risk factors. *Nat Rev Cardiol* 2011; 9: 53-62.
- 20) Juni RP, Duckers HJ, Vanhoutte PM, Virmani R, Moens AL. Oxidative stress and pathological changes after coronary artery interventions. *J Am Coll Cardiol* 2013; 61: 1471-1481.
- 21) Zhu HY, Zhao SZ, Zhang ML, Wang Y, Pan ZM, Cheng HR, *et al.* Elevated serum uric acid increases the risk of ischemic stroke recurrence and its inflammatory mechanism in older adults. *Front Aging Neurosci* 2022; 14: 822350.
- 22) Lin GM, Li YH, Zheng NC, Lai CP, Lin CL, Wang JH, *et al.* Serum uric acid as an independent predictor of mortality in high-risk patients with obstructive coronary artery disease: a prospective observational cohort study from the ET-CHD registry, 1997–2003. *J Cardiol* 2013; 61: 122-127.
- 23) So A, Thorens B. Uric acid transport and disease. *J Clin Invest* 2010; 120: 1791-1799.
- 24) Xing Y, Guo JT, Gai LY, Liu B, Luo DL. Association of Uric Acid and C-reactive Protein with the Severity of Coronary Artery Disease Using SYNTAX Score and Clinical SYNTAX Score.
- 25) *Heart Surg Forum*. 2019 Jun; 22(3): E247–52. Kanbay M, Segal M, Afsar B, Kang DH, Rodriguez-Iturbe B, Johnson RJ. The role of uric acid in the pathogenesis of human cardiovascular disease. *Heart* 2013; 99: 759-766.
- 26) Jin M, Yang F, Yang I, Yin Y, Luo JJ, Wang H, *et al.* Uric acid, hyperuricemia and vascular diseases. *Front Biosci* 2012; 17: 656-669.
- 27) Don BR, Kaysen G. Serum albumin: relationship to inflammation and nutrition. *Semin Dial* 2004; 17: 432-437.
- 28) Arques S. Human serum albumin in cardiovascular diseases. *Eur J Intern Med* 2018; 52: 8-12.
- 29) Roche M, Rondeau P, Singh NR, Tarnus E, Bourdon E. The antioxidant properties of serum albumin. *FEBS Lett* 2008; 582: 1783-1787.
- 30) Zhu L, Chen M, Lin X. Serum albumin level for prediction of all-cause mortality in acute coronary syndrome patients: a meta-analysis. *Biosci Rep* 2020; 40: BSR20190881.