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RELATIONSHIP BETWEEN URIC ACID TO ALBUMIN RATIO AND IN-STENT RESTENOSIS IN PATIENTS WITH CORONARY ARTERY DISEASE UNDERGOING DRUG-ELUTING STENTING

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ABSTRACT

Background: In-stent restenosis remains an important complication following percutaneous coronary intervention despite advances in drug-eluting stent technology. The uric acid-to-albumin ratio combines serum uric acid and albumin, which may reflect metabolic, inflammatory, and vascular abnormalities associated with adverse coronary outcomes.

Objective: To determine the frequency of angiographically confirmed in-stent restenosis and to assess the relationship between serum uric acid-to-albumin ratio and in-stent restenosis in patients with coronary artery disease undergoing drug-eluting stent implantation**.**

Methods: A descriptive longitudinal study was conducted among adult patients with coronary artery disease undergoing index percutaneous coronary intervention with drug-eluting stent implantation. Serum uric acid and albumin were measured within 24–48 hours after PCI, and the uric acid-to-albumin ratio was calculated. Patients were followed for six months, with follow-up coronary angiography performed to identify in-stent restenosis, defined as ≥50% luminal diameter narrowing within the stent or within 5 mm of its proximal or distal edges. Statistical analysis was performed using SPSS version 25. Categorical variables were analyzed using the chi-square or Fisher's exact test, while continuous variables were compared using the independent-samples t-test or Mann–Whitney U test according to data distribution. A p-value <0.05 was considered statistically significant.

Results: A total of 161 patients were included. The mean age was 56.8 ± 9.7 years, and 104 (64.6%) patients were male. In-stent restenosis was identified in 22 (13.7%) patients at six-month follow-up. Patients with ISR had higher serum uric acid levels than those without ISR (7.1 ± 1.2 vs. 5.9 ± 1.1 mg/dL; p<0.001) and lower serum albumin levels (3.8 ± 0.4 vs. 4.1 ± 0.4 g/dL; p=0.002). The mean UAR was significantly higher among patients with ISR than among those without ISR (1.87 ± 0.38 vs. 1.45 ± 0.31; p<0.001). Longer stent length was also observed among patients with ISR (28.6 ± 7.4 vs. 25.1 ± 6.8 mm; p=0.026).

Conclusion: Higher uric acid-to-albumin ratio was associated with angiographically confirmed in-stent restenosis following drug-eluting stent implantation. UAR may serve as a readily available biomarker for identifying patients with an adverse post-PCI vascular profile. Larger prospective multicenter studies are required to determine its independent predictive value and potential role in routine post-PCI risk assessment.

KEYWORDS: Uric acid; albumin; uric acid-to-albumin ratio; in-stent restenosis; coronary artery disease; drug-eluting stent; percutaneous coronary intervention.

INTRODUCTION

Although important progress has been achieved in the field of preventive cardiology and coronary revascularisation, coronary artery disease (CAD) remains a major cause of morbidity and mortality world wide with a significant burden. The Global Burden of Disease 2021 estimates show that the burden of ischaemic heart disease (IHD) on the public health was still high in 2021 with about 254.3 million prevalent cases and 9.0 million deaths globally [1]. The treatment of obstructive CAD has significantly improved over the past few years with PCI (percutaneous coronary intervention) and drug eluting stents (DES), but in-stent restenosis (ISR) remains a significant late complication. Despite the improvements in the quality of the stents, current evidence suggests that ISR and repeat revascularisation continue to be a problem, with about 1-2% of target lesion revascularisation for ISR reported per year [2]. ISR is a multifactorial process that includes mechanical vascular injury, delayed endothelial healing, inflammation, oxidative stress, vascular smooth muscle cell proliferation, deposition of the extracellular matrix and in-stent neoatherosclerosis, particularly during follow-up [2,3]. Furthermore, the development of ISR may be affected by DM, lesion characteristics, stent size

and procedural factors. Identification of accessible markers that reflect the biological processes underlying the restenosis may help to better stratify the risk after PCI in this context. Uric acid has been linked to cardiovascular disease and can lead to oxidative stress, endothelial dysfunction, inflammation and adverse vascular remodelling [4]. On the other hand, serum albumin possesses antioxidant, anti-inflammatory and antithrombotic characteristics and is known to be one of the important biomarkers related to cardiovascular risk [5]. Therefore, the relationship between these parameters, namely uric acid/albumin ratio (UAR) could serve as a single biochemical indicator of oxidative/inflammatory stress. There is recent evidence that higher UAR is associated with severity of CAD [6] and coronary vascular abnormalities [7]. Most importantly, Liu et al investigated 503 patients who had DES implantation and discovered that UAR was an independent predictor of ISR with an area under the receiver operating characteristic curve of 0.767, which was superior to uric acid or albumin [8]. However, there is a lack of data on the UAR and ISR, particularly in South Asian populations. Thus, this study is aimed at determining the incidence of ISR after DES implantation and to assess the relationship between serum uric acid, UAR and ISR in CAD post PCI.

METHODOLOGY

This descriptive longitudinal study was done in Department of Cardiology at Pakistan Institute of Medical Sciences (PIMS) in Islamabad, Pakistan for six months after approval from College of Physicians and Surgeons Pakistan (CSPS). The study population comprised all adult patients (18–80 years old) with coronary artery disease who underwent index PCI with DES implantation. The subjects were followed up for 6 months, and the rate of in-stent restenosis (ISR) and the relationship between uric acid-to-albumin ratio (UAR) and ISR were evaluated. Non-probability consecutive sampling technique was used to recruit the eligible participants based on the pre-defined inclusion and exclusion criteria. Sample size was estimated using WHO sample size calculator for estimation of single population proportion. The sample size was calculated according to the standard deviation of a previous study that studied the role of uric acid to albumin ratio in predicting the rate of repeat revascularisation in patients with acute coronary syndrome with a population proportion of 11.9% for repeat revascularisation/in-stent restenosis. The minimum sample size required was calculated to be 161 patients with an absolute precision of 5% with a confidence level of 95%. Sample size was to be expanded to allow for some loss to follow-up. Patients were included if they were 18–80 years of age, had a diagnosis of coronary artery disease, had been treated for index PCI and DES implantation and were able to adhere to the 6 months follow-up protocol. Patients were not eligible to enroll if they had severe renal impairment (eGFR <30 mL/min/1.73 m2), chronic liver disease, serum albumin <3.0 g/dL (other than acute illness), active infection, malignancy, or an allergy or hypersensitivity to any procedure used in this study at the time of enrolment. If a patient met the criteria, he was consecutively included after approval from the CPSP and the institutional ethical review committee, after having received index PCI with DES implantation. All participants gave written informed consent before data collection. Baseline demographic, clinical, laboratory, procedural and angiographic data were obtained from the patient interview, physical examination, medical records and laboratory reports. Demographic and clinical parameters were age, sex, height, weight, BMI, diabetes mellitus, smoking status and LDL cholesterol. Blood was drawn after 24–48 hours of fasting and serum uric acid and serum albumin were determined by standard laboratory techniques. The UAR was calculated as the ratio of serum uric acid level (mg/dL) to serum albumin level (g/dL). Data recorded were procedural and angiographic data, lesion characteristics, length of stent, stent diameter, stent number, and target vessel. Follow up at 1, 3 and 6 months. Symptoms, adverse cardiac events, medication adherence, repeat angiography and other complications were recorded. At 6 months follow-up, coronary angiography was performed for the evaluation of ISR. ISR was defined as narrowing of the luminal diameter ≥ 50% inside the stent or in a distance of 5 mm from the edges of the stent. The uric acid level and the UAR were compared between ISR and non-ISR patients. Data on potential confounding variables such as age, sex, diabetes mellitus, and smoking status were obtained, along with variables related to the stents themselves. Selection bias was minimized by consecutive sampling, information bias was minimized by the use of standardised data collection methods and observer bias was minimized by blinded angiographic assessment. All the data was recorded on a standard proforma and securely stored. ISR was the primary outcome variable while UAR was the main study variable. Serum uric acid and serum albumin were also excluded separately. The other study variables were age, sex, height, weight, body mass index, diabetes mellitus, smoking status, LDL cholesterol, number of stents, stent length, stent diameter, location of the lesion, adherence to medication and adverse cardiac events, as well as other complications. Data were analyzed by the software of Statistical Package for Social Sciences (SPSS) version 25. The frequencies and percentages were used to represent the qualitative variables, and the relationship between two categorical variables was evaluated using the chi-square test or Fisher's exact test, depending on the sample size. The Shapiro-Wilk test was used to test the normality of quantitative variables. Data for quantitative variables with a normal distribution was reported as mean ± SD and for those with a non-normal distribution as a median with interquartile range. Independent-samples t-test was used to compare normally distributed quantitative variables with and without ISR, and the Mann–Whitney U test was used for non-normally distributed variables. The level of p values < 0.05 was considered statistically significant. The study was conducted after approval from the College of Physicians and Surgeons Pakistan and institutional ethical review committee. All participants gave informed written consent prior to enrolment and data

collection. All participation was voluntary with the participant's information kept confident throughout the study. All study data were collected on standard proforma and kept securely.

RESULTS

A total of 161 patients with coronary artery disease who underwent percutaneous coronary intervention with drug-eluting stent implantation were included in the analysis. The mean age of the study population was 56.8 ± 9.7 years. Among the participants, 104 (64.6%) were male and 57 (35.4%) were female. The mean body mass index was 27.1 ± 3.8 kg/m². Diabetes mellitus was present in 67 (41.6%) patients, while 59 (36.6%) were current smokers. The mean LDL cholesterol level was 104.6 ± 28.7 mg/dL. The baseline demographic and clinical characteristics are presented in Table 1.

At 6-month follow-up, angiographically confirmed in-stent restenosis was identified in 22 (13.7%) patients, while 139 (86.3%) patients did not develop ISR. Thus, the overall frequency of ISR was 13.7%. The distribution of patients according to ISR status is presented in Table 2.

Patients with ISR had a mean serum uric acid level of 7.1 ± 1.2 mg/dL compared with 5.9 ± 1.1 mg/dL among patients without ISR. This difference was statistically significant ($p<0.001$). Mean serum albumin concentration was 3.8 ± 0.4 g/dL among patients with ISR and 4.1 ± 0.4 g/dL among those without ISR ($p=0.002$). The comparison of serum uric acid and serum albumin according to ISR status is presented in Table 3.

The mean uric acid-to-albumin ratio was higher among patients who developed ISR than among those who did not develop ISR (1.87 ± 0.38 vs. 1.45 ± 0.31, respectively; $p<0.001$). The distribution and comparison of UAR according to ISR status are presented in Table 4.

Regarding procedural and angiographic characteristics, the mean stent length was 28.6 ± 7.4 mm among patients with ISR compared with 25.1 ± 6.8 mm among patients without ISR ($p=0.026$). Multivessel stenting was observed in 9 (40.9%) patients with ISR compared with 32 (23.0%) patients without ISR ($p=0.083$). The mean stent diameter was 2.9 ± 0.4 mm in the ISR group and 3.0 ± 0.4 mm in the non-ISR group ($p=0.318$). Other angiographic and procedural characteristics are presented in Table 5.

Categorical variables were expressed as frequencies and percentages and were compared using the chi-square test or Fisher's exact test where appropriate. Continuous variables were assessed for normality using the Shapiro–Wilk test and were expressed as mean ± standard deviation when normally distributed or median with interquartile range when non-normally distributed. Comparisons between patients with and without ISR were performed using the independent-samples t-test or Mann–Whitney U test, as appropriate. A p-value <0.05 was considered statistically significant.

The analysis demonstrated a higher serum uric acid level and higher uric acid-to-albumin ratio among patients with angiographically confirmed ISR compared with patients without ISR. The findings corresponded to the prespecified objectives of determining the frequency of ISR and comparing serum uric acid and UAR between the two groups.

Table 1. Baseline demographic and clinical characteristics of the study population (n=161)

Variable	Value
Age, years, mean ± SD	56.8 ± 9.7
Male, n (%)	104 (64.6)
Female, n (%)	57 (35.4)
BMI, kg/m², mean ± SD	27.1 ± 3.8
Diabetes mellitus, n (%)	67 (41.6)
Current smoking, n (%)	59 (36.6)
LDL cholesterol, mg/dL, mean ± SD	104.6 ± 28.7

Table 2. Frequency of in-stent restenosis at 6-month follow-up (n=161)

ISR status	n	%
ISR present	22	13.7
ISR absent	139	86.3
Total	161	100.0

Table 3. Comparison of serum uric acid and serum albumin according to ISR status

Variable	ISR (n=22)	No ISR (n=139)	p-value
Serum uric acid (mg/dL), mean ± SD	7.1 ± 1.2	5.9 ± 1.1	<0.001
Serum albumin (g/dL), mean ± SD	3.8 ± 0.4	4.1 ± 0.4	0.002

Table 4. Comparison of uric acid-to-albumin ratio according to ISR status

Variable	ISR (n=22)	No ISR (n=139)	p-value
UAR, mean ± SD	1.87 ± 0.38	1.45 ± 0.31	<0.001

Table 5. Comparison of angiographic and procedural characteristics according to ISR status

Variable	ISR (n=22)	No ISR (n=139)	p-value
Stent length (mm), mean ± SD	28.6 ± 7.4	25.1 ± 6.8	0.026
Multivessel stenting, n (%)	9 (40.9)	32 (23.0)	0.083
Stent diameter (mm), mean ± SD	2.9 ± 0.4	3.0 ± 0.4	0.318

DISCUSSION

The aim of the present study was to investigate the association between serum uric acid-to-albumin ratio (UAR) and angiographically confirmed in-stent restenosis (ISR) after drug-eluting stent implantation. The primary result was that serum uric acid level was elevated and serum albumin was decreased in patients with ISR, which led to a significantly elevated UAR in patients with ISR as compared to patients without ISR. This finding is biologically plausible since uric acid-related oxidative and endothelial stress, as well as albumin-related inflammatory and nutritional status, are two variables with different, but potentially complementary aspects of cardiovascular risk, that have been combined in the UAR. In the recent cardiovascular literature, UAR has been examined as a composite marker and not uric acid or albumin alone. [11–13] The finding of an association between UAR and ISR in this study is in line with the evidence showing a relationship of UAR with the severity and prognosis of coronary artery disease. Yalcinkaya et al. , found that patients with more extensive chronic coronary artery disease had higher uric acid concentrations, lower albumin concentrations and thus higher UAR values. Furthermore, UAR was independently related with augmented angiographic burden of disease[9,10,14]. In the same way, Wang et al. proposed urocortin as a new CV biomarker and its possible application in the synergistic action of uric acid (UA) and albumin (ALB) on cardiovascular damage. The composite biomarker was also found to be linked with poor long-term cardiac outcomes in patients with unstable angina who underwent PCI, further underscoring the overall cardiovascular relevance of the composite biomarker [12,13] The present results are also in line with studies evaluating UAR in other coronary phenotypes. Toprak et al. found that UAR was independently correlated with coronary slow flow and was more discriminatory than various conventional inflammation-based markers. In a study of stable coronary artery disease patients with chronic total occlusion (CTO), higher UAR was correlated with worse coronary collateral, confirming the relationship between UAR and unfavorable coronary vascular findings. These studies were not conducted to specifically assess ISR but did indicate that UAR reflects a systemic biologic milieu which is linked to coronary vascular dysfunction. Thus, the association seen in this study might not be just a result of stent restenosis but be a continuation of the association between UAR and coronary vascular pathology. Several biological mechanisms could be responsible for the relationship observed between UAR and ISR. The final product of purine metabolism is uric acid which has been associated with oxidative stress, endothelial dysfunction, and vascular smooth-muscle cell dysfunction and inflammation, particularly when elevated. Hiraga et al. demonstrated in ischaemic heart disease patients that serum uric acid level was inversely associated with systemic endothelial function, which is clinical evidence suggesting the association of high uric acid level and impaired vascular endothelial function. [16] Endothelial dysfunction is particularly important after PCI as stent implantation leads to mechanical injury and endothelial denudation with subsequent inflammatory and reparative responses in the treated vascular segment. ISR is a complex biological and mechanical process that is not one pathologic event. Modern literature regards vascular injury following a PCI as an inflammation trigger, smooth muscle cell migration and proliferation, extracellular matrix deposition, impaired endothelial recovery and, at later time, neoatherosclerosis. These processes can gradually narrow the lumen of the stented area or the area around it. Therefore, oxidative stress and inflammatory activation can be a potential mechanistic connection between systemic metabolic abnormalities and local vascular responses in the post-stent phase. Despite the present day technology of DES, Clare et al. pointed out that inflammation and redox stress are still relevant to restenosis. The mechanical injury, inflammation, endothelial healing, neointimal hyperplasia and neoatherosclerosis in ISR were described by Pelliccia et al. [17,18] The following biological pathway may contain additional information about Albumin. Besides its action on maintaining oncotic pressure in plasma and carrying several endogenous substances, Albumin is linked to systemic inflammatory and nutritional status. So the lower the level of albumin, the more inflammatory or physiologically stressed the patient. As uric acid goes up and/or albumin goes down, the UAR rises, thus potentially providing a broader biological context than either uric acid or albumin alone. This could explain the various cardiovascular outcomes and coronary angiographic features reported in recent studies as being associated with UAR. As these mechanistic pathways were not directly measured in this study, however, it was not possible to determine if inflammation, oxidative stress or endothelial dysfunction were involved

in the relationship between UAR and ISR. The connection of procedural characteristics and ISR should also be taken into account. In the present analysis, the patients with ISR had longer stents than patients without ISR. This is consistent with recent data which has shown stent length to be an important procedural determinant of DES related restenosis. A recent systematic review and meta-analysis showed that the overall incidence of DES-ISR was 13% and diabetes mellitus, stent length, number of stents, lesion length, left anterior descending artery involvement, previous myocardial infarction and previous PCI were significant risk factors. Increased surface area of the artery that is exposed to the mechanical injury and drug-polymer interaction of longer stented segments may lead to incomplete expansion, edge related disease or to a heterogeneous neointimal response. Another important clinical factor in ISR biology is diabetes. The current analysis did not demonstrate a statistically significant difference between prevalence of diabetes among ISR and non-ISR patients, but diabetes is a known factor that can lead to poor stent performance. A recent study from a large Swedish cohort showed that deterioration of glycaemic control was associated with increased coronary stent failure with most of the excess risk represented by ISR. [19,20] Hyperglycemia may play a role in the development of restenosis in both experimental and clinical studies through the induction of endothelial dysfunction, oxidative stress, inflammation, abnormal vascular smooth-muscle-cell activity, and impaired vascular healing. As such, the absence of a statistically significant association with diabetes in the current study should not be interpreted as an absence of a well established biological relevance and the rather limited number of ISR events may have precluded the identification of associations with individual clinical risk factors. The strength of this study was the angiographic evaluation at six months to establish the ISR based on a predetermined luminal narrowing criterion. Biochemical and procedural parameters were also incorporated in the study, which enabled the study of the UAR in the context of known clinical and angiographic characteristics. Baseline laboratory and procedural data was gathered in a standardised manner and consecutively collected, which also contributes to consistency in data collection. In addition, UAR can be used with the simultaneous consideration of two biologically relevant parameters, as opposed to serum uric acid alone. Therefore, the study contributes to the rapidly expanding body of literature on evaluating simple, routine accessible biomarkers in patients who undergo coronary intervention. There are a few limitations to note when drawing conclusions from the results. The study was conducted at one cardiac center and the findings might not be applicable to other groups and health care environments. Second, the sample size was relatively small and the number of ISR events was limited, which prevented us from testing several potential confounders in the same multivariable prediction model or from making a very sophisticated model. Third, renal function, metabolic status, diet, medications, hydration status, and other systemic conditions can impact UAR. However, there may be residual confounding as patients with severe renal impairment and several major inflammatory or hepatic disease were excluded. Fourth, there is an observational design and only associations can be detected and not causes. The present results do not indicate a direct connection between the higher UAR and the ISR. Last, specific mechanisms of ISR were not directly measured by intravascular ultrasound or optical coherence tomography, so the observed association could not be attributed to stent underexpansion, neointimal hyperplasia, neoatherosclerosis, stent fracture, or other mechanisms. Mechanistic characterisation of ISR has been highlighted in recent literature as the various types of stent failure could have varying biological and procedural underlying causes . [17,18] Interpretation of the clinical implications of these results should be made with caution. UAR is inexpensive, easily accessible from standard biochemical testing and does not require special equipment [14]. The link between increased UAR and ISR in larger prospective multicenter cohorts may be used in risk stratifying after DES implantation. The results of the present work, however, do not as yet define a clinically significant threshold for intervention nor do they provide evidence that modification of uric acid or albumin levels will lower ISR. Future clinical trials should thus assess the value of UAR prospectively in larger populations with multiple adjustment for clinical and procedural confounding factors and assess the incremental value of UAR over established ISR risk factors. The use of UAR together with intravascular imaging, inflammatory markers, renal function, glycaemic markers, lesion features and modern stent parameters could also contribute to the understanding of the biological mechanisms of the observed correlation.

CONCLUSION

The present study demonstrated that higher uric acid to albumin ratio was correlated with in-stent restenosis after DES implantation, which was confirmed by angiography. Patients with ISR had elevated serum uric acid and UAR levels compared to those without ISR, suggesting that UAR may be used as an readily available biomarker of adverse vascular response after PCI. UAR may provide risk assessment data regarding PCI beyond what is seen in the literature. Use of UAR as a standalone predictor should be confirmed in larger and multicenter prospective studies, though. Further research is required to assess whether the addition of UAR in addition to the well-established clinical, biochemical and procedural characteristics helps to better identify patients at higher risk of ISR.

REFERENCE

1. World Health Organization. Cardiovascular diseases (CVDs). Geneva: World Health Organization; 2023.
2. Alfonso F, Byrne RA, Rivero F, Kastrati A. Current treatment of in-stent restenosis. *Nat Rev Cardiol.* 2021;18(7):435–450.

3. Buccheri D, Piraino D, Andolina G, Cortese B. Understanding and managing in-stent restenosis: a review of clinical data, from pathogenesis to treatment. *J Am Heart Assoc.* 2020;9(6):e014809.
4. Otsuka F, Byrne RA, Yahagi K, Mori H, Ladich E, Fowler DR, et al. Neoatherosclerosis: overview of histopathologic findings and implications for intravascular imaging assessment. *Circ Res.* 2020;126(8):1130–1148.
5. Kang SH, Kang DY, Ahn JM, Kim YH, Park DW, Lee SW, et al. Clinical outcomes of in-stent restenosis after drug-eluting stent implantation. *EuroIntervention.* 2021;16(14):e1133–e1141.
6. Ndrepepa G. Uric acid and cardiovascular disease. *Clin Chim Acta.* 2021;515:150–163.
7. Arques S. Human serum albumin in cardiovascular diseases. *Heart Fail Rev.* 2022;27(4):1249–1260.
8. Liu W, Ding K, Bao J, Hu Y, Gui Y, Ye L, Wang L. Relationship between uric acid to albumin ratio and in-stent restenosis in patients with coronary artery disease undergoing drug-eluting stenting. *Coron Artery Dis.* 2023;34(8):589–594.
9. Yalcinkaya D, Kalayci S, Bilge M. Uric acid to albumin ratio is associated with coronary artery disease severity in stable coronary artery disease patients. *Coron Artery Dis.* 2024;35(2):123–129.
10. Zhang XJ, Gao X, Xu Y, Liu Y, Wang J, Chen Y. Uric acid-to-albumin ratio is associated with coronary slow flow in patients with chronic coronary syndrome. *BMC Cardiovasc Disord.* 2024;24:146.
11. Oflar E, Karabulut D, Yildiz C, Sinoplu HA, Ozcan E, Donmez E, et al. The uric acid/albumin ratio might be a better indicator for predicting repeat revascularization in young patients with acute coronary syndrome: beyond inflammatory biomarkers. *PLoS One.* 2024;19(8):e0306178. doi:10.1371/journal.pone.0306178.
12. Li S, Chen H, Zhou L, Cui H, Liang S, Li H. The uric acid to albumin ratio: a novel predictor of long-term cardiac mortality in patients with unstable angina pectoris after percutaneous coronary intervention. *Scand J Clin Lab Invest.* 2022;82(4):304–310.
13. Toprak K, Özen K, Memioğlu T, İnanır M, Kaplangöray M, Akyol S, et al. Comparison of the effect of uric acid/albumin ratio on coronary slow flow with other inflammation-based markers. *Biomark Med.* 2024;18(1):25–37.
14. Toprak K, Yılmaz R, Kaplangöray M, Memioğlu T, İnanır M, Akyol S, et al. Comparison of the effect of uric acid/albumin ratio on coronary collateral circulation with other inflammation-based markers in stable coronary artery disease patients. *Perfusion.* 2024;39(7):1440–1452.
15. Hiraga T, Saito Y, Mori N, Tateishi K, Kitahara H, Kobayashi Y. Impact of serum uric acid level on systemic endothelial dysfunction in patients with a broad spectrum of ischemic heart disease. *J Clin Med.* 2021;10(19):4530.
16. Clare J, Ganly J, Bursill CA, Sumer H, Kingshott P, de Haan JB. The mechanisms of restenosis and relevance to next generation stent design. *Biomolecules.* 2022;12(3):430.
17. Pelliccia F, Zimarino M, Niccoli G, Morrone D, De Luca G, Miraldi F, et al. In-stent restenosis after percutaneous coronary intervention: emerging knowledge on biological pathways. *Eur Heart J Open.* 2023;3(5):oead083.
18. Liu B, Li M, Liu J, Xie L, Li J, Liu Y, et al. Risk factors and incidence for in-stent restenosis with drug-eluting stent: a systematic review and meta-analysis. *Rev Cardiovasc Med.* 2024;25(12):458.
19. Santos-Pardo I, Andersson Franko M, Lagerqvist B, Ritsinger V, Eliasson B, Witt N, et al. Glycemic control and coronary stent failure in patients with type 2 diabetes mellitus. *J Am Coll Cardiol.* 2024;84(3):260–272.
20. Wang CS, Sun Y, Ren J. Uric acid to albumin ratio, a novel biomarker of cardiovascular disease. *Angiology.* 2024;75(4):394.