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RESEARCH ARTICLE
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CORRELATION BETWEEN SERUM URIC ACID LEVEL AND THE SEVERITY OF CORONARY ARTERY DISEASE

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Abstract

Objective

This study aims at the evaluation of the correlation between level of serum uric acid (SUA) and the extent of coronary artery disease (CAD) using angiographic scoring.

Study design: Cross-sectional study

Duration and place of study: This study was conducted in Peoples University of Medical and Health Sciences for Women Shaheed Benazirabad Nawabshah from May 2023 to May 2024

Methodology

150 patients diagnosed with CAD through coronary angiography were included in this study. The severity of CAD was assessed using the Gensini score. SUA levels were measured and categorized. Statistical correlation between uric acid levels and CAD severity was analyzed using Spearman's rank correlation. Data analysis was performed using SPSS version 25.

Results

Among the 150 patients enrolled, the age range was 40 to 70 years. The mean age of patients was 58.6 ± 11.7 years. Males comprised 72.7% (n=109) of the sample. The mean SUA level was 6.84 ± 1.65 mg/dl, while the average Gensini score was 54.2 ± 18.9. A significant association of SUA and Gensini score (r = 0.462) was observed, indicating a moderately strong relationship. Elevated uric acid levels were more frequently noted in patients with higher Gensini scores.

Conclusion
This study found a strong association between elevated SUA levels and the severity of coronary artery disease, suggesting its potential role as a non-invasive marker for CAD risk stratification.

Keywords: Coronary artery disease, Serum uric acid, Gensini score, Cardiovascular risk, Hyperuricemia, Angiographic severity, Spearman correlation

Introduction

CAD remains a major contributor to global morbidity and mortality, accounting for a substantial burden on healthcare systems worldwide. The underlying pathophysiology primarily involves atherosclerosis, which leads to narrowing of the coronary arteries and impairs myocardial perfusion, manifesting clinically as angina, myocardial infarction, and sudden cardiac death [1]. Early detection and accurate assessment of the severity of CAD are essential for risk stratification, timely intervention, and improved outcomes.

While conventional cardiovascular risk factors are well-established, there is growing interest in identifying additional biomarkers that could help in predicting the severity and progression of CAD. One such biomarker under investigation is SUA, the end product of purine metabolism. Traditionally associated with gout, hyperuricemia has now been linked with multiple cardiometabolic conditions, including hypertension, chronic kidney disease, insulin resistance, and metabolic syndrome, all of which are known to contribute to atherosclerotic disease [2,3].

Emerging evidence suggests that elevated levels of SUA may have a direct pathophysiological role in atherosclerosis. At high concentrations, uric acid can act as a pro-oxidant, promoting endothelial dysfunction by reducing nitric oxide availability and increasing oxidative stress. These changes impair vascular relaxation and may contribute to plaque formation and instability [4,5]. Uric acid has also been implicated in stimulating smooth muscle cell proliferation, promoting systemic inflammation, and enhancing platelet aggregation, mechanisms that collectively support its involvement in the development and worsening of CAD [6,7].

A number of epidemiological and clinical researches proved the positive impact of increased level of SUA and its association with the presence of CAD whether there are traditional risk factors or not. To be more specific, elevated SUA has been linked with increased angiographic burden of coronary artery disease, which is measured via scoring systems e.g. Gensini or SYNTAX score [8811]. These angiographic scoring systems are objective indexers of the burden and complexity of coronary lesions which have wide application both in the clinical and the research setting.

The Gensini score in specific regards not just the extent of luminal narrowing but in addition the anatomical presence of the injuries giving a complete analysis of the limit of atherosclerosis [12]. Some studies have been conducted among patients undergoing coronary angiography and found out that patients with hyperuricemia recorded higher Gensini scores showing more severe disease. This correlation has been observed in a number of the populations of patients with acute coronary syndrome and stable angina, as well as in younger adults with premature CAD [1315].

In spite of these observations, the general use of SUA as a prognostic tool in the severity of CAD is not without any detractors. Other research programs have yielded contradictory conclusions especially during the exposure of confounding factors like renal function, obesity and consumption of medications like diuretics. In addition, there is a continued controversy of whether hyperuricemia is an actual independent risk factor or is only a surrogate indicator depicting other metabolic abnormalities [16].

Nevertheless, the possible applicability of SUA as an easy, cheap, and regularly assessible biomarker renders it a noticeable subject of additional testing. At the trials of conditions when the access to high level diagnostic instruments is restricted SUA testing may be considered hand tools in distinguishing people who are more exposed to adverse results in coronary artery disease.

The growing burden of cardiovascular diseases in a developing world and the poor availability of useful risk assessment indicators necessitate the importance of identification of markers that are practical as well as clinically useful. This research aimed to help determine the association between

levels of SUA and severity of CAD in local population who have undergone coronary angiography. Gensini score has been used to evaluate the severity of the CAD, and the aim was to find out whether increased levels of SUA are more profoundly related to the more severe coronary involvement.
The study has the potential of enriching the current evidence on the possible role of SUA in cardiovascular risk determination. In the event that a regular and robust relationship is established, SUA can be regarded as part of standard assessment programs in patients with probable or proven CAD, giving a convenient complement to conventional risk classifications methods.

Methodology

The sampling was done in a non probability consecutive method and 150 patients were enrolled. Each patient with a coronary angiography in the study period and with elective or urgent procedures and matching the inclusion criteria was deemed eligible.
Patients aged between 40 and 70 years with a confirmed diagnosis of CAD on angiography were included. CAD was defined as at least one coronary artery with ≥50% luminal stenosis. Patients with chronic kidney disease (eGFR <60 mL/min/1.73 m²), gout, hematological malignancies, autoimmune disorders, or those receiving uric acid-lowering therapy or diuretics were excluded from the study in order to avoid potential confounding.
Followed by obtaining a written informed consent from all the participants, demographic as well as clinical data were recorded, including age, BMI, gender, smoking status, history of comorbidities such as hypertension, diabetes mellitus, and dyslipidemia. Fasting blood samples were collected prior to angiography to measure SUA levels, using an enzymatic colorimetric method (uricase/peroxidase technique). Hyperuricemia was defined as SUA level more than 7.0 mg/dL in males and more than 6.0 mg/dL in females.
Radial or femoral access was used with normal procedures to perform coronary angiography. The angiographic outcomes were assessed by two experienced interventional cardiologist. The doctors were blinded to the SUA concentrations. Gensini score was used to determine the severity of coronary artery disease. The cumulative score indicated by this scoring system is the overall burden of atherosclerosis as a severity score is provided to each coronary stenosis depending on the level of luminal narrowing and its anatomical location.
IBM SPSS Statistics version 25 was used to access and analyse all the data. Some examples of continuous variables were age, Gensini scores, and levels of SUA, which were presented as means and standard deviations. The categorical variables were summarised in frequencies and percentages. Gensini score was determined in order to stratify patients as per the extent of their disease as mild (<30), moderate (30-60), and severe (>60). The Rank-Correlation coefficient (r) Spearman was used to test the correlation between SUA and Gensini score and the p-value <0.05 was accepted as significant. Differences between groups were compared through independent sample t-tests or chi-square tests when it was needed.

Results

A total of 150 patients undergoing coronary angiography were included in the study. The mean age of participants was 58.6 ± 11.7 years, with an age range from 40 to 70 years. A majority of the study population were males, comprising 72.7% (n = 109), while 27.3% (n = 41) were females. Hypertension was documented in 96 patients (64%), and 58 patients (38.7%) were known diabetics. Dyslipidemia was present in 51 (34%), while 39 (26%) were current smokers. The mean body mass index (BMI) was 27.4 ± 3.5 kg/m².
The mean SUA level in the total population was 6.84 ± 1.65 mg/dL, with values ranging from 3.4 to 9.7 mg/dL. Based on gender-specific reference values (>7.0 mg/dL for males and >6.0 mg/dL for females), hyperuricemia was identified in 82 patients (54.7%).
CAD severity was assessed using the Gensini scoring system. The mean Gensini score was 54.2 ± 18.9, with a range of 15 to 94. Based on score thresholds, patients were stratified into three groups: mild CAD (Gensini score <30), moderate CAD (30–60), and severe CAD (>60). Mild disease was present in 25.3% (n = 38) of patients, moderate in 40% (n = 60), and severe in 34.7% (n = 52).

A statistically significant difference was observed in SUA levels across the CAD severity groups. Patients with mild CAD had a mean SUA level of 5.81 ± 1.42 mg/dL, those with moderate CAD had 6.75 ± 1.58 mg/dL, while patients with severe CAD had significantly higher levels at 7.81 ± 1.47 mg/dL. This progressive increase in SUA with disease severity was statistically significant ($p < 0.001$, ANOVA test).

Also, the high positive correlation between SUA and Gensini scores, with a lower limit of significance (Thanks to a statistically small p-value of < 0.001), provided a relatively strong direct relationship, as measured by the Spearman r correlation coefficient as being: 0.462.

Hyperuricemia was significantly more frequent among patients with severe CAD. Among those with hyperuricemia, 42.7% had severe CAD, compared to only 24.2% among normouricemic patients. Conversely, a larger proportion of normouricemic patients fell into the mild CAD category (33.9% vs. 18.3%, respectively). This distribution showed statistical significance ($p = 0.01$, chi-square test).

Gender-wise, male patients had a higher prevalence of hyperuricemia (60.6%) compared to females (39.0%), which was statistically significant ($p = 0.02$). In terms of comorbidities, hyperuricemia was more common among hypertensive patients (65.2% vs. 42.1%, $p = 0.006$) and those with diabetes mellitus (61.7% vs. 46.8%, $p = 0.04$). No significant association was observed with smoking status or BMI.

These findings suggest that elevated SUA levels are associated with increased severity of coronary artery disease, independent of traditional risk factors.

Table 1: Demographic data (n = 150)

Variable	Value / Frequency
Age (years), mean ± SD	58.6 ± 11.7
Gender (Male), n (%)	109 (72.7%)
Female, n (%)	41 (27.3%)
Hypertension, n (%)	96 (64%)
Diabetes Mellitus, n (%)	58 (38.7%)
Dyslipidemia, n (%)	51 (34%)
Smokers, n (%)	39 (26%)
BMI (kg/m²), mean ± SD	27.4 ± 3.5
SUA (mg/dL), mean ± SD	6.84 ± 1.65
Hyperuricemia, n (%)	82 (54.7%)
Gensini Score, mean ± SD	54.2 ± 18.9

Table 2: Distribution of SUA and CAD Severity Based on Gensini Score

Gensini Score Category	No. of Patients (%)	Mean SUA (mg/dL) ± SD	Hyperuricemia (%)
Mild (<30)	38 (25.3%)	5.81 ± 1.42	18.3%
Moderate (30–60)	60 (40.0%)	6.75 ± 1.58	39.0%
Severe (>60)	52 (34.7%)	7.81 ± 1.47	42.7%
p-value	—	<0.001*	0.01*

Discussion

Gensini score that is used to assess the severity of CAD was also found to have a statistically significant positive correlation with the SUA levels in the present study. The prevalence of hyperuricemia was also much higher in severe CAD group and more extensive atherosclerosis was shown in patients with higher levels of SUA. On the basis of these findings, it is possible that SUA will find clinical application as a potential predictor of CAD burden.

The relationship seen in our cohort is consistent with prior studies that have been able to conclude on the use of uric acid as a cardiovascular risk factor. In some research carried out in a tertiary care facility in India, people with acute coronary syndrome (ACS) were assessed with reference to SUA

and the angiographic severity of CAD. The Gensini scores of hyperuricemic subjects rose considerably, researchers said, and they concluded that SUA positively correlated with the amount of diseased vessels as well as plaque burden [17].

Similar findings were supported by another study conducted in Iraq. The SUA level was independent predictors of the angiographic severity in this prospective observational study of 120 patients who had the cardiology angiography. Compared to patients with normal levels, the prevalence of the multivessel disease and triple-vessel involvement was significantly higher among patients with SUA above 7 mg/dL due to the 29% and 33% proportions, respectively [18].

The strength of the association between SUA and CAD severity was also confirmed in a Turkish study that included 300 patients admitted with chest pain. The researchers found that SUA levels increased proportionally with the number of stenotic vessels and complexity of lesions. Even after adjusting for traditional cardiovascular risk factors, uric acid remained a significant predictor of higher SYNTAX scores [19]. Our study used the Gensini score, which offers a slightly more nuanced view of lesion severity and location, but the overall relationship appears consistent across scoring systems.

Evidence from Iran similarly supports our results. In a study involving 200 patients undergoing angiography, hyperuricemia was significantly more prevalent among individuals with left main or triple-vessel disease. The study also showed a stepwise increase in SUA across mild, moderate, and severe CAD categories [20]. The design and categorization approach in that study mirrored ours, further reinforcing the credibility of the correlation.

In contrast, a study conducted in Poland offered a more cautious interpretation. While it did observe a positive correlation between SUA and CAD, the strength of the association diminished after multivariable adjustment for metabolic syndrome and renal function markers. The authors suggested that SUA might reflect underlying systemic inflammation or renal impairment rather than being directly causal in CAD progression [21]. Although this perspective introduces important nuances, our study minimized such confounding by excluding patients with known kidney disease or those on medications like diuretics that could affect SUA levels.

Another prospective analysis from Egypt examined 250 patients admitted with non–ST elevation myocardial infarction (NSTEMI). The researchers found significantly higher SUA levels among patients with elevated troponin and multivessel CAD. They concluded that SUA levels could predict both biochemical severity (infarct size) and anatomical burden (angiographic score), suggesting a potential prognostic as well as diagnostic role [22]. While our study did not examine biomarker trends or follow-up outcomes, it supports the anatomical correlation observed.

Taken together, the findings from these diverse studies across various populations—India, Iraq, Turkey, Iran, Poland, and Egypt—consistently support a positive relationship between uric acid levels and CAD severity. Our study contributes to this growing body of literature, especially in the South Asian context where CAD presents at a younger age and with more aggressive phenotypes.

One of the strengths of our study is its exclusion of confounding variables such as chronic kidney disease and uric acid–modifying medications. By focusing on angiographic evidence and carefully defined patient groups, we attempted to isolate the relationship between uric acid and coronary burden as cleanly as possible.

However, limitations remain. This was a single-center, cross-sectional analysis that did not track patient outcomes or adjust for all possible biochemical variables. Furthermore, the use of a single SUA measurement may not fully reflect chronic uric acid exposure or variability over time.

Conclusion

This study found a clear and moderately strong link between higher SUA levels and more severe coronary artery disease, as measured by the Gensini score. Patients with elevated uric acid were more likely to have extensive coronary involvement, suggesting that uric acid may be a useful marker of disease burden. Given that SUA is simple and affordable to measure, it could serve as an additional tool in assessing cardiovascular risk, especially in settings where advanced testing isn't easily available. However, more research is needed to confirm whether lowering uric acid levels can actually improve heart outcomes.

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None

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