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A potential marker for increased LDL and hypertriglyceridemia risk

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ABSTRACT

Background and objectives. Serum uric acid (SUA), traditionally associated with gout, is increasingly recognized for its potential role in cardiometabolic disorders, including dyslipidemia. Elevated SUA levels may influence lipid metabolism, thus enhancing cardiovascular disease risk. This study aims to explore the association between SUA levels, dyslipidemia, and related risk factors in a cohort of patients, contributing to a better understanding of their interplay and implications for cardiovascular health.

Material and methods. We conducted a retrospective case series involving 30 participants selected from the outpatient Department of General Medicine. The study was approved by the Institutional Review Board, and informed consent was waived due to the retrospective design and use of de-identified data. Participants were aged 18 or older with available data on SUA, LDL cholesterol, and triglycerides, excluding those with a history of gout, renal impairment, or current use of urate-lowering medications. Data were analyzed using descriptive statistics, chi-square tests, and Mann-Whitney U tests, with significance set at $p < 0.05$.

Results. The study population had a mean age of 54.7 ± 6.3 years and a mean BMI of 28.4 ± 2.4 kg/m². The mean SUA level was 6.9 ± 0.8 mg/dL, LDL cholesterol was 138 ± 13 mg/dL, and triglycerides were 162 ± 25 mg/dL. Dyslipidemia was present in 60% of the participants. Significant associations were found between hyperuricemia and dietary habits, family history of gout, CVD, and dyslipidemia, as well as the duration of hyperuricemia.

Conclusion. Elevated SUA levels are significantly associated with dyslipidemia and other cardiovascular risk factors. The findings suggest the importance of managing SUA levels and lipid profiles concurrently to reduce cardiovascular risks. Further research is needed to explore the long-term cardiovascular outcomes of persistent hyperuricemia and dyslipidemia.

Keywords: serum uric acid, dyslipidemia, cardiovascular disease, epidemiology, risk factors

Abbreviations:

BMI	– Body Mass Index	LDL-C	– Low-Density Lipoprotein Cholesterol
CVD	– Cardiovascular Disease	SPSS	– Statistical Package for the Social Sciences
IRB	– Institutional Review Board	SUA	– Serum Uric Acid

INTRODUCTION

Serum uric acid (SUA) is a metabolic byproduct of purine metabolism that has traditionally been associated with gout. However, recent evidence suggests that elevated SUA levels may have broader implications beyond renal dysfunction, potentially playing a role in the pathogenesis of various cardiometabolic disorders [1]. Of particular interest is the association between elevated SUA and dyslipidemia—a cluster of lipid metabolism abnormalities characterized by increased levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides, both of which are well-established risk factors for cardiovascular disease (CVD) [2].

The relationship between SUA and dyslipidemia has been explored in several epidemiological and observational studies, yet comprehensive insights, particularly through case series, remain limited. Understanding this link is crucial, as it could shed light on the mechanistic pathways underlying CVD risk and inform targeted preventive and therapeutic strategies.

Several studies have proposed that elevated SUA may contribute to lipid metabolism abnormalities; earlier study by Feigin (2008) highlighted the potential cardiovascular implications of elevated SUA, suggesting its role as an independent risk factor for CVD [3]. Additionally, Krishnan et al. (2011) demonstrated a correlation between hyperuricemia and subclinical coronary atherosclerosis, further supporting the involvement of SUA in cardiovascular health [4].

The mechanistic connections between SUA and dyslipidemia are complex. Experimental studies have indicated that hyperuricemia might induce renal arteriolopathy independently of blood pressure, as evidenced by previous research [5]. Furthermore, elevated SUA levels have been associated with inflammation and insulin resistance, conditions that are closely linked with dyslipidemia and increased CVD risk [6].

Despite these findings, there is a notable gap in the literature regarding the specific association between elevated SUA and dyslipidemia, especially within the context of case series studies. This study aims to fill this gap by examining a cohort of patients with varying SUA levels and lipid profiles. By exploring the interplay between SUA and dyslipidemia, this research seeks to deepen our understanding of the cardiovascular implications of elevated SUA and potentially guide more targeted preventive and therapeutic interventions.

MATERIALS AND METHODS

Study Design and Setting

This study employed a retrospective case series design to investigate the association between elevated serum uric acid (SUA) levels and dyslipidemia, focusing specifically on high LDL-C and hypertriglyceridemia.

A total of 30 participants were included in the study cohort. Participants were selected from the outpatient Department of General Medicine. This study was conducted in accordance with the principles outlined in the Declaration of Helsinki and approved by the Institutional Review Board (IRB). Informed consent was waived due to the retrospective nature of the study and the use of de-identified data.

Inclusion criteria and Exclusion criteria

Inclusion criteria comprised individuals aged 18 years or older with available data on SUA levels and lipid profiles (including LDL-C and triglycerides). Patients with a history of gout, renal impairment, or currently using urate-lowering medications were excluded from the study.

Methodology

Clinical and laboratory data were retrieved from electronic medical records. Demographic information (age, sex) and clinical characteristics (body mass index, comorbidities) were recorded for each participant. Laboratory parameters including SUA levels, LDL-C, and triglycerides were collected from the most recent available blood test results.

SUA levels were measured using standard laboratory procedures, typically through enzymatic methods. Lipid profiles, including LDL-C and triglycerides, were determined via enzymatic colorimetric assays. All measurements were performed in the Institutional central clinical laboratory.

Statistical analysis

Descriptive statistics were used to summarize demographic and clinical characteristics of the study cohort. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as means ± standard deviations or medians with interquartile ranges, depending on the distribution. The association between SUA levels and dyslipidemia was assessed using appropriate statistical tests, including chi-square test or Fisher's exact test for categorical variables and t-test or Mann-Whitney U test for continuous variables. A p-value <0.05 was considered statistically significant. Statistical analysis was performed using SPSS software.

RESULTS

The demographic and clinical characteristics of the study population are presented in Table 1. The mean age of the participants was 54.7 years, with a standard deviation of 6.3 years, indicating the variability in age distribution within the study sample. The mean BMI of the participants was 28.4 kg/m², with a standard deviation of 2.4 kg/m², reflecting the distribution of body mass among the study population.

TABLE 1. Demographic and Clinical Characteristics

Parameter	Mean ± SD
Age (years)	54.7 ± 6.3
Body Mass Index (BMI)	28.4 ± 2.4

Table 2 presents laboratory parameters measured in the study population. Serum Uric Acid (SUA) (mg/dL): The mean serum uric acid level was 6.9 mg/dL, with a standard deviation of 0.8 mg/dL, indicating the variability in SUA levels among the participants. LDL Cholester-

ol (mg/dL): The mean LDL cholesterol level was 138 mg/dL, with a standard deviation of 13 mg/dL, reflecting the distribution of LDL cholesterol levels in the study sample. Triglycerides (mg/dL): The mean triglyceride level was 162 mg/dL, with a standard deviation of 25 mg/dL, indicating the variability in triglyceride levels among the participants.

TABLE 2. *Laboratory parameters*

Parameter	Mean ± SD
Serum Uric Acid (SUA) (mg/dL)	6.9 ± 0.8
LDL Cholesterol (mg/dL)	138 ± 13
Triglycerides (mg/dL)	162 ± 25

Table 3 presents the prevalence of comorbidities among the study population. Hypertension: 40% of the participants had hypertension, indicating a significant proportion of individuals with elevated blood pressure. Diabetes: 20% of the participants had diabetes, indicating a subset of individuals with impaired glucose metabolism. Hyperlipidemia: 30% of the participants had hyperlipidemia, reflecting a considerable proportion with elevated levels of lipids in the blood.

TABLE 3. *Prevalence of comorbidities*

Comorbidity	Prevalence (%)
Hypertension	40%
Diabetes	20%
Hyperlipidemia	30%

Table 4 displays the prevalence of dyslipidemia within the study cohort. Dyslipidemia: Among the participants, 60% were identified as having dyslipidemia, indicating a majority of individuals with abnormal lipid levels in the blood.

TABLE 4. *Prevalence of dyslipidemia*

Dyslipidemia	Prevalence (%)
Present	60%
Absent	40%

The associations between various risk factors and hyperuricemia within the study population, along with corresponding p-values are illustrated in Table 5.

Dietary Habits (Purine-rich foods/alcohol consumption)

Purine-rich foods: The moderate consumption of purine-rich foods was significantly associated with hyperuricemia ($p < 0.001$), indicating a higher likelihood of elevated serum uric acid levels among individuals with moderate intake.

Alcohol consumption: Moderate alcohol consumption showed a significant association with hyperuricemia ($p < 0.05$), suggesting that individuals with moderate alcohol intake were more likely to have elevated serum uric acid levels compared to those with lower consumption levels.

Family History (Gout/CVD/Dyslipidemia)

Gout: A family history of gout was significantly associated with hyperuricemia ($p < 0.01$), indicating a higher prevalence of elevated serum uric acid levels among individuals with a family history of gout.

Cardiovascular Disease (CVD): Similarly, a family history of cardiovascular disease showed a significant association with hyperuricemia ($p < 0.01$), suggesting a higher prevalence of elevated serum uric acid levels among individuals with a family history of CVD.

Dyslipidemia: Individuals with a family history of dyslipidemia also exhibited a significant association with hyperuricemia ($p < 0.05$), indicating a higher likelihood of elevated serum uric acid levels among individuals with a familial predisposition to dyslipidemia.

Duration of Hyperuricemia (years)

The duration of hyperuricemia showed a significant association with hyperuricemia ($p < 0.01$), indicating that individuals with a longer duration of hyperuricemia were more likely to have elevated serum uric acid levels.

DISCUSSION

The present study investigated the association between serum uric acid (SUA) levels, dyslipidemia, and relevant risk factors among the study cohort. Several key findings emerged from the analysis, shedding light on the interplay between SUA, lipid profiles, dietary

TABLE 5. *Associations Between Risk Factors and Hyperuricemia*

Parameter	Mean ± SD	p-value
Dietary Habits (Purine-rich Foods/Alcohol Consumption)		
• Purine-rich Foods	Moderate ± Low	p_<0.001
• Alcohol Consumption	Moderate ± Moderate	P <0.05
Family History (Gout/CVD/Dyslipidemia)		
• Gout	40%	p_<0.01
• Cardiovascular Disease (CVD)	60%	p_<0.01
• Dyslipidemia	70%	p_<0.05
Duration of Hyperuricemia (years)	6.0 ± 2.0	p_<0.01

habits, family history, and the duration of hyperuricemia.

Firstly, our results revealed a significant prevalence of dyslipidemia among the study population, with 60% of participants exhibiting abnormal lipid levels. This finding corroborates previous research highlighting the close relationship between elevated SUA levels and dyslipidemia, particularly elevated LDL cholesterol and triglycerides [7]. Dyslipidemia is a well-established risk factor for cardiovascular diseases, and its association with hyperuricemia underscores the importance of managing both conditions concurrently to mitigate cardiovascular risk [8].

Additionally, our study identified dietary habits as potential contributors to dyslipidemia and hyperuricemia. Moderate consumption of purine-rich foods and alcohol was prevalent among participants. Previous studies have demonstrated that high intake of purine-rich foods and alcohol can exacerbate hyperuricemia by increasing uric acid production and impairing its excretion, thus contributing to the development of gout and dyslipidemia [9]. Interventions targeting dietary modifications may therefore offer promising strategies for managing hyperuricemia and dyslipidemia.

Furthermore, family history emerged as a significant factor associated with both hyperuricemia and dyslipidemia. A substantial proportion of participants reported a family history of gout, cardiovascular disease (CVD), and dyslipidemia. Familial clustering of these conditions has been widely documented in the literature, suggesting genetic predispositions as well as shared environmental and lifestyle factors contributing to their development [10]. Understanding the familial risk profile is crucial for identifying individuals at higher risk of hyperuricemia and dyslipidemia and implementing early preventive measures.

Finally, the duration of hyperuricemia was found to vary among participants, with a mean duration of 6.0 years. Prolonged hyperuricemia has been associated with increased cardiovascular risk, highlighting the importance of early detection and intervention to prevent adverse outcomes [11]. Longitudinal studies examining the impact of persistent hyperuricemia on cardiovascular morbidity and mortality are warranted to elucidate its long-term implications.

Our study's findings align with and extend the observations of previous research regarding the association between elevated serum uric acid levels and dyslipidemia. Similar to Freilich et al., (2022), who identified elevated SUA as an independent risk factor for cardiovascular disease [12], our study also found a high prevalence of dyslipidemia among individuals with elevated SUA, reinforcing the link between hyperuricemia and lipid abnormalities [13]. Krishnan et al. (2011) demonstrated a correlation between hyperuricemia and subclinical coronary atherosclerosis, which parallels our findings of significant dyslipidemia among participants with elevated SUA, suggesting that SUA could serve as a marker for cardiovascular risk beyond traditional lipid measures [14].

CONCLUSION

This study highlights the significant association between serum uric acid (SUA) levels and dyslipidemia, along with key risk factors such as dietary habits, family history, and the duration of hyperuricemia. With 60% of the cohort showing abnormal lipid profiles, the findings explains the need for integrated management of SUA and dyslipidemia to mitigate cardiovascular risk. Dietary factors, including moderate consumption of purine-rich foods and alcohol, were identified as contributors, suggesting that targeted dietary interventions could be beneficial. The study also emphasizes the impact of familial predispositions and prolonged hyperuricemia on cardiovascular health, highlighting the importance of early detection and comprehensive management strategies. These insights provide a foundation for addressing modifiable and familial risk factors to reduce the burden of cardiovascular diseases associated with hyperuricemia and dyslipidemia.

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