Original Article

Serum Uric Acid–To–Creatinine Ratio as an Indicator of Metabolic Alterations: A Retrospective Biochemistry Study

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Abstract

Background: Serum uric acid (SUA) is associated with components of metabolic dysfunction; however, interpretation is influenced by renal function and muscle mass. The serum uric acid–to–creatinine ratio (UCR) has been proposed as a normalized index that may better reflect metabolic alterations. **Material and Methods:** A retrospective analysis was conducted on 600 adult laboratory records (>18 years) with available measurements of SUA, serum creatinine, fasting plasma glucose, and lipid profile. UCR was calculated as SUA/creatinine (mg/dL). Descriptive statistics were expressed as mean ± SD. Associations between UCR and metabolic parameters were assessed using Pearson's correlation, and gender-wise differences were evaluated using independent t-tests. **Results:** The study included 300 males and 300 females (mean age 45 ± 12 years). Although males had significantly higher SUA and creatinine levels than females ($p < 0.001$), UCR values were comparable between genders (6.15 ± 1.28 vs. 6.12 ± 1.25; $p = 0.83$). UCR showed weak correlations with fasting glucose ($r = −0.075$), triglycerides ($r = 0.012$), and HDL cholesterol ($r = −0.011$). No significant age-related trends were observed. **Conclusion:** In this cohort, UCR did not demonstrate strong associations with metabolic parameters, though it effectively normalized gender-related differences in SUA. Prospective studies in high-risk populations are required to establish its clinical utility.

Keywords: Uric acid–creatinine ratio; metabolic alterations; glucose; lipid profile; retrospective study; biomarkers.

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Introduction

Hyperuricemia has been consistently associated with a broad spectrum of metabolic abnormalities, including insulin resistance, type 2 diabetes mellitus, hypertension, and dyslipidemia, and is increasingly recognized as an integral component of cardiometabolic risk clustering.[1-4] Beyond its traditional role as an end product of purine metabolism, serum uric acid (SUA) has been implicated in the pathogenesis of metabolic syndrome through its pro-oxidant effects, promotion of endothelial dysfunction, and activation of low-grade inflammatory pathways. Elevated uric acid levels may contribute to impaired nitric oxide bioavailability, adipose tissue inflammation, and altered glucose and lipid metabolism, thereby reinforcing metabolic derangements.[5,6] However, interpretation of SUA levels in clinical and epidemiological studies is complicated by several confounding factors. Statistical-level renal uric acid renal elimination, muscle mass, dietary intake, age and sex can significantly have an effect on serum uric acid and would confound its relationship with metabolic risk, which reduces its specificity as a metabolic biomarker. These shortcomings have led to a search into other indices that can potentially improve on endogenous uric acid production and reducing the effects of renal clearance.

Serum creatinine, which is regularly assessed in clinical practice, is a surrogate variable of renal performance and skeletal muscle mass. Most recently, uric acid to creatinine ratio (UCR) has been proposed as a normalised value that can correct uric acid levels to adjust uric acid metabolism to insulin resistance, adiposity and dyslipidemia, and not simply to renal dysfunction itself.[10]

Recent research studies have found that UCR is significantly associated with metabolic syndrome components, non-alcoholic fatty liver disease, insulin resistance, and poor lipid profiles, but inconsistent results have been described across populations, age groups, and studies, and as such, there is a need to further test its usefulness in various clinical contexts. Information based on regular lab cohorts, especially among the Indian population, is scanty.

This is the backdrop upon which the current retrospective biochemistry study was conducted to determine the relationship between UCR and fasting blood glucose and lipid profiles parameters in relation to laboratory data of these parameters that were available on a routine basis. Also, the research sought to determine age and gender-related changes in UCR, so as to

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establish the possible use of the latter as an inexpensive, easy to use biomarker of detecting changes in metabolism in adult populations.

Materials and Methods

Study Design: The study is a retrospective observational study done in the archived data of Sree Mookambika Institute of Medical Sciences clinical biochemistry laboratory, which is a tertiary care hospital in South India. Laboratory records of adult individuals (>18 years) with complete data on SUA, serum creatinine, fasting plasma glucose, and lipid profile were included. Ethical clearance for the study was obtained from the Institutional Ethics Committee of SMIMS

Inclusion and Exclusion Criteria

The study employed laboratory data of the adult population 18 years or older who had had their serum uric acid, serum creatinine, fasting blood glucose, and lipid profile simultaneously estimated on a single visit. Records that had all the necessary biochemical parameters and are complete were only analyzed. The records were not included in the data that has incomplete biochemical parameters, known renal failure (creatinine >2.0 mg/dL), and extreme outliers that are indicative of an analytical error.

Data collection and variables

Laboratory registers were used to obtain demographic information such as gender and age. Some of the biochemical variables studied were serum uric acid, serum creatinine, fasting blood glucose, total cholesterol, triglycerides, and high-density lipoprotein (HDL) cholesterol where necessary as well as calculated low-density lipoprotein (LDL) cholesterol. Each of the participants was calculated as being this ratio by dividing serum uric acid (mg/dL) by serum creatinine (mg/dL). They were randomly stratified into three age groups (18-40 years, 41-60 years and age 61 and above) in order to assess age differences.

Venous blood samples were collected following an overnight fast of 8–12 hours under standard aseptic precautions. Serum uric acid and creatinine were measured using enzymatic colorimetric methods on an automated clinical chemistry analyzer following manufacturer-recommended protocols. Fasting blood glucose was estimated using the glucose oxidase–peroxidase method. Lipid profile parameters, including total cholesterol, triglycerides, and HDL cholesterol, were analyzed using enzymatic methods, while LDL cholesterol was calculated using the Friedewald formula when triglyceride levels were <400 mg/dL. UCR was calculated as:

UCR = Serum uric acid (mg/dL) / Serum creatinine (mg/dL)

Internal quality control procedures and external quality assurance programs were routinely followed to ensure analytical accuracy and reliability.

Statistical Analysis: Data were analyzed using Python (SciPy library). Continuous variables were expressed as mean ± standard deviation. Pearson's correlation coefficient was used to assess associations between UCR and metabolic parameters. Independent t-tests were applied to compare gender-wise differences. A p-value <0.05 was considered statistically significant.

Results

A total of 600 adult laboratory records were analyzed, comprising 300 males and 300 females with a mean age of 45 ± 12 years. There was no statistically significant difference in age between males and females (p = 0.62).

Males exhibited significantly higher mean serum uric acid and creatinine levels compared to females (p < 0.001 for both). However, despite these differences, the uric acid–to–creatinine ratio (UCR) did not differ significantly between genders (6.15 ± 1.28 vs. 6.12 ± 1.25; p = 0.83). Fasting glucose and lipid parameters were also comparable between males and females. The gender-wise distribution of biochemical parameters is summarized in [Table 1].

Table 1: Gender-wise distribution of biochemical parameters

Parameter	Males (n = 300)	Females (n = 300)	p-value
Age (years)	45.2 ± 12.1	44.8 ± 11.9	0.62
SUA (mg/dL)	6.0 ± 1.5	4.8 ± 1.2	<0.001*
Creatinine (mg/dL)	1.0 ± 0.2	0.8 ± 0.15	<0.001*
UCR	6.15 ± 1.28	6.12 ± 1.25	0.83
Glucose (mg/dL)	112 ± 35	111 ± 34	0.78
Triglycerides (mg/dL)	150 ± 80	150 ± 80	0.98
HDL (mg/dL)	50 ± 15	50 ± 15	0.95

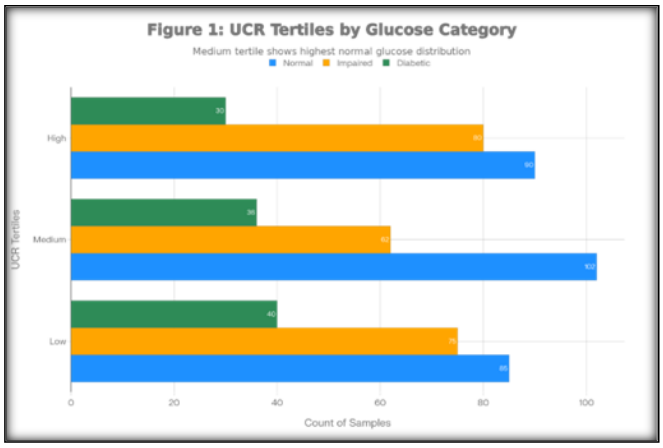
*Statistically Significant

Pearson correlation analysis demonstrated weak and statistically non-significant associations between UCR and fasting plasma glucose (r = −0.075, p = 0.068), triglycerides (r = 0.012, p = 0.766), HDL cholesterol (r = −0.011, p = 0.784), LDL cholesterol (r = 0.005, p = 0.902), and total cholesterol (r = 0.008, p = 0.845). Correlation coefficients and corresponding p-values are presented in [Table 2].

Table 2: Pearson Correlations

Parameter	r with UCR	p-value
Glucose	-0.075	0.068
TG	0.012	0.766
HDL	-0.011	0.784
LDL	0.005	0.902
Total Cholesterol	0.008	0.845

When UCR was stratified into tertiles, the distribution of normal, impaired, and diabetic glucose categories appeared relatively uniform across low, medium, and high UCR groups. This even distribution suggests the absence of a strong association between UCR levels and glycemic status in the study population. The distribution of glucose categories across UCR tertiles is illustrated in [Figure 1].



Discussion

The present retrospective study evaluated the utility of the serum uric acid–to–creatinine ratio (UCR) as a marker of metabolic alterations in an adult laboratory-based population. Although males in the current cohort exhibited significantly higher serum uric acid and creatinine levels compared with females, UCR values were comparable across genders. This observation supports the premise that UCR effectively adjusts serum uric acid concentrations for variations in renal function and muscle mass, thereby minimizing sex-related bias inherent to serum uric acid alone. Similar gender-independent behavior of UCR has been reported by Sun et al,[10] and Chen et al,[12] who demonstrated that UCR provides a more stable metric than absolute uric acid levels when comparing metabolic risk across sexes.

In the present study, UCR demonstrated weak and statistically non-significant correlations with fasting plasma glucose and lipid parameters, including triglycerides, total cholesterol, LDL cholesterol, and HDL cholesterol. Comparable modest associations between uric acid–derived indices and glucose metabolism have been reported in population-based cohorts comprising largely normoglycemic individuals. Cao T et al,[15] and Han AL et al,[16] observed that serum uric acid and related indices may exert a modulatory rather than a primary role in glucose homeostasis in metabolically stable populations. These findings suggest that the metabolic impact of uric acid may not be readily apparent in unselected or low-risk cohorts.

In contrast, stronger associations between UCR and metabolic abnormalities have been documented in high-risk or clinically defined populations. Liu et al,[11] and Chen et al,[12] reported significant relationships between elevated UCR and components of metabolic syndrome. Similarly, Feng et al,[13] demonstrated a robust association between higher UCR values and non-alcoholic fatty liver disease, while Zhou et al,[14] identified UCR as a predictor of insulin resistance. The relatively smaller associations found in the current study could thus be blamed on the differences in the study population whereby our cohort used routine laboratory samples as opposed to those patients with known cases of metabolic disorders.

What is more, the fact that there were no apparent age-related patterns and that the glycemic category distributions were relatively homogenous across the three UCR tertiles of the present study support this explanation. It has been previously evidenced that the pathogenic effect of uric acid is more evident in case of prolonged hyperglycemia, obesity, hepatic steatosis, or chronic inflammation. These conditions involve insufficient clinical information on the current retrospective data and this could have diluted the capacity of UCR to differentiate initial metabolic changes.

In general, though UCR is analytically more valuable, by correcting serum uric acid in the presence of a confounding independent variable, creatinine; its value as a alone indicator of metabolic risk is restricted within an overall laboratory population. This result was equal to the previous publications that stressed on the idea that uric acid-based indices are more clinically relevant in high-risk populations than in metabolically heterogeneous ones.[17,18]

Limitations: There are various limitations of the current study. The retrospective design does not allow causal inference. Vital clinical factors like the body mass index, blood pressure, history of medication, dietary consumption, and smoking history were not available, and some of them might have altered the metabolic parameters. The study is limited by the single center qualification to generalization. Outcome-based analyses and receiver operating characteristic (ROC) analyses were not done as well since there were no clear clinical endpoints.

Conclusion

The serum uric acid-to-creatinine ratio gives an easy and normalized uric acid measure that reduces the effects of gender differences. Within this retrospective laboratory-based cohort, UCR had weak correlations with glucose, and lipid parameters unless it is used in a general population superiorly to serum uric acid alone. Future, multi-centric trials should be done on persons at high metabolic risk with the aim of elucidating the clinical significance of UCR as a metabolic biomarker.

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Conflicts of interest

There are no conflicts of interest.

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