

## A Study to Correlate Serum Uric Acid with HDL-c, LDL-c, TG and CRP in non-diabetic and non-hypertensive patients attending Rama medical college hospital & Research center

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### Abstract

**Background:** In humans and higher primates, the last oxidation result of purine catabolism is uric acid. The enzyme xanthine oxidoreductase controls the final metabolic process, which involves converting hypoxanthine to uric acid. **Material and Methods:** 100 cases with no history of diabetes and hypertension will be recruited from Rama Medical college hospital and Research centre. **Results:** The analysis presented in this study focuses on the correlation between serum uric acid and HDL-c, LDL-c, TG, and CRP. This study offers important new information about the relationships between lipid characteristics and serum uric acid. The results show a substantial positive association between blood uric acid and HDL-c levels, pointing to uric acid's possible preventive role in cardiovascular health. **Conclusion:** This study provides valuable insights into the correlations between serum uric acid and lipid parameters. The findings highlight a significant positive correlation between serum uric acid and HDL-c levels, suggesting a potential protective role of uric acid in cardiovascular health.

**Keywords:** Uric acid, Triglyceride, HDL-c, LDL-c and CRP.

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### Introduction

Uric acid is the final oxidation product of purine catabolism in humans and in higher primates. The last metabolic step, the conversion of hypoxanthine to uric acid is regulated by the enzyme xanthine oxidoreductase (XO). As a part of this process reactive oxygen species (ROS) are produced. The major sources of XO are the liver and the small intestine but there are also evidences for local production of XO by the endothelium and myocardium. XO is associated with enhanced oxidative stress; XO activity is up-regulated in myocardial infarction and heart failure. The Serum Uric Acid [SUA] level reflects the net balance between its constant production and excretion. Dietary intake provides a source of uric acid precursors to maintain homeostasis, SUA is eliminated by kidney and the gastrointestinal tract.<sup>[1,2]</sup> Hyperuricemia is a metabolic consequence originating with a wide range of etiology concerned with production and excretion of uric acid and also as a combination of both. By definition hyperuricemia is the increase in urate concentration  $>420\mu\text{mol/L}$  ( $7.0\text{ mg / dl}$ ) in blood.<sup>[3]</sup> Elevated serum uric acid levels have been linked to hypertension, hyperinsulinemia, reduced physical activity,

increased body mass index (BMI), increased alcohol consumption and decreased HDL cholesterol.<sup>[4]</sup> Hyperuricemia is said to be a mediator of pro-inflammatory endocrine imbalance in the adipose tissue which may be one of the factors for dyslipidemia and the inflammatory process leading to atherogenesis.<sup>[5]</sup> Uric acid levels are not a part of any of provided metabolic syndrome definition, although the of metabolic syndrome and hyperuricemia become higher whether hyperuricemia contributes to development of metabolic syndrome or is merely a by-product of other processes that cause this disorder has not been resolved. The relationship between serum uric acid and dyslipidemia is also complex and not fully elucidated. A few studies have been conducted to investigate the association between SUA and lipid profiles in the adult population of India. In this study we aimed to assess the independent relationship between SUA and lipid profile in Indian adult. Few previous studies showed that SUA concentrations were higher in individuals with coronary heart disease than in healthy subjects and elevated SUA was found to be associated with increased cardiovascular morbidity and mortality in the general adult population. The purpose of the study was to determine whether there may be a connection between serum uric acid levels and other biomarkers, such as CRP, HDL-C, LDL-C, and triglycerides, in non-diabetic and non-hypertensive patients seeking treatment at Rama Medical College Hospital and Research Centre. The study aims to gain a better understanding of the underlying mechanisms that may contribute to the development of various cardiovascular illnesses by analyzing the link between these biomarkers. The study's conclusions may ultimately guide future interventions and treatments for people who are at risk of developing these diseases.

### Methodology

**Study Setting:** This study will be conducted in Department of Biochemistry, Rama Medical College Hospital & Research Centre Kanpur. Study taken from IPD & OPD of Medicine Department of Rama Medical College Hospital will be collected.

**Study Subjects:** 100 cases with no history of diabetes and hypertension will be recruited from Rama Medical college hospital and Research Centre.

**Study Design:** An Observational study.

**Study Period:** This study was conducted from April 2022 to March 2023.

**Inclusion Criteria:** Nondiabetic patients and Non-Hypertensive patients.

**Exclusion Criteria:** Patients with endocrinological disorders. Patients with liver disorder, renal insufficiency, pregnant women. Also acutely ill patients and patients on statins. Participants with myeloproliferative disorders and in therapy with cytotoxic drugs, pregnant women lactating mothers, renal or hepatic disorders.

**Ethical clearance:** Ethical clearance was taken from ethical committee of Rama Medical College Hospital and Research Centre. As per the Institutional Medical Ethical Committee conducted on 27/07/2022, at Rama Medical College, Hospital & Research Centre Kanpur.

**Study tool:** A pretested questionnaire based on semi-constructed proforma was used as study tool to collect the data including basic profile of participants.

**Consent:** A verbal or written consent in their own native language was obtained from the participants before the sample collection.

**Specimen collection:** 5 ml of blood sample was collected from ante-cubital vein under aseptic conditions into a fluoride vial for estimation of serum glucose & Plain vial for serum uric acid, HDL-c, LDL-c, triglyceride and C - reactive protein after explaining the procedure to the study subject.

**Specimen processing:** Blood sample was allowed to clot at room temperature for 15 minutes and serum was obtained by centrifugation at 3000 rpm (rotation per minute) for 5 minutes in the biochemistry laboratory and stored at -20 degree Celsius until assayed. The supernatant

serum will be used for the analysis of serum uric acid, serum glucose, TC, HDL-c, LDL-c, triglyceride and c-reactive.

**Statistical analysis:** Appropriate statistical test will be applied to analyze the data. All the parameters were analyzed for mean and standard deviation. The results were expressed as Mean + Standard deviation.

## RESULTS

This observational study was conducted in Department of Biochemistry, Rama Medical College Hospital & Research Centre Kanpur. Study taken from IPD & OPD of Medicine Department of Rama Medical College Hospital will be collected. Total of 100 cases with known history of Hyperuricemia and no history of diabetes and hypertension will be recruited from Rama Medical college hospital and Research Centre.

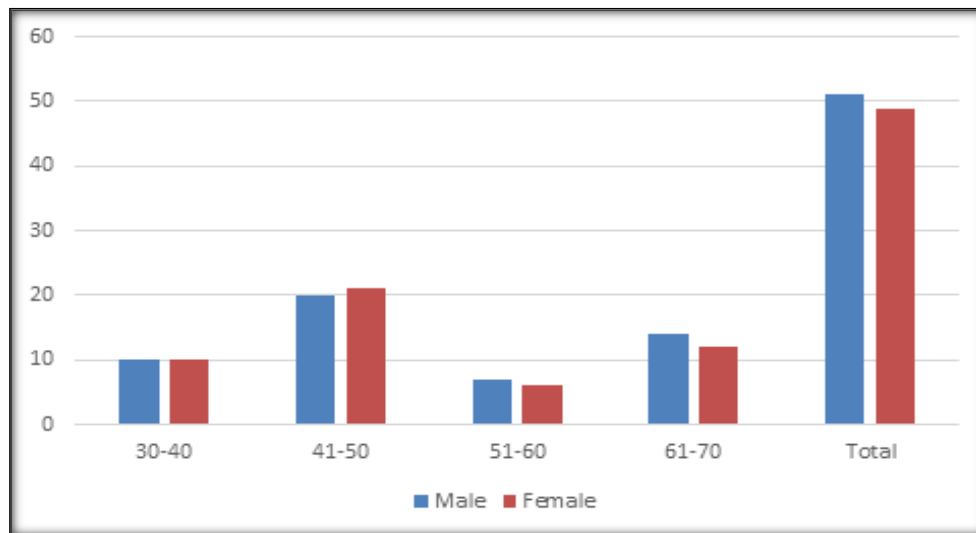


Figure 1: Age & Gender Distribution

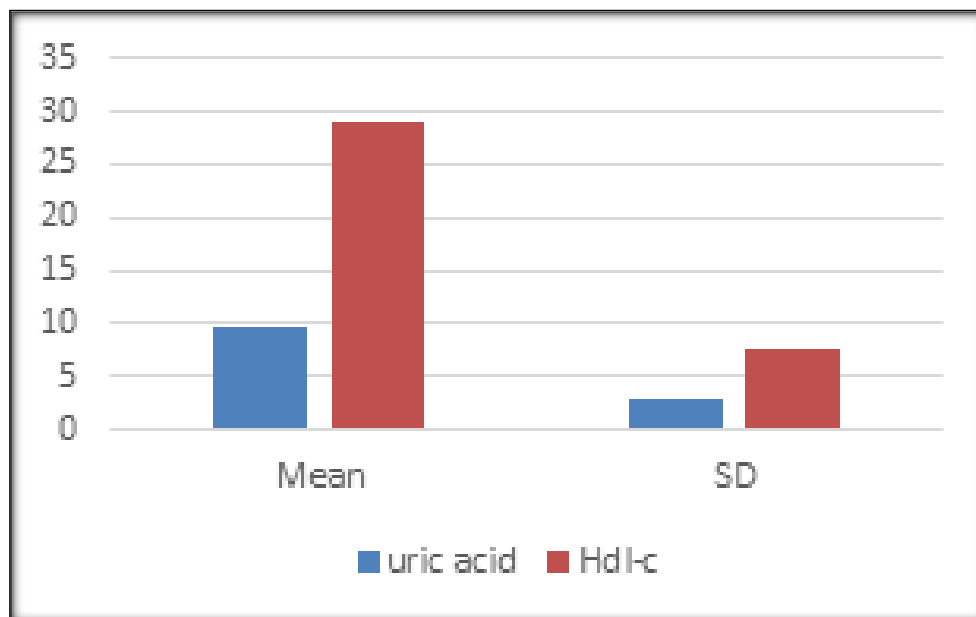


Figure 2: Showing mean and Standard deviation value for serum uric acid and HDL-C

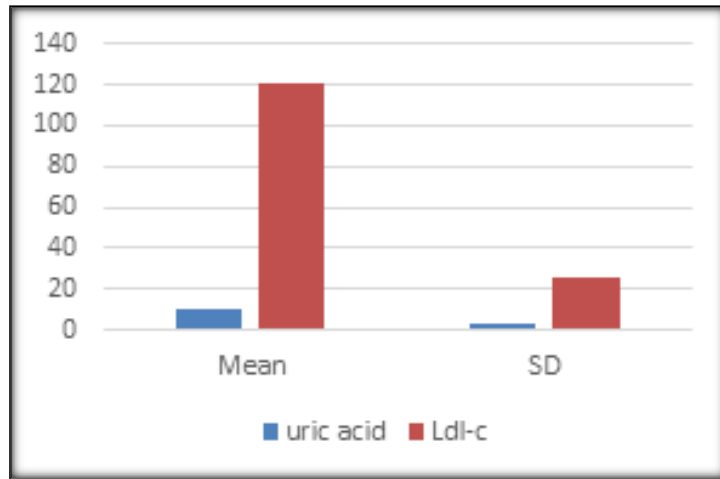


Figure 3: Showing mean and Standard deviation value for serum uric acid and LDL-c

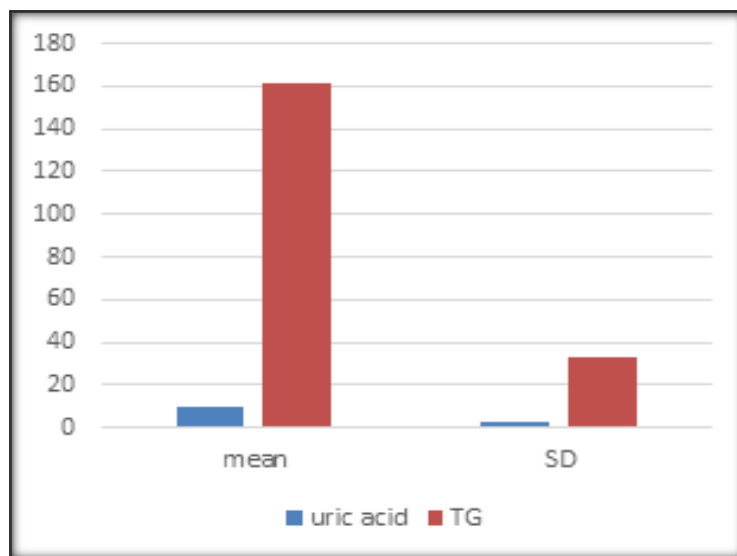


Figure 4: Showing mean and Standard deviation value for serum uric acid and TG

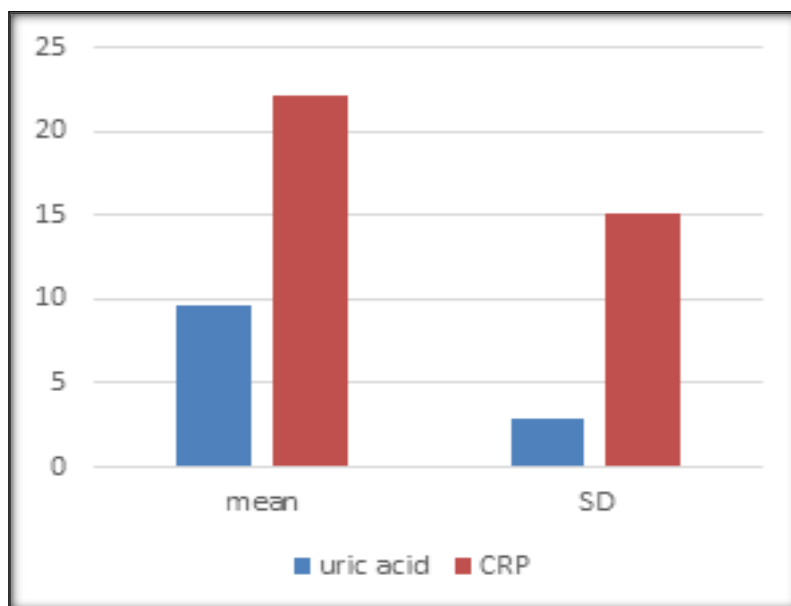


Figure 5: Showing mean and Standard deviation value for serum uric acid and CRP

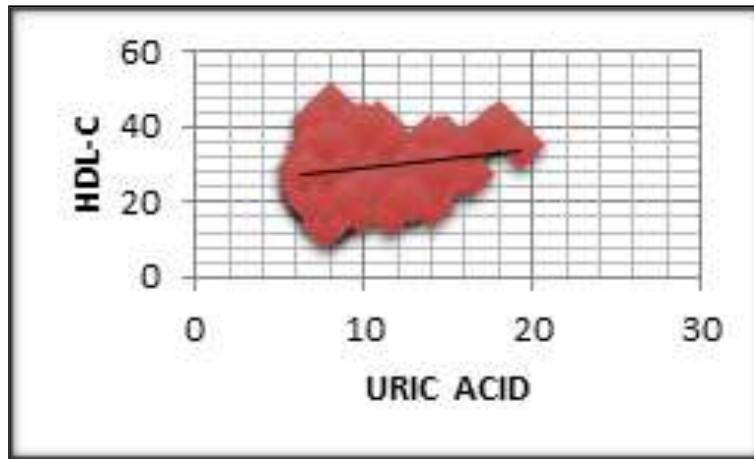


Figure 6: positive correlation between serum uric acid and HDL-C.

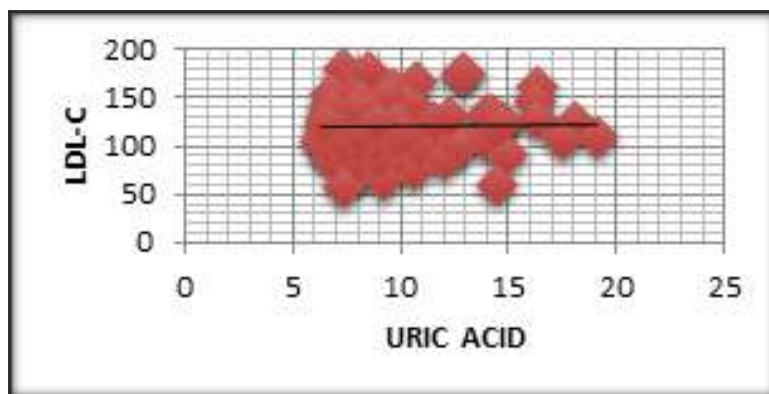


Figure 7: Showing stationary correlation between serum uric acid and LDL-C

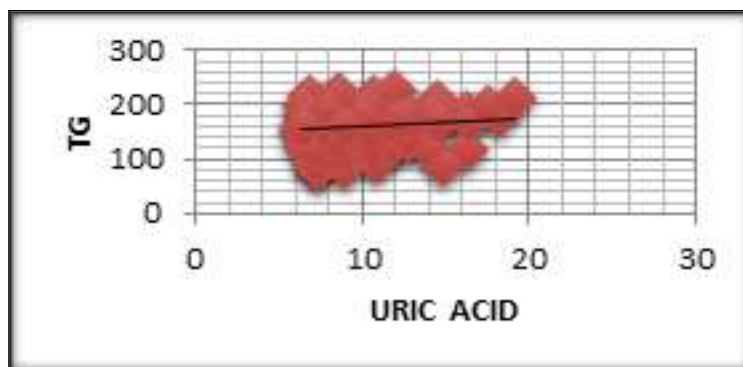


Figure 8: Showing stationary correlation between serum uric acid and Triglyceride.

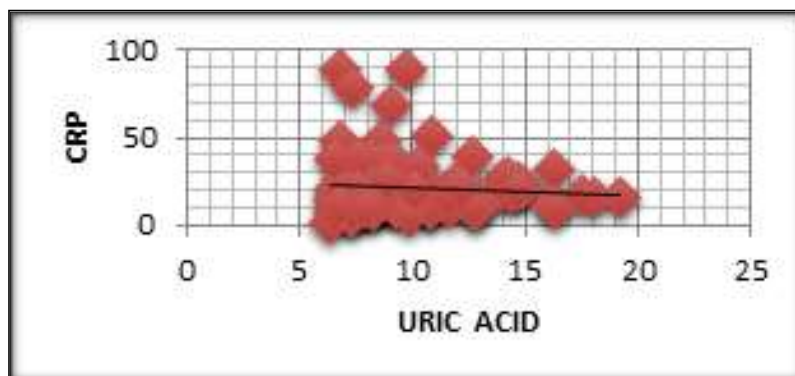


Figure 9: Showing negative correlation between serum uric acid and CRP

## DISCUSSION

The analysis presented in this study focuses on the correlation between serum uric acid and HDL-c, LDL-c, TG, and CRP. The aim is to explore the relationship between these variables and determine the significance of their associations. The study involved a sample size of 100 individuals, and the mean, variance, standard deviation, and standard error of the mean were calculated for each variable. Additionally, correlation coefficients (r-values) and their corresponding p-values were determined to assess the strength and statistical significance of the relationships. Scatter plots were generated to visualize the correlations.

**Serum Uric Acid and HDL-c:** [Figure 1] displays the analysis of serum uric acid and HDL-c. The mean serum uric acid level was 9.789, with a variance of 8.4408 and a standard deviation of 2.9053. The mean HDL-c level was 28.9503, with a variance of 56.2945 and a standard deviation of 7.503. The calculated r-value was 0.119, indicating a positive correlation between serum uric acid and HDL-c. The p-value was found to be statistically significant ( $p < 0.005$ ), suggesting a reliable association between the two variables. Similar findings were reported by Woo et al.<sup>[6]</sup> in their respective study.

**Serum Uric Acid and LDL-c:** [Figure 3] presents the analysis of serum uric acid and LDL-c. The mean serum uric acid level was 9.789, with a variance of 8.4408 and a standard deviation of 2.9053. The mean LDL-c level was 120.66, with a variance of 666.1527 and a standard deviation of 25.8487. The calculated r-value was 0.037, indicating a weak positive correlation between serum uric acid and LDL-c. However, the p-value ( $p > 0.005$ ) suggests that this correlation is not statistically significant. Minkookson et al.<sup>[7]</sup> Dyslipidemia components of serum total cholesterol, triglyceride and LDL-C levels are positively associated with serum uric acid levels. Similar findings were reported in the study.

**Serum Uric Acid and TG:** [Figure 5] presents the analysis of serum uric acid and TG. The mean serum uric acid level was 9.789, with a variance of 8.4408 and a standard deviation of 2.9053. The mean TG level was 161.908, with a variance of 1088.8947 and a standard deviation of 32.9984. The calculated r-value was 0.154, indicating a positive correlation between serum uric acid and TG. The p-value ( $p < 0.005$ ) confirms that this correlation is statistically significant. Simant Baliarsingh et al.<sup>[8]</sup> showed serum uric acid levels in the normal range might be a good indicator of the level of triglycerides and statistically significant positive correlation is observed between serum uric acid and serum triglycerides. Similar findings were reported in the study.

**Serum Uric Acid and CRP:** [Figure 7] presents the analysis of serum uric acid and CRP. The mean serum uric acid level was 9.789, with a variance of 8.4408 and a standard deviation of 2.9053. The mean CRP level was 22.1312, with a variance of 246.6066 and a standard deviation of 15.7037. The calculated r-value was -0.076, indicating a weak negative correlation between serum uric acid and CRP. However, the p-value ( $p > 0.005$ ) suggests that this correlation is not statistically significant. Nadkar et al. and Giuseppe et al.<sup>[9]</sup> also observed similar findings in their respective study. The results of this study provide insights into the relationships between serum uric acid and various lipid parameters. The analysis revealed a significant positive correlation between serum uric acid and HDL-c, indicating that higher levels of serum uric acid are associated with higher levels of HDL-c. This finding is consistent with previous research that has suggested a potential protective role of uric acid in cardiovascular health by increasing HDL-c. Interestingly, the analysis demonstrated a weak negative correlation between serum uric acid and CRP levels, although this correlation was not statistically significant. CRP is a marker of inflammation, and its relationship with uric acid may indicate potential interactions between the inflammatory response and uric acid metabolism. Further studies are needed to explore this relationship in more detail.

## CONCLUSION

In summary, this study provides valuable insights into the correlations between serum uric acid and lipid parameters. The findings highlight a significant positive correlation between serum uric acid and HDL-c levels, suggesting a potential protective role of uric acid in cardiovascular health. Additionally, a positive correlation was observed between serum uric acid and TG levels, indicating a potential association with metabolic disorders. However, the correlation between serum uric acid and LDL-c was statistically significant, and the correlation with CRP was weak and non-significant. Overall, this study contributes to our understanding of the relationships between serum uric acid and lipid parameters. The findings have potential implications for risk assessment, prevention, and management of cardiovascular and metabolic disorders. Future research in this field will provide a more comprehensive understanding of the complex interactions between uric acid and lipid metabolism.

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