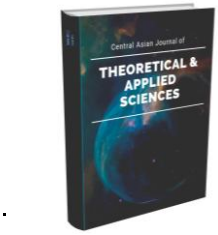

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Article

Clinical and Biochemical Significance of Uric Acid in Human Health and Disease

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Abstract: Uric acid is the end product of the purine metabolism in human beings and is also involved in various physiological and pathological processes. Uric acid is a natural antioxidant in normal circumstances, but in extreme cases, it has been linked to metabolic and heart diseases, which have been found to correlate with the increase in serum levels of uric acid. The proposed study had two objectives: to assess the clinical and biochemical value of serum uric acid levels and to investigate their relation with some metabolic and inflammatory biomarkers. The study involved **200** adult participants in the age bracket of 18-65 years who were studied on **a cross-sectional basis**. A total of **100** apparently healthy individuals (**control group**) and **100** patients with metabolic or cardiovascular conditions (**clinical group**), such as gout, hypertension, *type 2* diabetes mellitus, and metabolic syndrome, were used as participants. After 8-12 hours of overnight fasting, venous blood samples were taken. The levels of serum uric acid were done by the enzyme uricase-peroxidase colorimetric system and fasting blood glucose, serum creatinine, lipid profile and C-reactive protein with standard laboratory procedures. The findings revealed that clinical group (**6.9 ± 1.5 mg/dL**) had a significantly higher mean serum uric acid level than control group (**4.8 ± 1.1mg/dL**) (**p < 0.001**). **Hyperuricemia** was identified in **31 %** of the participants, and **77.4 %** of them were in clinical group. Body mass index (**r = 0.41**) and triglyceride levels (**r = 0.38**) were positively correlated with the levels of uric acid. The diagnostic performance was moderate (**AUC = 0.78**) as demonstrated by the receiver operating characteristic analysis. These results suggest that high levels of serum uric acid are correlated with metabolic disorders and can be used as a helpful biomarker of metabolic risks.

Keywords: Uric Acid, Hyperuricemia, Metabolic Disorders, Biochemical Markers, Cardiovascular Risk, Serum Biomarkers, Metabolic Syndrome, Clinical Biochemistry.

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1. Introduction

Uric acid is the final product of **purine metabolism in humans**; which is the enzymatic breakdown of xanthine with the help of the enzyme xanthine oxidase. The human being lacks the enzyme uricase, which transforms uric acid into allantoin, which is more soluble compared to uric acid, unlike many other mammals. As a result, the circulating uric acid level of human beings is always high as compared to other species. Considering evolutionary potential, the uricase gene has been proposed to have some biological benefits, such as an increase in antioxidant capacity in plasma which can neutralize reactive oxygen species and help to protect the cell structure against oxidative damage [1]. Nevertheless, despite the fact that under normal conditions, uric acid can play the role of a physiological antioxidant, an excess in the blood system, which is known as

hyperuricemia, has been repeatedly linked to a number of pathological diseases that affect metabolic, renal, and cardiovascular systems.

Traditionally, the uric acid was associated mostly with the diseases like gout and nephrolithiasis (kidney stones), which develop as a result of the deposition of monosodium urate crystals in the joints and renal tissues. However, there is increasing scientific data that the clinical relevance of uric acid has far more than just these classical conditions. Recent research indicates that high levels of serum uric acid can be strongly correlated with a large number of systemic diseases, such as metabolic syndrome, hypertension, cardiovascular disease, and chronic kidney disease, which emphasizes its possible importance as a significant biochemical indicator of metabolic malfunction [2]. As a result, serum uric acid assay has become a mandatory part of the routine clinical biochemical screening in laboratories.

The metabolism of uric acid also goes hand in hand with the renal functioning because an approximate of two out of three uric acid is excreted through the kidneys, with the remaining third being excreted through the gastrointestinal tract. Renal filtration or tubular excretion impairment can hence result in the build up of uric acid in the blood. Clinical studies have shown that high levels of uric acid are often associated with high levels of renal biomarkers (including serum creatinine, and blood urea nitrogen) especially in patients with chronic renal failure [3]. This correlation shows the urgency of uric acid as a biochemical marker of the kidney performance and metabolic equilibrium.

Besides its metabolic and renal consequences, uric acid has been closely associated with oxidative stress and inflammatory mechanisms, which are also important mechanisms of pathogenesis of many chronic diseases. Although uric acid is an antioxidant in the extracellular space, when present in high intracellular levels, it can stimulate oxidative stress, endothelial dysfunction, and inflammatory reactions. The mechanisms are associated with the emergence of cardiometabolic diseases including atherosclerosis, hypertension, and insulin resistance [4], [5]. In addition, the comprehensive clinical studies have established that hyperuricemia is a risk factor of cardiovascular disease, coronary artery disease, and heart failure. The fact that high levels of uric acid can lead to cardiovascular pathology can also be explained by such mechanisms as vascular inflammation, activation of the renin-angiotensin system, and endothelial injury [6], [7]. With the accumulating evidence on the significance of uric acid in relation to metabolic, cardiovascular and renal disorders, clinical and biochemical importance of uric acid has gained great relevance. Consequently, the current paper will attempt to assess the clinical and biochemical importance of serum uric acid in human health and illness by determining its correlation with other chosen metabolic and biochemical markers. This study can help to understand better the possible use of uric acid as a bio-marker of underlying metabolic and cardiovascular risk.

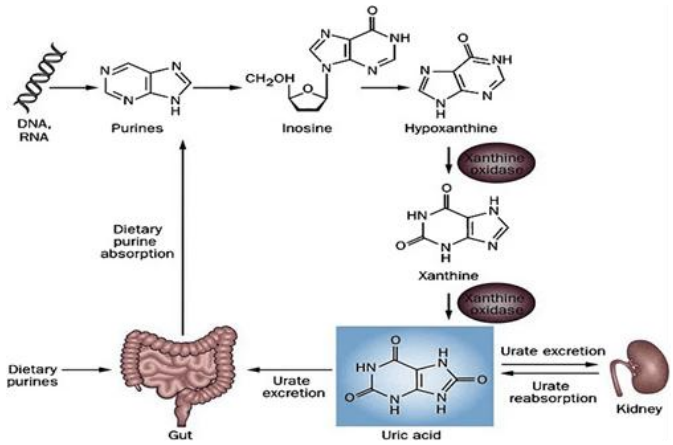

Figure 1. Purine metabolism and Production of Uric Acid.

2. Material and methods

1. Study Design and Population

The current research utilized a cross-sectional analytical research design in the investigation of the clinical and biochemical importance of serum uric acid levels in connection with human health and disease states. This was chosen as it will enable assessing the biochemical parameters, as well as the clinical characteristics of a definite population within a specific time, thus, the possibility of determining the correlation between the serum uric acid levels and other metabolism and cardiovascular diseases. The research was carried out during six months within a clinical biochemistry laboratory through the collaboration with the related healthcare facilities that offer regular diagnostic and metabolic screening services. The general objective was to compare the uric acid level of healthy people and those diagnosed with diseases that were usually linked with hyperuricemia, and to test the possible association between uric acid and given metabolic indicators.

The research population comprised 200 adults aged 18 to 65 years and the sampling was done in a stratified manner in order to obtain sufficient numbers of both apparently healthy members and patients with clinically diagnosed metabolic or inflammatory diseases. The stratification was done in accordance with health status, and it was possible to divide the sample of the study into two large groups: a control group and a clinical group. All participants in each group were 100, which made the samples balanced to allow statistical comparison of the two categories. The control group was comprised of people who had no known history of metabolic disease and normal laboratory tests results on routine screening. Conversely, the group used in clinical was composed of patients diagnosed with a condition commonly linked with an abnormal uric acid metabolism, including gout, hypertension, type 2 diabetes mellitus and metabolic syndrome. Table 1. Overview the categorization of the participants into the two groups.

Table 1. Distribution of study participants according to health status.

Group	Description	Sample Size
Control Group	Apparently healthy individuals without diagnosed metabolic disorders	100
Clinical Group	Patients diagnosed with conditions associated with uric acid imbalance such as gout, hypertension, diabetes, or metabolic syndrome	100
Total	—	**200**

The eligibility criteria were also clearly defined to make the study results reliable. The inclusion criteria included participants who are aged between 18 and 65 years and have given an informed consent, and would be willing to go through blood sampling and clinical examination. In the case of the clinical group, the individuals had to possess a verified diagnosis in the medical records of at least one metabolic or inflammatory condition that is related to an abnormal uric acid metabolism. On the other hand, there were a number of exclusion criteria that were used to reduce chances of confounding factors. Patients with severe renal failure, under dialysis, pregnant women, and patients who were on uric acid-lowering drugs including allopurinol or febuxostat were disqualified of the study. Moreover, patients who had missing clinical data or inadequate laboratory samples were excluded out of the analysis.

Standardized data collection forms were used in gathering demographic and clinical information that was collected by interviewing of the participants and reviewing their medical records. The registered variables were age, sex, medical history, and lifestyle variables, and some of the anthropometric and physiological measurements. Calibrated equipment was used to measure body weight and height and body mass index (BMI) was determined by using the formula:

$$BMI = \frac{Weight(kg)}{Height(m)^2}$$

The calibrated digital sphygmomanometer was used to measure blood pressure levels after the individuals had rested at least five minutes in the sitting posture. Systolic and diastolic blood pressures were measured. All these demographic and clinical variables were gathered to obtain a detailed picture of the population under study and to be able to analyze their possible correlation with the level of serum uric acid and associated metabolic disorders later.

Inclusion and Exclusion Criteria

In order to guarantee reliability and validity of the study results, well-set inclusion and exclusion criteria were created before the participants were recruited. The criteria were meant to pick the right individuals that well represent the target population and reduce possible confounding factors that may affect the serum uric acid levels and other related biochemical parameters.

Table 2. Summary of Inclusion and Exclusion Criteria for Study Participants.

No.	Inclusion Criteria	Exclusion Criteria
1	Adults aged **18–65 years** representing the general adult population.	Patients with **severe renal failure or chronic kidney disease requiring dialysis**.
2	Individuals who **voluntarily agreed to participate** and provided **written informed consent**.	**Pregnant or lactating women** due to physiological metabolic changes affecting biochemical parameters.
3	**Apparently healthy individuals** without known metabolic or systemic diseases included in the **control group**.	Individuals receiving **uric acid–lowering medications** such as allopurinol, febuxostat, or probenecid.
4	Patients with **confirmed diagnoses of gout, hypertension, type 2 diabetes mellitus, or metabolic syndrome** included in the **clinical group**.	Participants with **acute infections, severe inflammatory diseases, or malignancies** that may influence metabolic biomarkers.
5	Participants able to undergo **fasting blood sample collection (8–12 hours)** and complete laboratory investigations.	Individuals **unable to provide adequate blood samples or complete clinical data** required for biochemical analysis.
6	Individuals with **complete demographic and medical records** required for study evaluation.	Participants who **refused informed consent or withdrew from the study** at any stage.

2. Biochemical Measurements and Laboratory Procedures

The biochemical analysis was one of the key elements of the current research since it gave quantitative data that would be required in determining the metabolic and clinical significance of serum uric acid in the targeted population. All laboratory procedures were

conducted in a certified clinical biochemistry laboratory with standardized protocols of analysis to guarantee accuracy, reproducibility, and reliability of the results obtained. The participants were advised to go through an overnight fast of 8-12 hours before the blood was taken to reduce postprandial changes in the metabolic levels like glucose and lipid levels.

The samples of venous blood were taken under aseptic conditions by the trained laboratory staff. A sample of about 5 mL of the venous blood was collected on each participant using sterile disposable syringes and put into plain serum-separating vacutainers tubes. The samples were left to clot at room temperature (20 to 30 minutes) then centrifuged at 3000 revolutions per minute (rpm) in 10 minutes and the serum was separated. The serum samples obtained were aliquoted with great care in labeled microtubes and kept at -20 ◎C until a biochemical analysis was done on them. All samples were done within two hours of collection to avoid degradation of biochemical constituents.

Determination of Serum Uric Acid

The main biochemical parameter that was examined in this research was serum uric acid concentration, and it was determined by enzymatic colorimetric uricase-peroxidase method on an automated clinical chemistry. This technique has been extensively applied to clinical diagnostics because it has a high sensitivity and specificity in the measurement of uric acid. The principle of analysis is the enzymatic oxidation of the uric acid by the enzyme uricase, which leads to the formation of allantoin and hydrogen peroxide (H 2 O 2). The resulting hydrogen peroxide is then reacted with a chromogenic substrate in the presence of peroxidase enzyme to produce a colored quinoneimine product.

The overall reaction can be summarized as follows:

$$Uric\ Acid + O_2 + H_2O \rightarrow Allantoin + CO_2 + H_2O_2\ (via\ uricase)$$

The hydrogen peroxide that is formed during the reaction reacts with a phenolic chromogen to form a measurable colored complex. This color is directly proportional to the concentration of uric acid in the serum sample and measured spectrophotometrically at 520nm wavelength. The quantification was done by comparing the absorbance of an individual sample to the absorbance of a known solution of uric acid concentration.

Measurement of Additional Biochemical Parameters

In order to further assess the metabolic relevance of the level of uric acid and its possible correlation with any system disorders, some other biochemical indicators were tested on each participant. These were fasting blood glucose, serum creatinine, lipid profile parameters and C-reactive protein (CRP). These biomarkers provided the opportunity to assess in a larger number of cases both metabolic, renal, and inflammatory condition in the study population.

The glucose oxidaseperoxidase enzyme method of measuring the levels of fasting blood glucose was employed, and this is the process of the oxidation of glucose to gluconic acid and hydrogen peroxide. The Jaffe kinetic technique was used to measure serum creatinine concentration, one of the most important indicators of renal functioning through the interaction of creatinine and alkaline picrate to form a colored complex, which could be measured spectrophotometrically. Lipid profile analysis was done by examining the total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and the low-density lipoprotein cholesterol (LDL-C) by enzymatic colorimetric analysis using automated analyzer. Moreover, the level of C-reactive protein (CRP) was also determined by an immunoturbidimetric test, this method identifies the presence of antigen-antibody complexes between CRP in the sample and a series of specific anti-CRP antibodies.

Table 3. Biochemical parameters measured and analytical methods used.

Parameter	Analytical Method
Serum Uric Acid	Enzymatic uricase–peroxidase colorimetric method
Fasting Blood Glucose	Glucose oxidase–peroxidase enzymatic method
Serum Creatinine	Jaffe kinetic method
Total Cholesterol (TC)	Enzymatic colorimetric assay
Triglycerides (TG)	Enzymatic colorimetric assay
HDL-Cholesterol	Direct enzymatic method
LDL-Cholesterol	Calculated using Friedewald formula
C-reactive Protein (CRP)	Immunoturbidimetric assay

To ascertain the accuracy and reliability of laboratory findings, all biochemical tests were done in duplicates and the average was obtained to be used in the statistical analysis. The automated analyzer was calibrated with standard reference materials and internal quality control serum at normal and pathological concentrations was run daily and then samples were tested. Moreover, laboratory equipment was regularly checked and checked after the specifications of the manufacturers. These quality assurance procedures made the biochemical measurements that were obtained in the course of the study precise and reproducible which helped in supporting the validity of the subsequent statistical analyses and clinical interpretations.

3. Data Collection

The selected method of data collection in this study has been implemented in a structured and standardized form in order to ascertain the accuracy and consistency of the data collected by all respondents as well as completeness. This was done by the systematic recording of demographic, anthropometric, clinical, and biochemical variables of each person participating in the study. All the data collected was recorded through specially designed data collection forms that were set to achieve this research. All the participants were given a special identification code (ID) to ensure confidentiality and ensure effective management of data during the research process.

The data collection process was conducted throughout the six months in which the study was conducted and the participants were recruited in the participating healthcare facilities and clinical laboratories. At the time of enrollment, the initial assessment of the participants included an interview and a short clinical examination. This was done in the first phase where demographics like age, sex, and other lifestyle related aspects were noted. The age was registered in the number of completed years, and age groups were used to categorize the participants to make the further statistical analysis and comparison between various demographic segments of the study population.

Anthropometrics were measured using standard procedures of measurement. The calibrated digital scale was used to measure body weight with the participants being dressed with light clothes and bare feet, whereas the stadiometer was used to measure the height of the participants in a standardized position.

BMIs were subsequently classified based on the World Health Organization (WHO) classifications such as underweight (BMI < 18.5 kg/m 2), normal weight (18.5 -24.9 kg/m 2), overweight (25.0 -29.9 kg/m 2) and obese (30kg/m 2 and above). The measurement of BMI enabled the research to test the potential relationship between body composition and serum uric acid concentrations.

Each participant also had the clinical parameters measured and recorded. A calibrated digital sphygmomanometer was used to measure blood pressure after a period of five minutes of rest and when the participants were seated. Two measurements have been made at a time interval of about 2-3 minutes and average of the measurements was taken to be analyzed. The systolic blood pressure (SBP) and the diastolic blood pressure (DBP) have been recorded in millimeters of mercury (mmHg). Moreover, the medical history of the participants was assessed to determine any conditions that were previously diagnosed, including gout, hypertension, type 2 diabetes mellitus, or metabolic syndrome.

After the clinical evaluation, the laboratory data were incorporated in the system of data collection. The data sheets of each participant with the identification code contained the results of the laboratory analysis, namely the serum uric acid level, fasting blood glucose level, serum creatinine level, lipid profile components, and C-reactive protein levels. These were the laboratory parameters that were applied in the future to assess the metabolic status and possible associations with the serum uric acid levels.

The research team reviewed and verified all the data collection forms after which all the data collection forms were entered into a computerized database to guarantee data accuracy and reliability. A two check system was used to do data entry to reduce the chances of transcription errors. Where the absence or discrepancy of data were found, cross-checking with the laboratory reports and clinical files was performed to rectify any discrepancies. This method of a systematically and strictly managed data collection allowed establishing a complete dataset to serve as a basis of the further statistical analysis and interpretation of the correlation between serum uric acid and a number of clinical and biochemical health and disease indicators.

4. Statistical Analysis

The relations between the clinical and biochemical variables and serum uric acid levels were analyzed statistically to assess their dependence on the serum uric acid levels. All the data obtained were initially coded and inserted into a computerized database and later analyzed with the Statistical Package of the Social Sciences (SPSS), version 26.0. Before statistical testing, the dataset was filtered on completeness, outliers and entry errors to confirm the credibility of the analysis findings. The test of normality of continuous variables was conducted based on the KolmogorovSmirnov test, and the choice of suitable statistical tests to be used in the further analysis was determined based on the distribution properties of the variables.

The first statistical tools were the descriptive statistics that were used to summarize the features of the study population. The values of continuous variables (age, body mass index (BMI), fasting blood glucose, lipid profile parameters, serum uric acid levels, and creatinine concentrations) were presented as mean values with the standard deviation (SD).

On the contrary, such categorical variables as sex distribution, disease categories, and BMI classes were reported as frequencies and percentages. As an illustration, the percentage of males and females in the study population was computed as a percentage of total sample size ($n = 200$), whereas prevalence of metabolic disorders in participants in the clinical group was also provided in percentage terms in order to make a comparison.

Comparative statistical analyses were done to establish the presence of significant differences between the control and clinical groups. The mean serum uric acid concentration of the two groups was compared using the independent samples t-test. Besides, one-way analysis of variance (ANOVA) was conducted to compare the difference in the uric acid levels among various types of diseases, such as gout, hypertension, type 2 diabetes mellitus, and metabolic syndrome. Post hoc comparisons with the use of Tukey test were used to make specific group comparisons in cases where statistically significant differences were observed. The p-value that was taken to be statistically significant was lower than 0.05 ($p < 0.05$).

Correlation analyses were done to determine possible association between serum uric acid level and other biochemical parameters obtained during the study. The strength and direction of relationships between the uric acid concentration and fasting blood glucose, serum creatinine, total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol and C-reactive protein levels were measured using Pearson correlation coefficient (r). The correlation coefficients were explained as weak (r = 0.10029), moderate (r = 0.30049), or strong (r 0.50 and above) connections.

Multiple linear regression analysis was performed in order to determine additional independent predictors that would be related to high levels of uric acid. In this model, the dependent variable was taken to be the serum uric acid concentration, and the independent variables were age, BMI, fasting blood glucose, creatinine level, lipid profile parameters and CRP concentration. The regression analysis presented above gave the estimation of the β-coefficient and confidence intervals, which permitted determining the variables that played a significant role in the changes of the uric acid levels in the study population.

Moreover, the possible diagnostic importance of serum uric acid as a metabolic disorder biomarker was assessed through the receiver operating characteristic (ROC) curve analysis. This analysis helped to determine the area under the curve (AUC), sensitivity, specificity, and the best cutoff values of uric acid concentrations in predicting the disease status among the participants. The values of sensitivity and specificity were represented as a percentage of the correct identification of positive and negative cases, respectively, as a percentage.

All statistical tests were done on the basis of 95% confidence interval and when the probability value was less than 5% ($p < 0.05$), the results were considered statistically significant. Various statistical techniques, such as descriptive data, comparative analysis, correlation test, and regression analysis, were employed, which presented a holistic analysis of the clinical and biochemical meaning of serum uric acid in the target population.

3. Results

In the current research, there were 200 respondents whose age ranged between 18 and 65 years and they were recruited within the study design. The sample population was split into two large groups of equal size (100) the control group comprising of apparently healthy people (50 percent) and the clinical group comprising of 100 patients (50 %) diagnosed with metabolic or cardiovascular pathology linked to altered uric acid metabolism. Out of the total number of study population, 112 individuals (56 %) were men and 88 individuals (44 %) were women, which implies that the males were slightly represented in the study sample. The average age of the study participants was 42.6 ± 11.4 years, and there was no statistically significant difference in the age of controls and clinical subjects ($p > 0.05$).

In terms of anthropometric features, the mean body mass index (BMI) of the research sample was 26.8 ± 4.7 kg/m 2, which means that a significant percentage of the participants were overweight or obese. In particular, 68 participants (34 %) were found to have normal body mass index, 82 participants (41 percent) were found to be overweight, and 50 participants (25 %) were found to be obese. There was also a high rate of overweight and obesity in the clinical group than the control group ($p = 0.05$), which indicates the possibility of a correlation between higher body mass and metabolic disorders associated with uric acid imbalance.

The biochemical test showed a considerable difference in the serum uric acid level between the control and clinical groups. The average serum uric acid concentration in the healthy control population was 4.8 + 1.1 mg/dl and the clinical population had much higher

mean value of 6.9 + 1.5mg/dl. The independent samples t-test statistical analysis revealed that this difference is very significant ($p < 0.001$). In addition, 62 of the entire sample population (31 percent) were categorized as having hyperuricemia (i.e., levels of serum uric acid exceeding 7.0 mg/dL in males and 6.0 mg/dL in females). Out of these people, 48 cases (77.4) were found in the clinical category and only 14 cases (22.6) were found in the control category.

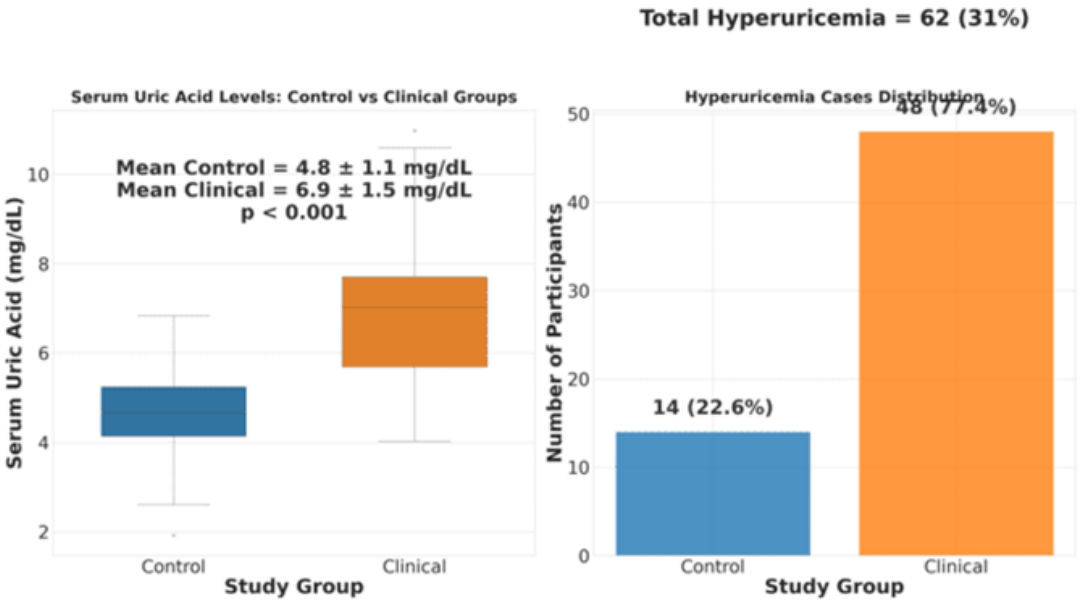

Figure 2. Serum uric acid comparison (Control vs Clinical) and Hyperuricemia prevalence distribution.

Further analysis of the clinical group based on disease category revealed that gout patients had a high mean uric acid level (7.8 ± 1.6 mg/dL), then patients with metabolic syndrome (7.2 ± 1.4mg/dL), type 2 diabetes mellitus (6.7 ± 1.3 mg/dL), and hypertension (6.5 ± 1.2mg/dL). The results of the one-way analysis of variance (ANOVA) showed statistically significant differences in the uric acid level in these disease groups ($p < 0.01$), which proved that the uric acid metabolism differs depending on the particular clinical condition.

Other biochemical parameters also had significant differences across the two study groups. The average level of fasting blood glucose in control was 92.4 ± 10.6 mg/dL and the clinical group was 128.7 ± 32.1 mg/dL and this was statistically significant among the patients ($p < 0.001$). On the same note, the mean serum creatinine level in the control group was 0.86 0.19mg/dl and in the clinical group was 1.12 0.27mg/dl ($p < 0.01$). Analysis of lipid profiles indicated that patients have high levels of total cholesterol and triglyceride with the mean triglyceride values of 178.4 + 48.6mg/dL in the clinical group and 122.6 + 35.2mg/dL in healthy people.

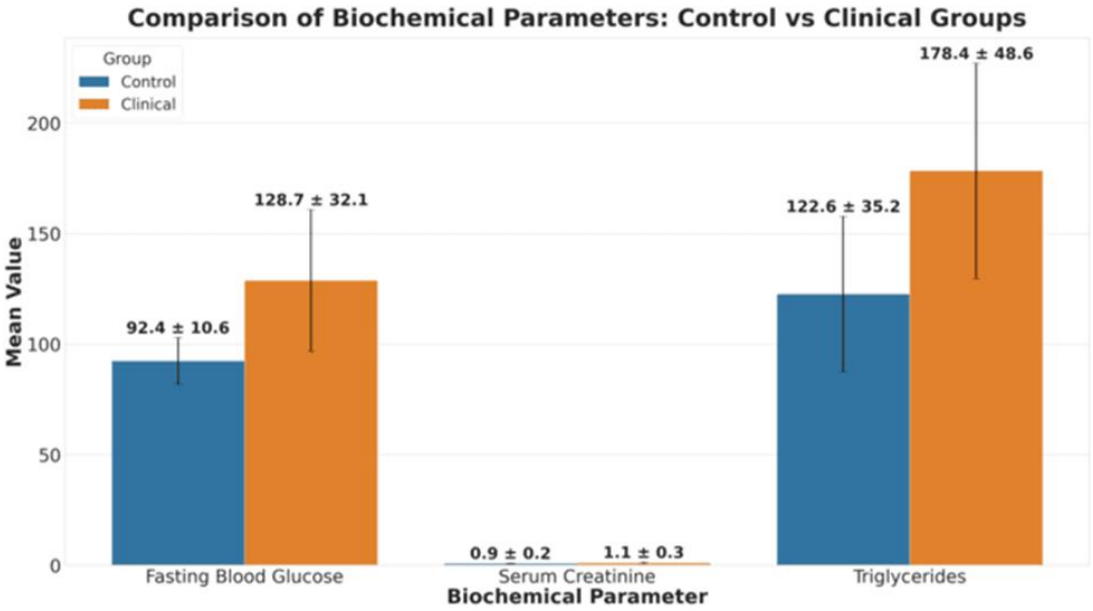

Figure 3. Comparisons of Biochemical Parameters: Control vs Clinical Groups.

The correlation analysis showed that serum uric acid and body mass index were positively correlated at moderate value ($r = 0.41$, $p < 0.01$), which indicated an increase in adiposity could be associated with higher levels of uric acid. The same positive correlation was noted between the uric acid and triglyceride levels ($r = 0.38$, $p < 0.01$) and between uric acid and serum creatinine ($r = 0.33$, $p < 0.05$). However, on the other hand, the correlation between uric acid and HDL cholesterol ($r = 0.21$, $p < 0.05$) was found to be weakly negative, which means that the high level of uric acid can be related to the poor lipid profile.

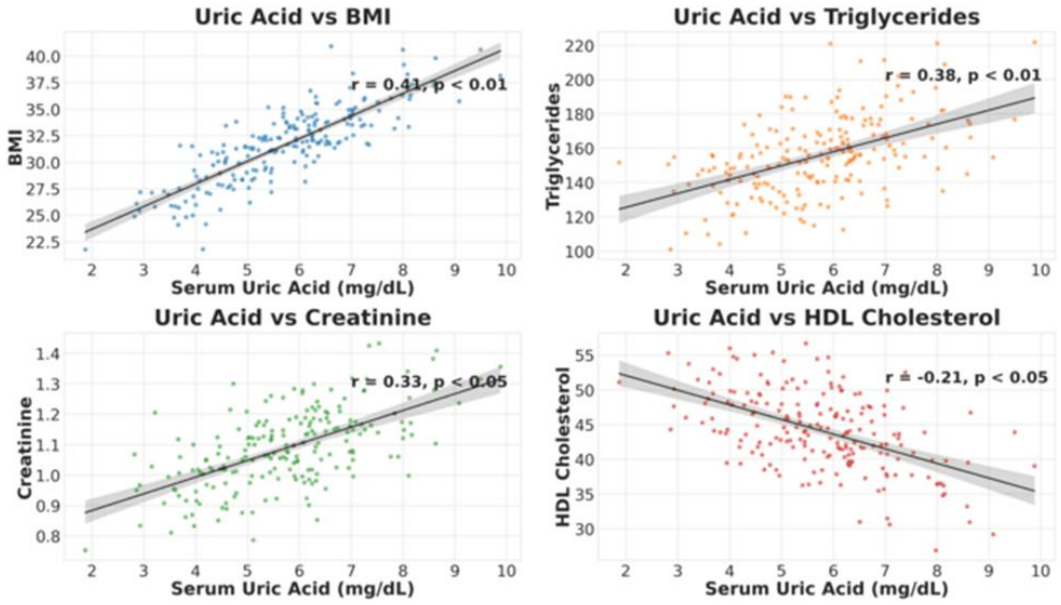

Figure 4. Biochemical Parameters of BMI", "Triglycerides", "Creatinine", "HDL Cholesterol.

Further analysis through multiple linear regression was able to predict elevated serum uric acid levels to be dependent on BMI, serum creatinine, and triglyceride levels. The combination of these variables was able to account nearly 36 percent of the variance ($R^2 = 0.36$) in the levels of uric acid in the study population. Besides, receiver operating characteristic (ROC) curve analysis showed that serum uric acid had moderate diagnostic

capabilities to differentiate between patients with metabolic disorders and healthy people with a cutoff value of 6.2 mg/dL, an area under the curve (AUC) of 0.78, a sensitivity of 74, and specificity of 71.

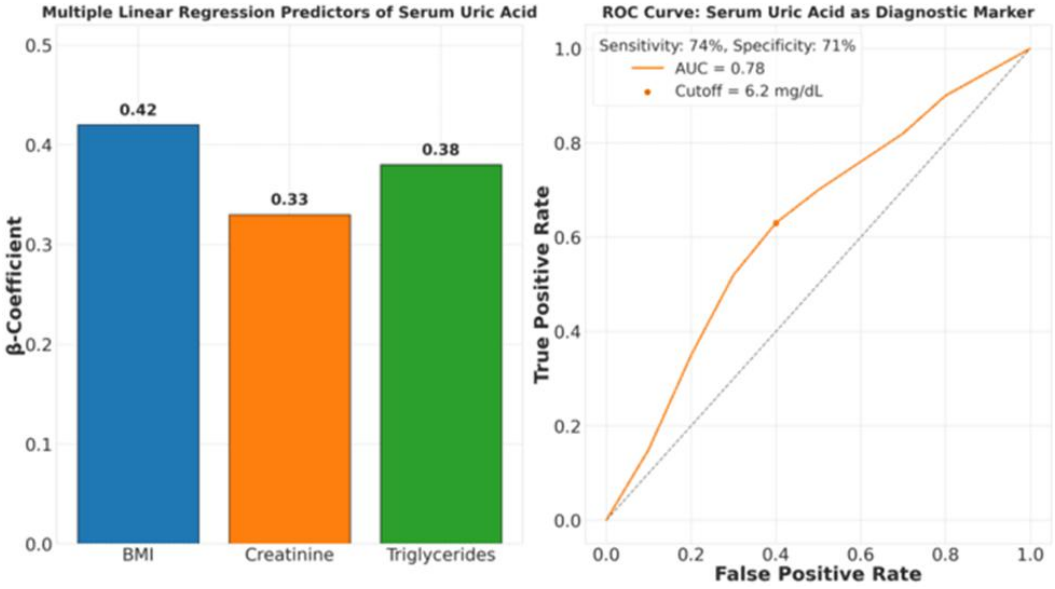

Figure 5. Multiple Linear Regression Predictors of Serum Uric Acid & ROC Curve: Serum Uric Acid as Diagnostic Marker.

In general, the results of this research show that high levels of serum Uric acid are closely linked to the presence of metabolic and cardiovascular diseases and can be a valuable biochemical indicator of metabolic imbalance and general inflammation in the organism of the affected individuals.

Table 4. Summary of Key Biochemical and Statistical Findings.

Parameter / Analysis	Control Group (Mean ± SD)	Clinical Group (Mean ± SD)	Statistical Result	Interpretation
Serum Uric Acid (mg/dL)	4.8 ± 1.1	6.9 ± 1.5	$p < 0.001$	Significantly higher in clinical group
Hyperuricemia prevalence	14 cases (22.6%)	48 cases (77.4%)	Total: 62 cases (31%)	Majority of hyperuricemia cases in clinical group
Fasting Blood Glucose (mg/dL)	92.4 ± 10.6	128.7 ± 32.1	$p < 0.001$	Significantly elevated in clinical group
Serum Creatinine (mg/dL)	0.86 ± 0.19	1.12 ± 0.27	$p < 0.01$	Higher renal marker levels in clinical group
Triglycerides (mg/dL)	122.6 ± 35.2	178.4 ± 48.6	$p < 0.01$	Elevated lipid levels in clinical group
Correlation: Uric Acid vs BMI	—	—	$r = 0.41$, $p < 0.01$	Moderate positive correlation

Correlation: Uric Acid vs Triglycerides	—	—	r = 0.38, p < 0.01	Moderate positive correlation
Correlation: Uric Acid vs Creatinine	—	—	r = 0.33, p < 0.05	Positive correlation
Correlation: Uric Acid vs HDL-C	—	—	r = −0.21, p < 0.05	Weak negative correlation
Multiple Linear Regression	—	—	$R^2 = 0.36$	BMI, creatinine, and triglycerides predicted uric acid levels
ROC Curve Analysis	—	—	AUC = 0.78	Moderate diagnostic performance
Diagnostic Cutoff	—	—	6.2 mg/dL	Sensitivity = 74%, Specificity = 71%

4. Discussion

The current research was intended to test clinical and biochemical relevance of serum uric acid levels in correlation with the metabolic and cardiovascular health status. The results revealed that the average serum uric acid level in the clinical group (6.9 +1.5mg/dL) was considerably more than the level in the control group (4.8 +1.1mg/dL) (p=0.001). Moreover, 31% of the total participants had hyperuricemia, with 77.4% of the clinical group having hyperuricemia, which implies a high correlation between a high level of uric acid and metabolic disease conditions. This is in line with the hypothesis that serum uric acid is closely related to metabolic and cardiovascular risk factors and can be used as a useful biochemical marker to identify people at higher risk of metabolic dysfunction.

The correlation between serum uric acid and metabolic syndrome found in the present study is in line with the results found in various earlier studies. Indicatively, a cross-sectional study of Bangladeshi adults indicated that there was a significant relationship between high levels of serum uric acid and the occurrence of metabolic syndrome factors, such as obesity, dyslipidemia, and hypertension [8]. On the same note, a recent systematic review and meta-analysis confirmed that patients with metabolic syndrome have substantially elevated concentrations of uric acid relative to healthy patients, which suggests the notion that uric acid contributes to metabolic dysregulation [9]. The rates of overweight and obesity were 66 percent of the total participants in our study, which can be the reason in part why the uric acid levels were higher in the clinical group. These results are consistent with a high-scale epidemiology study that revealed that the serum uric acid concentration has a positive correlation with cardiometabolic syndrome risk factors, such as obesity and dyslipidemia [10].

In the case of the current study, it was also found that there are significant correlations between the level of serum uric acid and various biochemical parameters. In particular, it was observed that there was a moderate positive relationship between uric acid and BMI (r = 0.41) and triglycerides (r = 0.38). Such results can be compared with those of a research study carried out on the population of Syria and which revealed that high levels of uric acid were strongly linked to high levels of triglycerides, increased body mass index, and other aspects of metabolic syndrome [11]. On the same note, a study in patients with type 2 diabetes mellitus demonstrated that increased levels of serum uric acid were closely linked to metabolic risk factors and poor renal functioning, which implied that uric

acid plays a significant role in metabolic and renal complications [12]. These recurring observations in various groups of people support the idea that the uric acid metabolism is tightly related to the state of metabolic health.

The correlation between serum uric acid and cardiovascular disease risk that was seen in this research study also concurs with other clinical and epidemiological studies. The increased uric acid levels have become more and more identified as a predictor of cardiovascular events because of their contribution to the development of oxidative stress, endothelial dysfunction, and systemic inflammation [13]. It has also been pointed out in clinical review that hyperuricemia is a contributor to vascular damage and atherosclerotic mechanisms, which predisposes the risk of cardiovascular disease [14]. The clinical group of the current study comprised patients with hypertension and metabolic syndrome that showed an increase in uric acid levels in comparison to healthy people. This fact is substantiated by the evidence that uric acid can be a contributory factor to the pathogenesis of hypertension by acting through the mechanisms of renal vasoconstriction and reninangiotensin system activation [15].

Further, the present results of increased uric acid levels in the patients in the hypertensive and metabolic disorder groups are agreeable to various recent clinical researches. A meta-analysis study on hypertensive patients revealed that high levels of serum uric acid were correlated with the increased risk of cardiovascular events and death [16]. On the same note, observational studies have proposed that hyperuricemia can be used as a predictor of cardiovascular risk in the general population, even before the acquisition of cardiovascular disease [17]. Further cohort studies have further indicated that hyperuricemic patients are more susceptible to cardiovascular events after coronary interventions, which can possibly indicate that uric acid can lead to disease progression and complications [18].

The other valuable observation of the current research was the correlation between serum uric acid levels and renal function measures like creatinine. Our results show a positive correlation between uric acid and creatinine, which is in agreement with previous studies that portray uric acid level to be positively associated with renal impairment and diabetic nephropathy. To illustrate, a clinical trial study of patients with type 2 diabetes mellitus showed independent relationships between serum uric acid levels and diabetic nephropathy and no relationships between serum uric acid levels and diabetic retinopathy [19]. This correlation can be attributed to the fact that uric acid is excreted mainly via the kidneys and in case of broken kidney functions, the uric acid can accumulate in bloodstream.

Moreover, it is suggested by the emerging evidence that hyperuricemia can play a role in cardiovascular and metabolic complications via complicated biochemical mechanisms involving oxidative stress and inflammatory reactions. Other studies indicate that metabolic imbalances such as the development of advanced glycation end-products (AGEs) can play off with the metabolism of uric acid and affect the structure and the functioning of the heart [20]. Moreover, some of these reviews have emphasized that prolonged hyperuricemia can cause vascular inflammation and endothelial dysfunction and contribute to the development of hypertension and cardiovascular diseases [21][22][23][24].

Altogether, the results of the current research are in line with an increasing amount of scientific data that suggest that serum uric acid is strongly correlated with metabolic dysfunctions, cardiovascular risk factors, and renal dysfunction. The identified correlations between the levels of uric acid, BMI, lipid profile items, and metabolic disorders prove the idea that hyperuricemia can be not only a metabolic product but also a possible biomarker of underlying cardiometabolic malfunction. The findings enlighten the necessity of serum uric acid levels measurement in terms of regular biochemical analysis of patients at risk of metabolic and cardiovascular illnesses.

5. Conclusion

The current research examined the clinical and biochemical implications of serum uric acid concentration on human health and disease with the specific focus on metabolic and cardiovascular risk factors. The results have shown that the levels of serum uric acid in patients with metabolic and cardiovascular conditions were significantly more than in seemingly healthy individuals, which has proved that hyperuricemia is closely related to metabolic disturbances. The prevalence of hyperuricemia was also found to be 31 per cent in the study population of 200 people with most of them in the clinical group. This distribution addresses the possible significance of uric acid as a predictor of some underlying metabolic abnormalities.

Moreover, the outcomes showed that there were significant correlations between serum uric acid levels and a number of significant clinical and biochemical parameters. Higher levels of uric acid had a positive correlation with body mass index, triglyceride levels, and serum creatinine, which meant that the higher the level of uric acid is, the more evidence it demonstrates metabolic imbalance and renal dysfunction. These results can be justified by the accumulating body of evidence that uric acid is not only a product of metabolism but also a biomarker that is closely associated with cardiometabolic health. Moreover, statistical tests showed that the variables BMI, triglyceride levels and creatinine were independent predictors of high uric acid levels, which underscores the multifactorial character of hyperuricemia.

The research also indicated that serum uric acid could have a clinical usefulness as a biochemical predictor of people at risk of developing metabolic and cardiovascular complications. The moderate diagnostic ability of the receiver operating characteristic (ROC) analysis indicates that uric acid measurements can be used in early detection interventions and risk evaluation in clinical care. Urine acid monitoring especially in patients with metabolic syndrome, hypertension, or diabetes may thus have some valuable information in preventive care and disease control.

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