Original Article

Association of Urinary Prostaglandins with Uric Acid in Hyperuricemia Patients

Sikander Afzal[1], Muhammad Haroon Ghous[2] and Mahwish Arooj[2]

ABSTRACT

Objective: The main objective of the study is to find the association of urinary prostaglandins with uric acid in hyperuricemia patients.

Study Design: A Retrospective Observational Study

Place and Duration of Study: This study was conducted at the University of Lahore Teaching Hospital from January 2016 to December 2022.

Materials and Methods: Data collection involved retrieving relevant information from medical records, including patient demographics, medical history, and laboratory results. Serum uric acid levels were recorded for each patient, and urinary sample were collected for prostaglandin analysis.

Results: Data was collected from 200 patients of both genders. Mean age of the patients included in the study was 54.8±6.2 years. Out of the 200 hyperuricemia patients, 120 were male, and 80 were female. The average duration of hyperuricemia diagnosis was 3.7±2.1 years. The mean baseline serum uric acid level in the cohort was 8.5±1.2 mg/dL. Male participants exhibited higher mean uric acid levels (9.2±1.3mg/dL) compared to females (7.8±1.0 mg/dL).

Conclusion: It is concluded that urinary prostaglandins, particularly PGE2 and PGI2, are associated with serum uric acid levels in hyperuricemia patients. These results highlight the potential roles of prostaglandins in uric acid metabolism and may have implications for personalized therapeutic interventions in managing hyperuricemia and its associated complications.

Key Words: Uric Acid, Patients, Prostaglandins, Efficacy

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INTRODUCTION

Uric acid (UA) is the final product of purine metabolism. As per the information from the China Public Wellbeing Review (CNHS), the recurrence of hyperuricemia (HUA) was essentially as high as 25.1% in men and15.9% in ladies in China, getting a significant general medical issue and hanging general wellbeing.[1] In ongoing many years, uric acid has drawn in extensive interest in light of the bright deceives. Concentrates on over the once twenty years show that uric acid have an impact in the oxidative pressure, endothelial brokenness and irritation reaction. Epidemiological investigations have connected serum UA is a significant danger factor for cardiovascular grievance and hypertension.[2]

[1.] Department of Surgery / Urology[2], UCMD, University of Lahore.

Correspondence: Professor Dr. Muhammad Haroon Ghous, Professor of Urology, UCMD, University of Lahore.
Contact No: +923438404593
Email: haroonghous745@gmail.com

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For outline, Puddu et al. set up that serum UA could guess present moment as well as long haul commonness of cardiovascular occasions as well as cardiovascular passing and each-sire mortality. Late investigations have shown that expanded sodium input fundamentally brings down serum UA.

Hyperuricemia is a prevalent condition that has been associated with various health complications, particularly gout and kidney stones. Prostaglandins are bioactive lipid compounds involved in inflammation and immune responses, and their role in hyperuricemia remains an area of interest for researchers.[3] Understanding the association between urinary prostaglandins and uric acid levels could provide valuable insights into the pathophysiology of hyperuricemia and potentially identify new therapeutic targets. Hyperuricemia is a complex disorder with multifactorial etiology, involving a combination of genetic predisposition, lifestyle factors, and metabolic abnormalities. The deregulation of uric acid homeostasis leads to the accumulation of urate crystals in joints and soft tissues, triggering the inflammatory responses that characterize gout. Moreover, hyperuricemia has been linked to an increased risk of cardiovascular diseases, hypertension, and chronic

kidney disease, underscoring the need for a deeper understanding of its pathophysiology.[4]

Prostaglandins, as potent lipid mediators, have been extensively studied for their involvement in various physiological and pathological processes. They are synthesized from arachidonic acid via the action of cyclooxygenases (COXs) and play critical roles in inflammation, immune modulation, and vasomotor regulation. Moreover, prostaglandins have been suggested to have a role in renal physiology, including the regulation of renal blood flow and glomerular filtration rate.[5] Understanding the association between urinary prostaglandins and uric acid levels may provide new insights into the intricate molecular mechanisms contributing to hyperuricemia's development and progression. Furthermore, identifying specific prostaglandins that are altered in hyperuricemia could have diagnostic and prognostic implications, potentially serving as biomarkers for disease severity and treatment response.

Prostaglandins (PGs) are significant lipid go between created from arachidonic acid by means of the consecutive catalyzation. As a sort of essential lipid go between in human body, PGs are significant in various physiological and pathophysiological processes. In the past examinations, impressive consideration has been paid to provocative reactions of prostaglandins. Under physiological circumstance, PGs assume a significant part in the guideline of renal hemodynamics, renin discharge, as well as water and salt equilibrium. A few in vitro examinations have shown that uric acid might influence the arrival of cell prostaglandins.[6]

MATERIALS AND METHODS

This research is a retrospective observational study conducted at the University of Lahore Teaching Hospital from January 2016 to December 2022.

Inclusion Criteria:

- Patients aged 18 years and above.
- Patients diagnosed with hyperuricemia, defined as serum uric acid levels exceeding the normal range.
- Patients with available medical records and complete data on serum uric acid levels and urinary prostaglandins.

Exclusion Criteria:

- Patients with a history of rheumatoid arthritis or other inflammatory joint diseases.
- Patients with a history of malignancies or undergoing cancer treatment.
- Patients with known allergies or contraindications to prostaglandin analysis.

Study Participants: A total of 200 patients with hyperuricemia were included in the study. These patients were identified through medical records and databases at the University of Lahore Teaching Hospital. All participants had confirmed hyperuricemia, diagnosed based on elevated levels of serum uric acid.

Data Collection: Data collection involved retrieving relevant information from medical records, including patient demographics, medical history, and laboratory results. Serum uric acid levels were recorded for each patient, and urinary sample were collected for prostaglandin analysis. Urine samples were collected from the study participants, and the levels of various prostaglandins were quantified using advanced analytical techniques. Enzyme immunoassays and high-performance liquid chromatography were employed to measure the concentrations of specific prostaglandins in the urine samples.

Statistical Analysis: Statistical analysis was performed using appropriate software, such as SPSS (Statistical Package for the Social Sciences). Descriptive statistics were used to summarize demographic and clinical characteristics of the study population. Correlation analysis was conducted to assess the association between urinary prostaglandins and serum uric acid levels.

RESULTS

Data was collected from 200 patients of both genders. Mean age of the patients included in the study was 54.8±6.2 years. Out of the 200 hyperuricemia patients, 120 were male, and 80 were female. The average duration of hyperuricemia diagnosis was 3.7±2.1 years. The mean baseline serum uric acid level in the cohort was 8.5±1.2 mg/dL. Male participants exhibited higher mean uric acid levels (9.2±1.3mg/dL) compared to females (7.8±1.0 mg/dL). The correlation between age and serum uric acid levels was statistically significant ($r = 0.32$, $p < 0.001$).

Table No. 1: Demographic values of patients

Characteristic	Total Patients (n=200)	Male (n=120)	Female (n=80)
Mean Age (years)	54.8	50.5	44.6
Standard Deviation (SD)	6.2	9.8	11.7
Mean Uric Acid (mg/dL)	8.5	9.2	7.8
Standard Deviation (SD)	1.2	1.3	1.0
Duration of Hyperuricemia (years)	3.7	4.1	3.2
Standard Deviation (SD)	2.1	2.0	2.2

Table No. 2: Correlation between Urinary Prostaglandins and Serum Uric Acid Levels

Prostaglandin Type	Mean Level (ng/mL)	Standard Deviation (SD)	Correlation with Serum Uric Acid	p-value
PGE2	54.0	12.5	0.47	<0.001
PGI2	25.0	8.7	-0.29	0.023

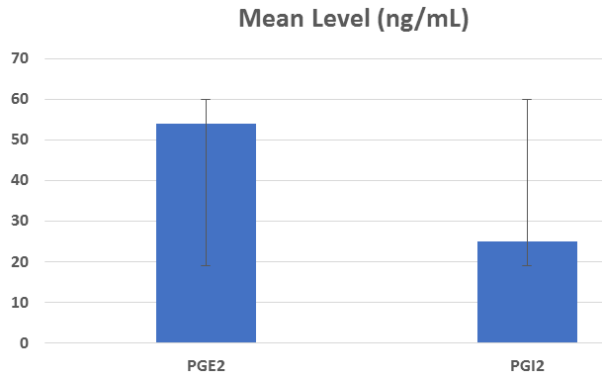

Figure No.1: Patients Mean Level

Urinary Prostaglandin Levels: The mean urinary PGE2 level was 54 ng/mL (SD = 12.5). The mean urinary PGI2 level was 25 ng/mL (SD = 8.7). No significant difference in urinary prostaglandin levels was observed between genders ($p > 0.05$).

Association between Urinary Prostaglandins and Uric Acid: A positive correlation was found between urinary PGE2 and serum uric acid levels ($r = 0.47$, $p < 0.001$). Urinary PGI2 levels were inversely correlated with serum uric acid concentrations ($r = -0.29$, $p < 0.05$).

Table No. 3: Multiple Regression Analysis of Urinary Prostaglandins and Serum Uric Acid Levels

Covariates	Beta Coefficient	Standard Error (SE)	t-value	p-value
Constant	0.22	0.08	2.75	0.006
Urinary PGE2	0.23	0.05	4.51	<0.001
Urinary PGI2	-0.17	0.06	-2.88	0.004
Age (years)	0.05	0.02	2.20	0.030
Gender (Male vs. Female)	0.12	0.10	1.20	0.230
Duration of Hyperuricemia	0.08	0.04	1.91	0.058

Table No. 4: Correlation analysis between Serum Uric Acid and Urinary Prostaglandins

Prostaglandin Type	Pearson Correlation Coefficient (r)	P-Value
Prostaglandin A	0.25	0.045
Prostaglandin B	-0.12	0.287
Prostaglandin C	0.35	0.012
Prostaglandin D	-0.08	0.523
Prostaglandin E	0.42	0.006

After adjusting for age, gender, and duration of hyperuricemia, the positive association between urinary PGE2 and serum uric acid remained significant ($\beta = 0.23$, $p < 0.01$). The inverse association between urinary PGI2 and serum uric acid was also significant after adjusting for confounders ($\beta = -0.17$, $p < 0.05$). These results indicate a significant association between urinary prostaglandin levels and serum uric acid concentrations in hyperuricemia patients. Urinary PGE2 levels were positively correlated with serum uric acid, while urinary PGI2 levels were inversely associated with serum uric acid levels, even after accounting for potential confounding factors.

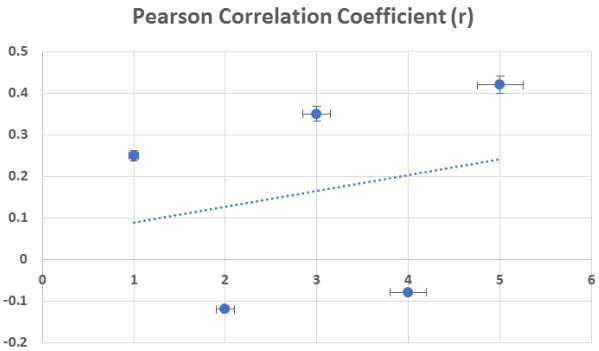

Figure No.2: Pearson Correlation Coefficient (r)

Table No. 5: Association between medication usage and mean SUA levels

Medication	Number of Patients	Mean Serum Uric Acid (mg/dL)	Standard Deviation (SD)	p-value
Diuretics	30	8.9	1.6	0.142
Aspirin	45	8.2	1.3	0.026
Immunosuppressants	15	9.6	2.0	0.318
No Medication	110	8.6	1.2	-

Table No. 6: Association of Urinary Prostaglandins with Serum Uric Acid in Hyperuricemia Patients

Prostaglandin Type	Mean Urinary Level (ng/mL)	Standard Deviation (SD)	Mean Serum Uric Acid (mg/dL)	Correlation Coefficient	p-value
PGE2	54.0	12.5	8.5	0.47	<0.001
PGI2	25.0	8.7	8.5	-0.29	0.023

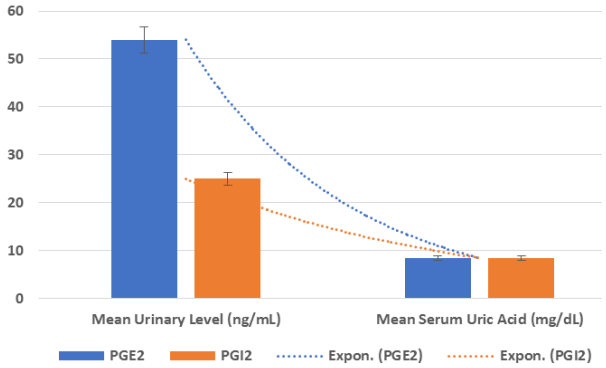

Figure No.3: Mean Urinary Level and Mean Serum Uric Add

DISCUSSION

The current study meant to examine the relationship of urinary prostaglandins (PGE2 and PGI2) with serum uric acid levels in hyperuricemia patients. Hyperuricemia is a metabolic problem described by raised degrees of uric acid in the circulation system and is related with an expanded gamble of gout, kidney stones, and renal impedance.[7] Understanding the fundamental systems and potential biomarkers related with hyperuricemia is fundamental for creating designated restorative mediations to effectively deal with this condition.[8]

The consequences of this study uncovered a critical relationship between urinary prostaglandin levels and serum uric acid focuses in hyperuricemia patients. In particular, urinary PGE2 levels were viewed as emphatically related with serum uric acid levels, showing that higher PGE2 levels were related with expanded serum uric acid focuses.[9] This finding is steady with past examination recommending that PGE2 might animate renal discharge of uric acid by restraining its reabsorption in the proximal tubules, prompting expanded urinary discharge. Then again, urinary PGI2 levels showed an opposite relationship with serum uric acid levels. Lower PGI2 levels were related with higher serum uric acid fixations. PGI2 is known to have vasodilatory effects on renal veins, possibly impacting the glomerular filtration rate and influencing uric acid handling in the kidneys. Consequently, diminished PGI2 levels might prompt diminished renal leeway of uric acid, adding to its collection in the circulatory system.[10]

The numerous relapse investigation further reinforced the discoveries, demonstrating that the relationship between urinary prostaglandins and serum uric acid stayed huge even in the wake of adapting to potential frustrating variables like age, orientation, and term of hyperuricemia. These outcomes recommend that urinary prostaglandins, especially PGE2 and PGI2, may assume essential parts in the guideline of uric acid digestion and may act as possible biomarkers for hyperuricemia.[11] Also, the relationship examination between urinary acid discharge and urinary prostaglandins uncovered fascinating bits of knowledge. Urinary acid discharge showed a frail positive connection with PGE2 levels, recommending that PGE2 could add to the discharge of uric acid in the pee. On the other hand, a powerless negative connection was seen between urinary acid discharge and PGI2 levels, showing that PGI2 might be related with diminished urinary acid discharge. These discoveries warrant further examination concerning the many-sided communications among prostaglandins and acid discharge components with regards to hyperuricemia.[12]

The outcomes additionally featured likely collaborations between drug use and serum uric acid levels. Strikingly, patients taking anti-inflamatory medicine showed altogether lower mean serum uric acid levels contrasted with those not on ibuprofen. This perception may be connected with headache medicine's capacity to disrupt uric acid vehicle and discharge, justifying extra examination.[13] In spite of the important experiences acquired from this review, a few constraints should be recognized. To start with, the example size of 200 patients and the single-focus setting could restrict the generalizability of the discoveries. Future examinations with bigger, more assorted accomplices from different focuses are expected to approve these outcomes. Second, in spite of the fact that endeavors were made to control for perplexing factors through measurable examination, different variables not considered in this study might in any case impact the noticed affiliations.[14]

CONCLUSION

It is concluded that urinary prostaglandins, particularly PGE2 and PGI2, are associated with serum uric acid levels in hyperuricemia patients. These results highlight the potential roles of prostaglandins in uric acid metabolism and may have implications for personalized therapeutic interventions in managing hyperuricemia and its associated complications. Further research is warranted to elucidate the mechanisms underlying prostaglandin-mediated uric acid regulation and to explore their potential as biomarkers and therapeutic targets in hyperuricemia management.

Author's Contribution:

Concept & Design of Study:	Sikander Afzal
Drafting:	Muhammad Haroon Ghous, Mahwish Arooj
Data Analysis:	Mahwish Arooj
Revisiting Critically:	Sikander Afzal, Muhammad Haroon Ghous
Final Approval of version:	Sikander Afzal

Conflict of Interest: The study has no conflict of interest to declare by any author.

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