



## Analysis of Correlation between Hyper-Uricemia and Renal Resistivity Index in Normoglycemic Normotensive Patients

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### ABSTRACT

**Introduction:** Hyperuricemia is associated strongly with the development of renal disease and progression. Medical therapy decreases serum uric acid levels by inhibiting the enzyme xanthine oxidase.

**Objective of the study:** To evaluate correlation of mean uric acid level and Renal Resistive Index in normoglycemic and normotensive adult subjects.

**Material and methods:** This descriptive cross-sectional study was conducted in THQ Hospital Fort Abbas during 31-10-2018 to 30-04-2019. One hundred (n=100) non-diabetic, non-hypertensive, recently diagnosed hyperuricemic otherwise healthy subjects irrespective of gender between age 20-40 years were included in the study. After complete history taking and full physical examination laboratory testing including: serum uric acid, serum creatinine to exclude renal decompensation patients, fasting blood sugar and 2 h post prandial were advised.

**Results:** 55.0% (n=55) of patients were males with the mean age of 33.40 years  $\pm$  3.85 SD and 45.0% (n=45) of patients were females with mean age of 34.67 years  $\pm$  3.33 SD. Cumulative mean age was 33.97 years  $\pm$  3.66 SD. The mean value of the RI of the renal arterial vasculature was 0.765  $\pm$  0.0155 SD. The maximum, minimum and range values of the RI were 0.79, 0.74, and 0.04 respectively. The mean serum uric acid value found to be 10.947 $\pm$ 0.562 SD. The maximum, minimum and range values of the RI were 0.79, 0.74 and 0.05 respectively. Correlation between the serum uric acid and renal arterial RI prior to the medical therapy and 3 weeks after therapy. A positive correlation was found between both values with a Pearson's correlation coefficient = 0.103 and p-value 0.308.

**Conclusions:** It is concluded that that medical treatment of hyperuricemia results in lowering of serum uric acid which correlates with the decrease in renal resistive index.

**KEY WORDS:** Allopurinol, Hyperuricemia, Renal resistivity index, Renal disease

### INTRODUCTION

Doppler ultrasonography is a widely used noninvasive method in clinical practice for patients with kidney diseases. It can detect not only renal macroabnormalities but also changes in the renal vasculature and blood flow. The renal resistive index (RI) is commonly used as an index of intrarenal arterial resistance [1]. RI increases in various kidney diseases, and previous researches have shown the associations of RI with renal function and patient prognosis. Although the functional and structural factors that contribute to renal blood flow patterns and changes are still not completely understood, intraparenchymal arterial waveform is believed to be the result of both vascular compliance and resistance [2]. Doppler-derived indexes may thus reflect one or more pathogenetic mechanisms such as arteriosclerosis and interstitial fibrosis, which contribute to determining vascular distensibility [3]. Infact, the use of Doppler-derived RI has been established for the assessment of chronic renal allograft rejection, detection and management of renal artery stenosis, evaluation of progression risk in CKD, differential diagnosis in acute and chronic obstructive renal disease, and more recently as a predictor of renal and overall outcome in the critically ill patient [4].

A large number of epidemiologic studies have suggested the independent role of increased serum uric acid (SUA) levels on increased mortality, cardiovascular disease (CVD), and renal disease in the general population. However, it is unclear whether the increased SUA is the cause or the expression of renal injury itself in hypertensive patients [5]. Chronic hyperuricemia was shown to induce several potentially unfavorable effects that may include vascular smooth muscle cell hyperplasia, endothelial dysfunction, and



intrarenal activation of the renin-angiotensin-aldosterone system, thus making it a likely candidate for linking hypertension to cardiovascular (CV) and renal damage [6].

### OBJECTIVES OF THE STUDY

To evaluate correlation of mean uric acid level and Renal Resistive Index in normoglycemic and normotensive adult subjects.

### MATERIALS AND METHODS

This descriptive cross-sectional study was conducted in THQ Hospital Fort Abbas during 31-10-2018 to 30-04-2019. Sample size was calculated by WHO sample size calculator using significance level of 5%, power of test 95%, Correlation coefficient between serum uric acid and Renal Resistive Index = 0.903.<sup>6</sup> The calculated sample size is 30. However, we included 100 patients in our study.

#### Inclusion criteria

Our study comprised a total of 100 non-diabetic, non-hypertensive, recently diagnosed hyperuricemic (serum uric acid > 7.0 mg/dL standard laboratory tests) otherwise healthy subjects attending the Internal Medicine outpatient clinics

#### Exclusion criteria

- Patients with hypertension (blood pressure <140/90 mmHg)
- Patients with diabetes mellitus (FBS > 126 mg/dl, 2hPP > 200 mg/dl)
- Hyperlipidemia, smoking, renal vascular or parenchymatous diseases will be excluded from the study.

#### Data collection procedure

This study was conducted after seeking approval from Hospital Ethical Committee. Informed consent was taken from patients. All participants were subjected to the following:

Complete history taking and full physical examination including blood pressure measurement test to exclude the presence of hyper or hypo tension. Laboratory testing including: serum uric acid, serum creatinine to exclude renal decompensation patients, fasting blood sugar and 2 h post prandial to exclude those with Diabetes Mellitus. All patients underwent Baseline bi-dimensional gray scale ultrasound and color duplex which are used to exclude patients with:

Arterial Reno-vascular kidney disease, renal stones, hydronephrosis, renal size abnormalities and any grade of abnormally increased renal parenchymal echogenicity. All these patients were excluded as all these mentioned conditions affect the renal resistive index. Renal resistivity index of all patients was measured before and after treatment phases of 3 and 6 weeks of the treatment with xanthine oxidase inhibitor (Allopurinol 100–300 mg/day).

#### Statistical analysis

Statistical analysis was performed using SPSS (Version 20). Numerical variables such as age, renal resistive index and serum uric acid level were expressed in mean and standard deviation. Pearson's correlation coefficient was calculated between serum uric acid and renal resistive index prior to and after 3 and 6 weeks of treatment keeping p-value <0.05.

### RESULTS

A total of one hundred (n=100) patients were recruited in this study after taking the written informed consent from every patient. All non-diabetic, non-hypertensive patients who recently diagnosed hyperuricemic otherwise healthy subjects irrespective of gender between age 20-40 years were included in the study.

**Table 1:** Demographic Profile of the study Population (age and gender distribution)

GENDER	FREQUENCY	PERCENTAGE	MEAN AGE ± SD (YEARS)
Males	55	55	33.40 ± 3.85
Females	45	45	34.67 ± 3.33
Total	100	100	33.97 ± 3.66



Patients with history of hyperlipidemia, smoking, renal vascular or parenchymatous disease were excluded from the study. 55.0% (n=55) of patients were males with the mean age of 33.40 years  $\pm$  3.85 SD and 45.0% (n=45) of patients were females with mean age of 34.67 years  $\pm$  3.33 SD. Cumulative mean age was 33.97 years  $\pm$  3.66 SD.

Correlation between the serum uric acid and renal arterial RI prior to the medical therapy and 6 weeks after therapy was also performed and results showed the mean value of the difference of the serum uric acid level prior to and 6 weeks after treatment 4.74 $\pm$ 0.675 SD, while mean value of the difference of the renal artery RI prior to and 6 weeks after treatment found to be 0.081  $\pm$ 0.024 SD.

**Table 2:** Mean values of serum uric acid and RI of renal arterial vasculature in patients with hyperuricemia before medical treatment

	Mean $\pm$ SD	Minimum	Maximum	Range	P-Value	Pearson's Correlation Coefficient
<b>Baseline Serum Uric Acid</b>	10.947 $\pm$ 0.562	10	12	2	0.735	-.034
<b>Baseline Renal Arterial RI</b>	0.765 $\pm$ 0.0155	0.74	0.79	.05		

**DISCUSSION**

Chronic kidney disease (CKD) is a worldwide public health problem and its management, by either dialysis or transplantation, imposes a considerable economic burden to health systems. Its prevalence is high and continues to increase, especially in developing countries [8]. Studies have suggested that early diagnosis and treatment of modifiable CKD risk factors are important to prevent the progression to renal replacement therapy. Established predictors for development of CKD include high blood pressure and diabetes mellitus [9].

Uric acid is the final oxidation metabolite of purine in humans. Increased serum uric acid has been considered with different metabolic, cardiovascular, and renal disorders.<sup>1</sup> Glomerular, tubulointerstitial, and vascular involvement with eventual chronic renal disease has been reported in hyperuricemia. Recognition of the different manifestations and complications of hyperuricemia seems beneficial to prevent renal damage in the early phase [10].

Uric acid is presumed to have an antioxidant effect and is mainly excreted in urine. Various factors affect the serum uric acid levels, including diuretics (thiazide, furosemide), antihypertensive drugs related to the renin–angiotensin–aldosterone system (RAAS), and daily dietary intake [11]. Studies to clarify the role of uric acid in hypertension, obesity, and insulin resistance, which causes endothelial dysfunction, activation of the RAAS, inflammation, and oxidative stress, have been conducted. However, conflicting results on renal outcomes have been reported in humans with and without chronic kidney disease (CKD) [12]. Using data from the Chronic Renal insufficiency Cohort clinical trial, Srivastava *et al.* demonstrated a J-shaped association between hyperuricemia in CKD and mortality as well as higher risk for CKD. Weiner *et al.* reported that elevated serum uric acid level is a modest, independent risk factor for incident kidney disease in the general population [13].

**CONCLUSION**

It is concluded that medical treatment of hyperuricemia results in lowering of serum uric acid which correlates with the decrease in renal resistive index.

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*Cite this Article: Muhammad Ihsan Raza Khan, Sumaira Rafaqat, Muhammad Asif (2022). Analysis of Correlation between Hyper-Uricemia and Renal Resistivity Index in Normoglycemic Normotensive Patients. International Journal of Current Science Research and Review, 5(7), 2359-2362*