https://doi.org/10.29309/TPMJ/2025.32.03.8800

The Professional Medical Journal
www.theprofesional.com
2025, Volume, 32 Issue, 03

ORIGINAL ARTICLE

Role of Losartan in decreasing uric acid levels at JPMC.

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Article Citation: Khalid T, Sultan MO, Ghayas A, Khan MI, Abdul Sattar R, Parwasha. Role of Losartan in decreasing uric acid levels at JPMC. Professional Med J 2025; 32(03):337-342. https://doi.org/10.29309/TPMJ/2025.32.03.8800

ABSTRACT… Objective: To determine the mean change in uric acid level after use of losartan in hypertensive patients at tertiary care hospital. **Study Design:** Quasi Experimental study. **Setting:** Department of Medicine at JPMC. **Period:** 18/01/2024 to 17/07/2024. **Methods:** 100 eligible patients were selected written informed consent was given by each patient. The baseline assessments included demographics, medical history, and serum uric acid levels. At four, eight, and twelve weeks following therapy, patients were observed to assess changes in uric acid levels, medication adherence, and adverse events. Data was gathered using a prepared proforma, ensuring comprehensive documentation for each study component. **Results:** Participants in the study were mostly between the ages of 41 and 80 (91%), with a mean age of 57.89 years (SD = 11.15**)**. Measurements included height (173.42 cm ± 9.32), weight (76.99 kg ± 9.51), BMI (25.83 ± 2.21), duration of hypertension (26.00 months ± 11.80), and serum uric acid levels at baseline (7.38 mg/dL ± 0.78), 4 weeks (6.91 mg/dL ± 0.52), 8 weeks (6.70 mg/dL ± 0.52), and 12 weeks (6.09 mg/dL ± 0.25). 55% of the population was male and 45% was female, and 63% of them lived in urban and 37% in rural areas. In terms of employment status, 62% were employed while 38% were unemployed, and 38% reported having diabetes. Serum uric acid levels significantly decreased with losartan, going from 7.38 mg/dL (SD = 0.78) at the beginning to 6.09 mg/dL (SD = 0.25) after 12 weeks (p = 0.001), indicating that hypertension-related hyperuricemia was effectively controlled. **Conclusion:** This result highlights losartan's potential usefulness in treating hyperuricemia linked to hypertension by indicating that it is effective in lowering serum uric acid levels during the course of treatment.

Key words: Hypertension Treatment, Losartan, Uric Acid Levels.

INTRODUCTION

The global prevalence of non-communicable diseases is largely caused by cardiovascular disease, chronic renal disease, and stroke, all of which are significantly impacted by hypertension.[1] By 2025, 29% of adults worldwide are expected to have hypertension, up from the current 25%.[2] By the year 2025, the figure will rise to 22.9 for men in India and 23.6 for women in India, respectively.[3] A systematic analysis of population-based studies from 90 countries revealed that the rate of hypertension was 31.5% in low- and middle-income countries (LMIC), while it stood at 28.5% in high-income countries.[4]

Hypertension significantly affects cardiovascular health and medical facilities in Pakistan.[5] Up to this point, it has proven to be challenging to keep hypertension patients undergoing treatment at normal systolic and diastolic blood pressure levels. To obtain their target blood pressure, many hypertension patients need to take at least two antihypertensive drugs.[6] The high pill load in these people may lead to non-adherence to medication use, making it more difficult to control blood pressure and increasing the risk of cardiovascular disease.[7]

Highly selective antagonists of the angiotensin II type-1 receptor known as angiotensin receptor blockers are effective for treating hypertension, especially when paired with diuretics. SUA concentrations and the risk of gout are shown to be reduced by losartan, an angiotensin II receptor blocker, and calcium channel blockers.[8,9]

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Article received on: 08/10/2024
Date of revision: 10/12/2024
Accepted for publication: 11/12/2024

Calcium channel blockers and thiazide diuretics are the primary treatments for stage 1 hypertension. For individuals with a 10% or higher risk of atherosclerotic cardiovascular disease, combination treatment is employed to assist them in achieving their blood pressure targets. Angiotensin II receptor blockers are effective as a standalone treatment when comorbidities such as diabetes, ischemic heart disease, cerebrovascular disease, heart failure, and chronic renal disease are absent.

The transporters URATI and GLUT 9 found in the proximal tubule of the kidney have an essential function in the movement of uric acid, and losartan is one of these transporters.[10] Losartan has the ability to lower uric acid levels as a result, which lowers cardiovascular and renal morbidity and mortality. It has been shown that losartan monotherapy can reduce SUA levels by up to 25%.[11]

A previous study by reported Baseline The serum uric acid level (SUA) measured at 6.7 mg/dL for both placebo and losartan-treated participants. In the initial 6 months, losartan reduced SUA by −0.16 mg/dL.[12]

Another study by reported Serum uric acid levels were reduced by 50 mg of losartan once day, going from 538 ± 26 to 491 ± 20 micromol/l.[13]

Rationale: Several studies have suggested a connection between hypertension and hyperuricemia. Hypertension often coexists with high uric acid levels, and addressing both conditions simultaneously could lead to improved patient outcomes. Losartan, as an antihypertensive medication, may have an additional benefit of lowering uric acid levels, which warrants further investigation. Losartan has shown cardiovascular beneficial properties, and its main use is the treatment of hypertension. Losartan may help lower cardiovascular risk if it is shown to lower uric acid levels and lower the likelihood of gout. The investigation of this dual advantage may have important effects on public health.

OBJECTIVE

To determine the mean change in uric acid level after use of losartan in hypertensive at tertiary care hospital

METHODS

A quasi experimental was carried out in department of, Karachi. It was conducted from 18th January 20224 till 17th July 2024. Prior ethical approval was sought from review board (F.2-8/2023-GENL/222/JPMC on 08.01.24). Sample size was calculated using WHO calculator. Sample size of 62 is estimated with an 95% CI, a 5% level of significance, and taking mean uric acid level after losartan 491 ± 20 micromol/l.13[13] We were taken 100 patients.

Non- probability consecutive patients, 18 to 80 years old, of either gender with diagnosis of hypertension, hyperuricemia, gout, or a combination of these conditions, currently prescribed losartan for the management of hypertension, baseline uric acid levels within the hyperuricemic range, typically taken as serum uric acid levels exceeding 6.8 mg/dL (404 μmol/L) and provide informed consent to participate in the study.

EXCLUSION CRITERIA

Pregnant or breastfeeding individuals due to potential risks associated with medication exposure during pregnancy and lactation, Known hypersensitivity or intolerance to losartan or related medications, Individuals with significant kidney dysfunction (eGFR < 30 mL/min/1.73 m^2) due to potential safety concerns and altered drug metabolism, Individuals currently taking medications specifically designed to lower uric acid levels (e.g., allopurinol, probenecid) other than losartan and Participants with uncontrolled hypertension requiring immediate intervention or hospitalization.

A total number of 100 patients fulfilling the inclusion criteria were selected. Before including any individuals in the study, we required their written informed consent. All participants were informed of the study's goals and eligibility conditions in accordance with the standards.

Baseline assessment including medical history, demographics (age, gender, residence, occupation, BMI, duration of hypertension and uric acid level were measured.

Postoperatively Patients were examined at 4th weeks, 8th weeks and final at 12th weeks. At each follow up visits change in uric acid levels, medication adherence and adverse event were nosted.
Various demographic parameters as age, gender, residence, occupation, BMI, duration of hypertension and uric acid level were measured were collected on predesigned proforma. Change in uric acid level was measured from the baseline after 12 weeks standardized laboratory methods for uric acid measurements to ensure accuracy.

All data was entered and analyzed in SPSS version 25. Qualitative data such as gender, gender, residence, occupation ws presented as frequency and percentage. Quantitative data like age, BMI, Duration of hypertension and uric acid level at baseline, 4th weeks, 8th weeks and 12th weeks as mean ± S.D. Paired t test was applied to utilized the change in uric acid level from the baseline. P-value ≤ 0.05 was used as significant. Data was stratified with age, gender, residence, occupation, BMI and duration of hypertension. A post-stratification paired t-test was utilized. A p-value of ≤ 0.05 were regarded as significant.

RESULTS

A total of 100 patients were included in this study fulfilling the inclusion criteria. Among these age distribution shows that the majority of participants (91%) are between 41 and 80 years old, with only 9% aged 18 to 40 years. The mean age is 57.89 ± 11.15 years. (Table-I)

The study findings reveal that the mean height (173.42 cm ± 9.32), weight (76.99 kg ± 9.51), BMI (25.83 ± 2.21), duration of hypertension (26.00 months ± 11.80). (Table-I)

Serum uric acid levels at baseline (7.38 mg/dL ± 0.78), at 4 weeks (6.91 mg/dL ± 0.52), at 8 weeks (6.70 mg/dL ± 0.52), and 12 weeks (6.09 mg/dL ± 0.25). (Figure-1)

Gender distribution was 55% male and 45% female. Regarding residence, 63% of participants lived in urban areas, with the remaining 37% residing in rural areas. Employment status showed that 62% were employed, while 38% were unemployed. 38% of participants had diabetes, while 62% did not. (Table-I)

The study aimed to investigate the mean change in serum uric acid levels after using losartan in hypertensive patients. Results indicated a significant reduction from a baseline mean of 7.38 mg/dL (SD = 0.78) to 6.09 mg/dL (SD = 0.25) at 12 weeks (p = 0.001). This suggests that losartan effectively lowers serum uric acid levels over the treatment period. Table-II

Results showed significant reductions in uric acid levels across all groups: younger participants (18-40 years) decreased from 7.38 mg/dL (SD = 0.89) to 6.05 mg/dL (SD = 0.26) at 12 weeks, while older participants (41-60 years) started at 7.38 mg/dL (SD = 0.77) and decreased to 6.10 mg/dL (SD = 0.25). Both males and females experienced similar reductions, with urban and rural residents also showing significant decreases. Participants with higher BMI and longer duration of hypertension exhibited consistent decreases in serum uric acid levels after treatment with losartan (Table-III).

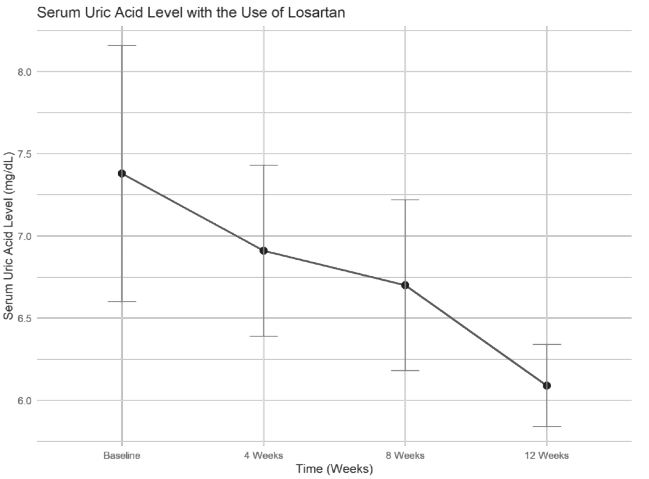

Figure-1. Losartan level at baseline, 4 weeks, 8 weeks & 12 weeks.

Variables		Frequency	Percent
Age	18 - 40 years	9	9.0%
	41 - 80 years	91	91.0%
Gender	Male	55	55.0
	Female	45	45.0
Residence	Urban	63	63.0
	Rural	37	37.0
Employment status	Employment	62	62.0
	Unemployed	38	38.0
Diabetes	Yes	38	38.0
	No	62	62.0
	Total	100	100.0%
		Mean	**SD**
Height		173.42	9.32
Weight		76.99	9.51
BMI		25.83	2.21
Duration of hypertension (months)		26.00	11.80

Table-I. Distribution of Age, gender, residence, employment, diabetes, height, weight, BMI, duration of hypertension and SUA. (n=100)

Change in Uric Acid	Mean	Standard Deviation	P-Value
Serum uric acid at baseline (mg/dL)	7.38	.78	0.001
Serum uric acid at 12 weeks (mg/dL)	6.09	.25	

Table-II. Mean change in uric acid level after use of losartan in hypertensive Patients. (n=100)

DISCUSSION

This study delved into the role of losartan in reducing serum uric acid levels among hypertensive patients, encompassing a detailed analysis of demographic variables and clinical outcomes. Our findings underscore a significant reduction in serum uric acid levels following losartan treatment, evidenced by a decrease from a baseline mean of 7.38 mg/dL to 6.09 mg/dL at 12 weeks ($p = 0.001$). This reduction was consistently observed across various subgroups: participants aged 18-40 years and 41-60 years, both males and females, urban and rural residents, those with BMI ≤ 25 and > 25, and those with durations of hypertension ≤ 18 months and > 18 months.

The efficacy of losartan in reducing serum uric acid levels aligns with existing literature suggesting that angiotensin receptor blockers (ARBs) exert uricosuric effects by modulating renal uric acid excretion (Feig et al., 2008.[14]; Gherghina ME et al, 2022.[15]). This mechanism is important because hyperuricemia is frequently linked to hypertension and serves as a risk factor for cardiovascular illnesses and kidney dysfunction (Feig et al., 2008.[14]).

Variables		Serum Uric Acid	n	Change in Uric Acid Level After Losartin		
				Mean	SD	P-Value
Age	18 - 40 years	at baseline	9	7.38	.89	0.001
		at 12 weeks	9	6.05	.26	
	41 - 60 years	at baseline	91	7.38	.77	0.001
		at 12 weeks	91	6.10	.25	
Gender	Male	at baseline	55	7.41	.85	0.001
		at 12 weeks	55	6.10	.27	
	Female	at baseline	45	7.34	.69	0.001
		at 12 weeks	45	6.09	.22	
Residence	Urban	at baseline	63	7.37	.83	0.001
		at 12 weeks	63	6.09	.26	
	Rural	at baseline	37	7.39	.70	0.001
		at 12 weeks	37	6.10	.23	
BMI	≤ 25	at baseline	39	7.15	.69	0.001
		at 12 weeks	39	6.03	.22	
	> 25	at baseline	61	7.52	.80	0.001
		at 12 weeks	61	6.14	.26	
Duration of Hypertension	≤ 18 months	at baseline	31	7.37	.76	0.01
		at 12 weeks	31	6.09	.23	
	> 18 months	at baseline	69	7.38	.80	0.001
		at 12 weeks	69	6.09	.25	

Table-III. Stratification of change in serum uric acid with age, gender, residence, BMI & Duration of hypertension. (n=100)

Our study's findings expand understanding of angiotensin receptor blockers (ARBs) beyond their fundamental role of reducing blood pressure. highlighting their potential as a treatment for hyperuricemia and related issues. This is especially important given the growing recognition of hyperuricemia as a modifiable risk factor for gout and cardiovascular events.

However, the study has several of limitations. Because the majority of the participants were middle-aged and older, it might not be as applicable to younger populations. Moreover, dietary influences, which can markedly affect serum uric acid levels, were not sufficiently accounted for in this research. To establish the effect of uric acid decrease with losartan and its implications on clinical outcomes including cardiovascular events and gout flare-ups, more long-term trials with bigger and more diverse populations are required.

Future studies could examine the relative efficacy of different ARBs or how well they work in conjunction with other treatments that reduce uric acid levels. Additionally, It would be beneficial to do mechanistic studies that define the ways in which ARBs impact uric acid metabolism. Investigating the financial effects of using ARBs to treat hyperuricemia may also provide information on more affordable treatment options.

In summary, The study's findings demonstrate losartan's potential as a successful treatment option for lowering serum uric acid levels in hypertension patients. This study contributes to improving the management of hypertension and maybe improving patient outcomes in clinical settings by identifying its uricosuric characteristics and bringing the results within the body of existing literature.

CONCLUSION

The study's findings show that when losartan is administered to hypertensive patients, their serum uric acid levels significantly drop. Serum uric acid levels significantly decreased with losartan, going from an initial mean of 7.38 mg/dL to 6.09 mg/dL after 12 weeks (p-value = 0.001). This result highlights losartan's potential effectiveness in treating hyperuricemia linked to hypertension by indicating that it is effective in lowering serum uric acid levels during the course of treatment.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

SOURCE OF FUNDING

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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AUTHORSHIP AND CONTRIBUTION DECLARATION

No.	Author(s) Full Name	Contribution to the paper	Author(s) Signature
1	Tayyaba Khalid	Study planning, Data analysis, Drafting introduction and Discussion, section Manuscript write-up.	
2	Mohammad Omer Sultan	Conceptualization of the study, Supervision of clinical data collection, Review and critical revisions of the manuscript.	
3	Abdul Ghayas	Facilitating, Resources and permissions, biostatistical analysis and interpretation, Drafting and reviwwing results section, Ensuring statistical soundness of the study.	
4	Muhammad Inam Khan	Study oversight and Supervision Facilitating, Resources and Permissions, Final approval of the manuscript.	
5	Rukhsana Abdul Sattar	Study design, Guidance, Overall supervision, Critical review of manuscript for intellectual content.	
6	Parwasha	Data collection and organization, Literature review, Assisting with manuscript preparation.