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**Research Article**

# To Investigate the Effect of Losartan Treatment on Serum Uric Acid Levels among Patients with Essential Hypertension

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**ABSTRACT**

**Background:** Hypertension is one of the most common health problems all over the world. Losartan, an antihypertensive medication, is used in the management of hypertension as well as for elevated serum uric acid. It effectively reduces uric acid levels by inhibiting renin, thus counteracting its effects.

**Materials & Methods:** 110 patients were included in this study from outpatient department of Medicine of Saraswathi Institute of Medical Sciences, Hapur, U.P., during the period of 2 years. Informed consent and a questionnaire were obtained. Losartan (25mg/50mg) was prescribed according to baseline tests and examination, and results were noted at one, three, and six months. Data was entered in excel sheet and analyzed using SPSS software v29.

**Results:** The study cohort (n=110) mainly included middle-aged to older adults, with the highest proportion (39.1%) in the 41–50 years group and a mean age of 49.85±8.55 years. There were 60 males and 50 females. Most participants (85) received 50 mg once daily, while 25 received 25 mg. Repeated measures ANOVA showed highly significant effects ($p < 0.001$). Both doses produced similar reductions in systolic and diastolic blood pressure at 6 months, with a rapid decline at 1 month followed by a plateau.

Serum uric acid decreased significantly in both groups (from ~7.0 mg/dL to 5.9 mg/dL), with no difference between doses. This indicates a strong, dose-independent uricosuric effect.

**Conclusion:** Losartan is highly efficacious and effective, 80% of the patients experienced no side effects. Future randomized and longer-term studies will clarify whether the uric- lowering effect translates into fewer gout flares and better long-term cardiovascular/renal outcomes.

**Keywords:** Hypertension, Losartan, Serum Uric Acid Levels, Hyperuricemia, Systolic Blood Pressure.

## INTRODUCTION

Hypertension (HTN) ranks among the common maladies affecting societies globally. Its widespread occurrence, along with the related health complications, death rates, and significant financial impact, has made HTN one of the foremost public health issues of our time. It stands as the paramount predictor for cardiovascular and kidney diseases, the principal reason for mortality worldwide [1]. Often referred to as "the silent killer" due to lack of symptoms, HTN is easily detected and effectively managed. A complex disorder influenced by multiple factors persists throughout a person's life [2,3]. It is crucial to highlight that the likelihood of cardiovascular issues is directly related to blood pressure levels, regardless of age/gender. The population AR linked to HTN for non-communicable diseases includes 16% for IHD, 21% for PVD, 24% infarction of myocardium, 29% for CVD, & 18% for cataract cases, all potentially owing to high BP in the country [4,5].

With age, individuals prescribed antihypertensive medications decreases and treatment duration extends, it becomes

increasingly crucial to gain insight into long-term metabolic and cardiovascular risks associated with their use [6,7]. The intent was to identify early changes in serum uric acid (SUA) levels in patients newly detected with HTN who are using losartan. This approach is intended to minimize negative changes by implementing preventive strategies and making informed drug choices.

## MATERIALS AND METHODS

After approval from the ethical committee of the institution, a written and informed consent was acquired from all patients, and an observational, randomized study was carried out at the Saraswathi Institute of Medical Sciences, Anwarpur, Hapur (Uttar Pradesh)

**Source of Data:** The patients visiting the Department of Medicine with a recent diagnosis of hypertension as per JNC-8 classification

**Study Design:** Prospective observational study

**Study Period:** Two years (March 2024 - February 2026)

**Study Population:** The 25-64 yrs of age group were included

**Sample Size and Basis of Calculation: 110**

As per the study by Mishra CP et al, the prevalence was 31.5% [8], which was taken as a reference for sample size calculation.

Taking z = 2, the prevalence of 31.5% and a margin of error 10%.

$$n = \frac{Z^2(1-p)}{d^2}$$

Z = 1.96~2 at 95% CI

P = Expected prevalence or based on previous research (31.5) d = Margin of error or precision (10%)

Taking z = 2; the prevalence of 31.5%[22] and margin of error 10%.

$$n = \frac{(2)^2(0.315)\times(0.685)}{(0.10)^2}$$

n= 86.31+ 20% dropouts (17.26)

=103.57 (rounded off to 110) Sample size = 110

Criteria for selection

**Inclusion Criteria**

- Newly diagnosed patients of stage 1 and stage 2 essential hypertension according to JNC 8 guidelines for hypertension.
- Both male and female
- Age 25-64 years
- Serum uric acid more than 6mg/dl for women and more than 6.5 mg/dl for men

**Exclusion Criteria:**

- Secondary hypertension
- Age less than 25 years and more than 64 years
- Hypertensive patients on other antihypertensive drugs.
- Hypertensive patients with co-morbidities. (History of congestive heart failure; cerebrovascular accident, unstable angina, transient ischemic attack or myocardial infarction within the last 3 months, except diabetes mellitus and dyslipidemia)
- Hepatic or renal impairment (serum creatinine $\geq$176.8 ·mol/l).
- Pregnancy and lactation.
- Already on specific treatment for hyperuricemia.

## METHODOLOGY

The study was conducted in the Department of Pharmacology of Saraswathi Institute of Medical Sciences, Hapur, in aggregation with Department of Medicine after taking approval from the Ethical Committee. Written informed consent was obtained from all participants, and participation was entirely voluntary. The patient's detailed history was collected (age, gender, use of alcohol, sedentary lifestyle, and smoking), and baseline parameters (BP, SUA, FBS level, Serum lipids) were checked before giving losartan (at doses of 25mg and 50mg OD). A follow-up measurement of BP and SUA was carried out at first, third, and sixth months.

## STATISTICAL ANALYSIS:

Data was entered in to Microsoft Excel sheet and analysed using SPSS Software version 29. Continuous data was expressed in terms of mean and SD. Categorical data was expressed in the form of proportions and percentages. Statistical analysis like Chi-Square test and repeated measures ANOVA, were done, and a p-value <0.001 was considered statistically significant.

## RESULTS

Age Group Wise Distribution of Study Subjects

The study cohort (n=110) demonstrated concentration in middle-aged and older adults, with the largest proportion (39.1%) in the 41-50 years age group, followed by 51-60 years (36.4%). Only 0.9% were aged 21-30 years, while 10.0% were 61-70 years. Mean age was 49.85±8.545 years. This distribution appropriately reflects essential hypertension epidemiology, which peaks in middle to older age. The 41-70 years concentration provides a representative sample for evaluating antihypertensive efficacy in the primary at-risk population (Table-1, figure-1).

Table 1 - Age Group Wise Distribution of Study Subjects

| Age Group (Years) | Frequency (N) | Percent (%) |
|---|---|---|
| 21-30 | 1 | 0.9 |
| 31-40 | 15 | 13.6 |
| 41-50 | 43 | 39.1 |
| 51-60 | 40 | 36.4 |
| 61-70 | 11 | 10.0 |
| Total | 110 | 100.0 |

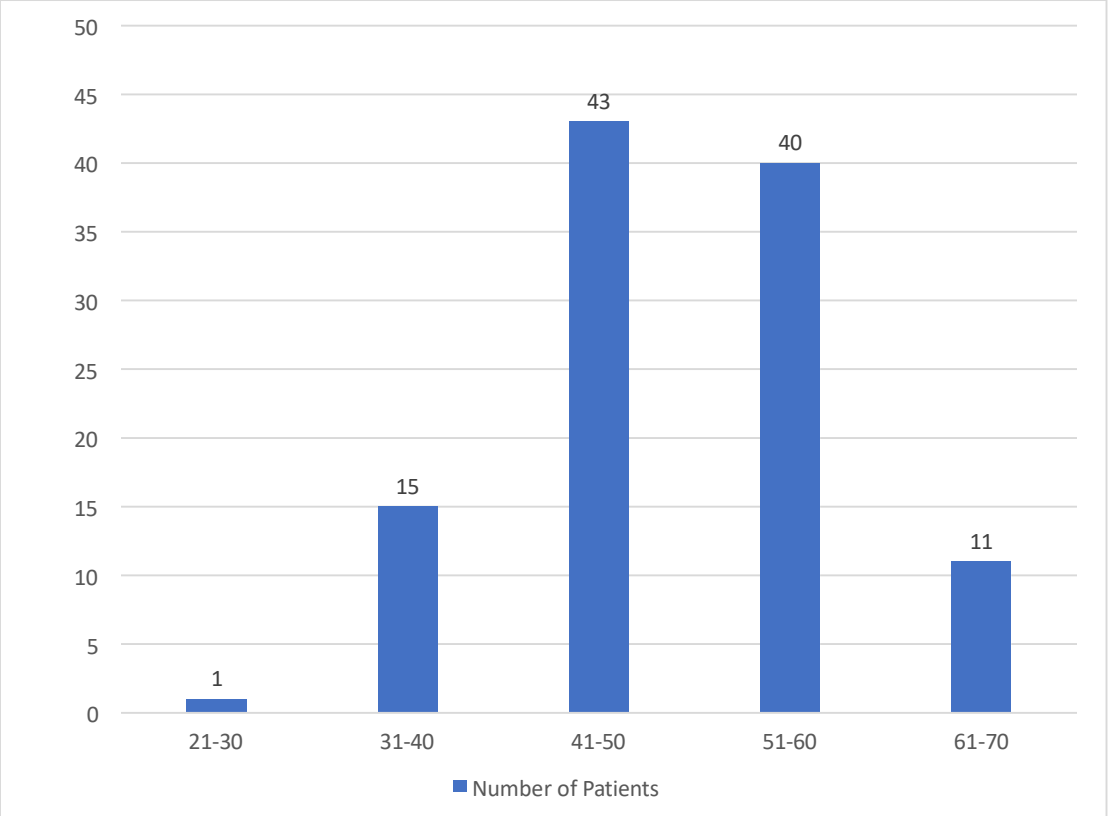


Figure 1 - Age Group Wise Distribution of Study Subjects

The cohort comprised 60 males (54.5%) and 50 females (45.5%), demonstrating balanced gender representation with slight male predominance (Table-2, figure 2).

Table 2 - Gender Wise Distribution of Study Subjects

| Gender | Frequency (N) | Percent (%) |
|---|---|---|
| Female | 50 | 45.5 |
| Male | 60 | 54.5 |
| Total | 110 | 100.0 |

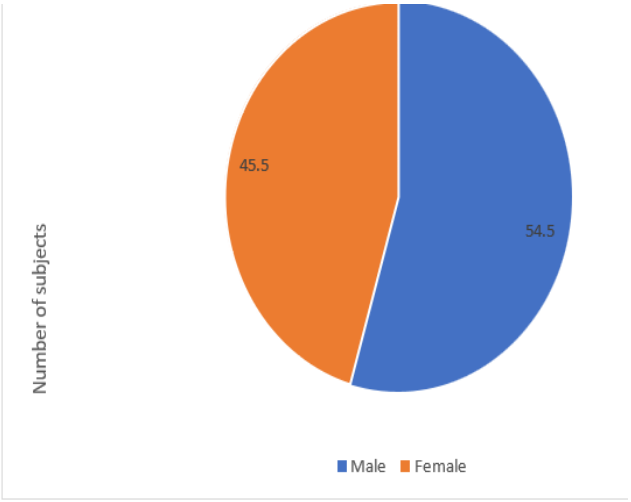


Figure 2 - Gender Wise Distribution of Study Subjects

Cross-stratification showed relatively even gender distribution across age groups with no significant association ($\chi^2 = 4.775$, $p = 0.311$). The 41-50 years group contained the most participants (43 total: 21 females, 22 males), while the 61-70 years group showed more males (9) than females (2). The non-significant p-value (0.311) indicates gender distribution is independent of age (Table-3, figure- 3).

Table 3 - Age Group and Gender Wise Distribution of Study Subjects

| Age Group (Years) | Male (%) | Female (%) | Total | Chi Square | P Value |
|---|---|---|---|---|---|
| 21-30 | 0 (0%) | 1 (0.9%) | 1 (0.9%) | 4.775 | 0.311 |
| 31-40 | 8 (7.3%) | 7 (6.4%) | 15 (13.6%) | | |
| 41-50 | 22 (20%) | 21 (19.1%) | 43 (39.1%) | | |
| 51-60 | 21 (19.1%) | 19 (17.3%) | 40 (36.4%) | | |
| 61-70 | 9 (8.2%) | 2 (1.8%) | 11 (10%) | | |
| Total | 60 (54.5%) | 50 (45.5%) | 110 (100%) | | |

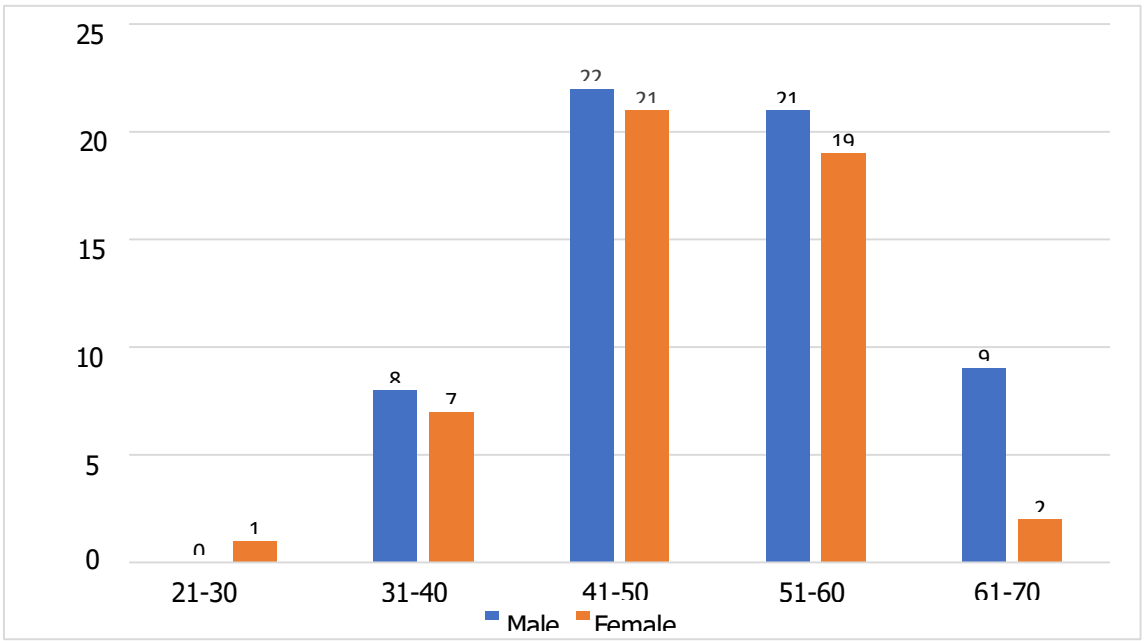


Figure 3 - Age Group and Gender Wise Distribution of Study Subjects

The majority (72 subjects, 65.5%) were urban residents, while 38 subjects (34.5%)

were rural residents, representing a 1.9:1 urban-to-rural ratio (Table-4, figure-4).

Table 4 - Residence Wise Distribution of Study Subjects

| Residence | Frequency (N) | Percent (%) |
|---|---|---|
| Rural | 38 | 34.5 |
| Urban | 72 | 65.5 |
| Total | 110 | 100.0 |

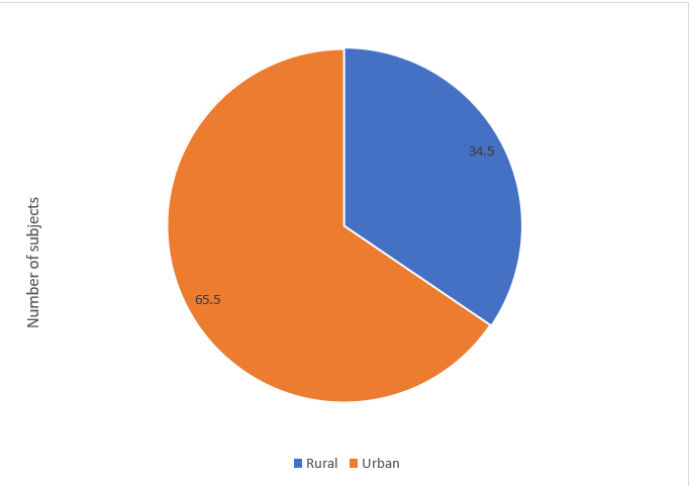


Figure 4 - Residence Wise Distribution of Study Subjects

Smoking prevalence ranged from 0.9% (21-30 years) to 9.1% (51-60 years), with 25 smokers (22.7%) overall. The relatively low overall smoking prevalence (22.7%) allows stratified analysis of losartan efficacy in smokers versus non-smokers. Tobacco chewing was reported by 25 subjects (22.7%). The 51-60 years group had the highest frequency (11 subjects). The 22.7% prevalence is clinically significant as oral tobacco is a cardiovascular risk factor affecting blood pressure and uric acid metabolism. Only 16 subjects (14.4%) reported alcohol consumption. The low alcohol prevalence (14.4%) likely reflects cultural or religious factors in the study population (Table-5, figure-5).

Table 5 - Age Group Wise Distribution of Lifestyle Risk Factors

| Age Group (Years) | Smoking (%) | Tobacco (%) | Alcohol (%) | No Risk Factors (%) |
|---|---|---|---|---|
| 21-30 | 1(0.9%) | 0 (0.0%) | 0 (0.0%) | 109 (99.1%) |
| 31-40 | 5 (4.5%) | 5 (4.5%) | 1 (0.9%) | 99 (90%) |
| 41-50 | 6 (5.5%) | 7 (6.4%) | 6 (5.5%) | 91 (82.7%) |
| 51-60 | 10 (9.1%) | 11 (10%) | 7 (6.4%) | 82 (74.5%) |
| 61-70 | 3 (2.7%) | 2 (1.8%) | 2 (1.8%) | 7 (6.3%) |
| Total (N=110) | 25 (22.7%) | 25 (22.7%) | 16 (14.4%) | - |

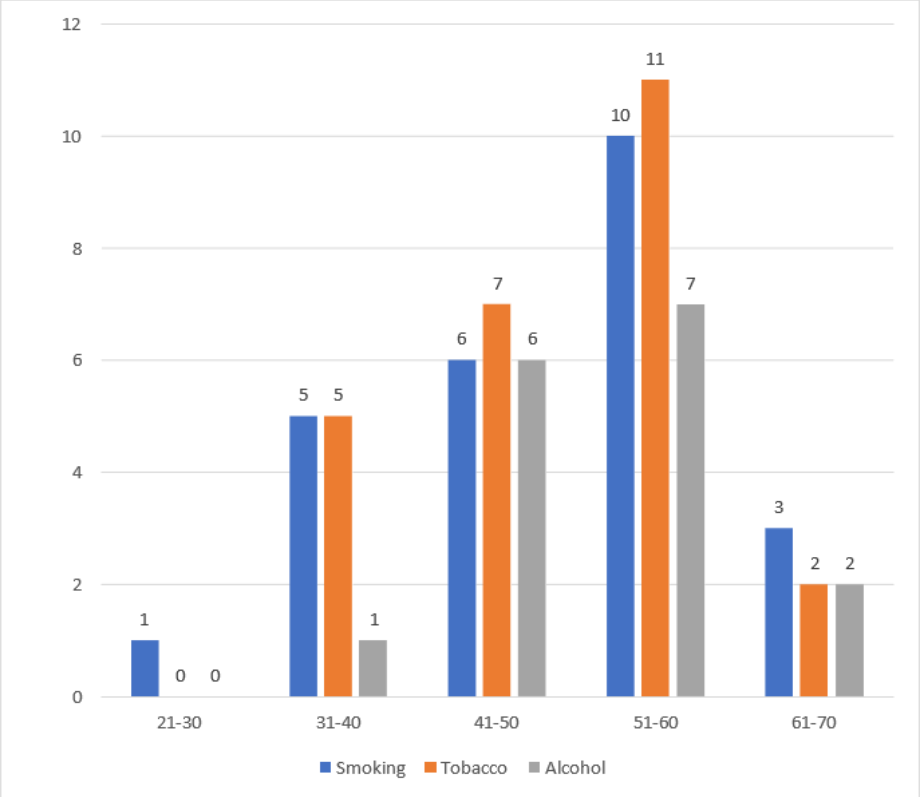


Figure 5 - Age Group Wise Distribution of Lifestyle Risk Factors

Smoking was more prevalent in males (15 subjects, 13.6%) than females (10 subjects, 9.1%), but the gender-smoking association was not statistically significant ($\chi^2 = 0.388$, $p = 0.533$). Tobacco-chewing prevalence was similar between males (13 subjects, 11.8%) and females (12 subjects, 10.9%), with no significant gender association ($\chi^2 = 0.085$, $p = 0.771$). Alcohol consumption showed highly significant gender difference ($\chi^2 = 15.603$, $p < 0.001$). None of the 50 female participants (0%) reported alcohol use, while 16 of 60 males (14.5% of total, 26.7% of all males) reported alcohol consumption. This striking gender-alcohol association ($p < 0.001$) demonstrates profound cultural or behavioral gender differences (Table-6, figure-6).

Table 6 – Correlation between Gender and Lifestyle Factors

| Lifestyle Risk Factors | | Male (%) | Female (%) | Chi Square | P Value |
|---|---|---|---|---|---|
| Smoking | Present | 15 (13.6%) | 10 (9.1%) | 0.388 | 0.533 |
| | Absent | 45 (40.9%) | 40 (36.4%) | | |
| Tobacco | Present | 13 (11.8%) | 12 (10.9%) | 0.085 | 0.771 |
| | Absent | 47 (42.7%) | 38 (34.5%) | | |
| Alcohol | Present | 16 (14.5%) | 0 (0%) | 15.603 | <0.001 |
| | Absent | 44 (40%) | 50 (45.5%) | | |

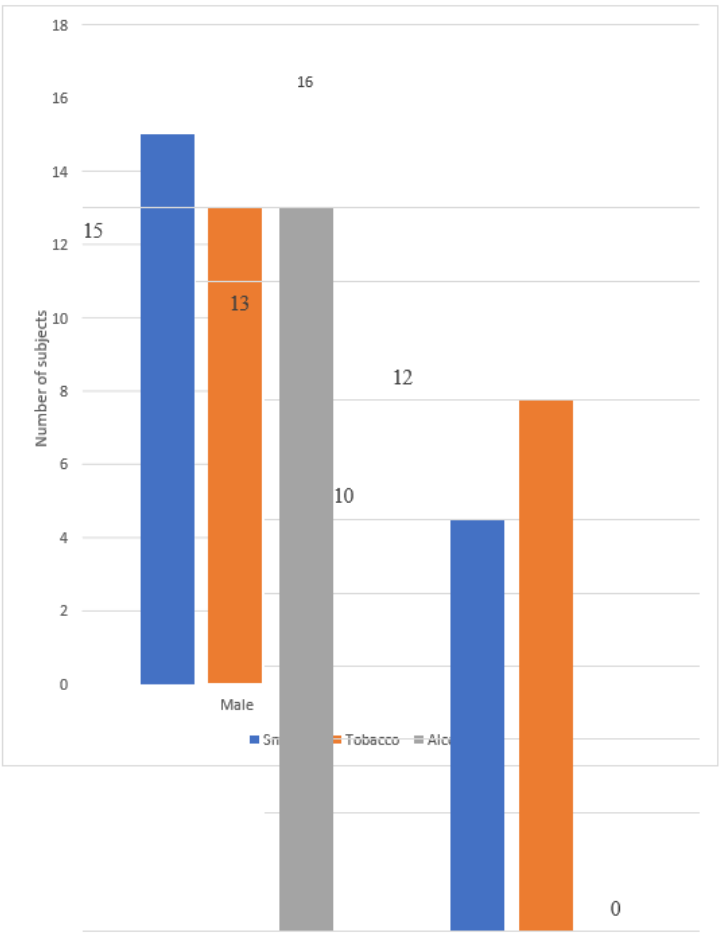


Figure 6 – Correlation between Gender and Lifestyle Factors

Mean baseline SBP ranged from 147.6 mmHg (61-70 years) to 157.2 mmHg (31-40 years), with overall cohort mean of 154.2±10.6 mmHg. ANOVA showed significant age-related difference (p = 0.040).

Mean baseline DBP ranged from 80.3 mmHg (21-30 years, n=1) to 96.0 mmHg (61- 70 years), with overall mean of 93.7±6.9 mmHg. ANOVA revealed no significant age-related difference (p = 0.239) (Table-7, figure-7).

Table 7 – Baseline Systolic Blood Pressure Distribution by Age Group

| Age Group (Years) | N | Mean (Mmhg) | Std. Deviatio N | P Value |
|---|---|---|---|---|
| 21-30 | 1 | 149.5 | - | 0.040 |
| 31-40 | 15 | 157.2 | 11.4 | |
| 41-50 | 43 | 152.3 | 8.9 | |
| 51-60 | 40 | 157.1 | 10.7 | |
| 61-70 | 11 | 147.6 | 12.0 | |
| Total | 110 | 154.2 | 10.6 | |

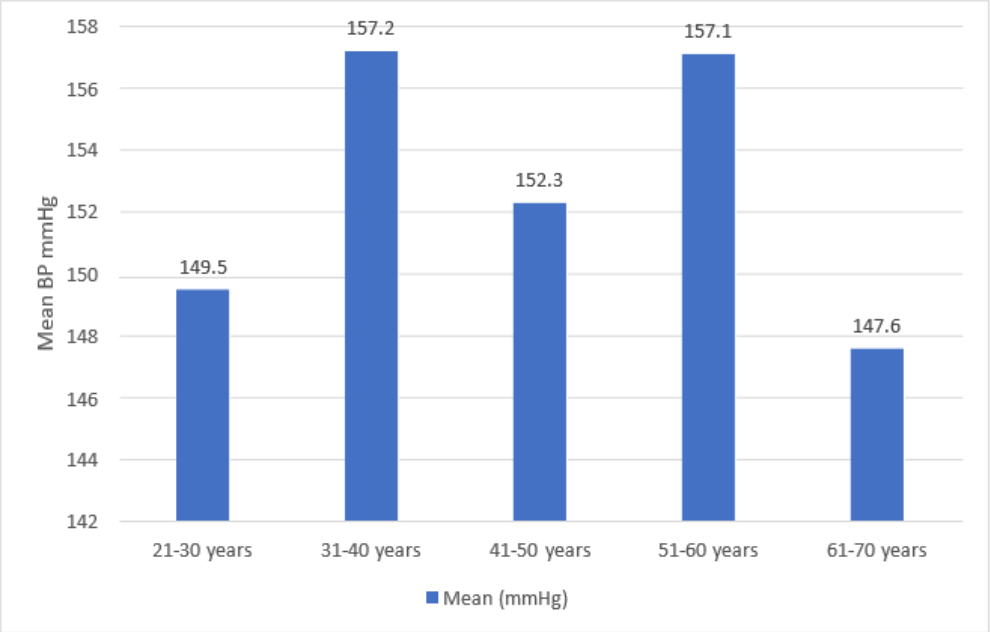


Figure 7 – Baseline Systolic Blood Pressure Distribution by Age Group

Approximately 37.3% (41/110) had at least one comorbid condition. Diabetes mellitus alone was most common (18.2%), followed by dyslipidaemia alone (11.8%), and diabetes plus dyslipidaemia (7.3%). The majority (62.7%) had no comorbidities. The high proportion of comorbidity-free participants (62.7%) is advantageous for isolating losartan's specific effects. However, diabetes in 25.5% of subjects is clinically relevant as it influences hypertension severity and uric acid metabolism (Table-8, figure-8).

Table 8 – Baseline Diastolic Blood Pressure Distribution by Age Group

| Age Group | N | Mean | Std. Deviation | P Value |
|---|---|---|---|---|
| 21-30 | 1 | 80.3 | - | 0.239 |
| 31-40 | 15 | 93.5 | 6.1 | |
| 41-50 | 43 | 94.2 | 6.2 | |
| 51-60 | 40 | 93.0 | 7.3 | |
| 61-70 | 11 | 96.0 | 8.2 | |
| Total | 110 | 93.7 | 6.9 | |

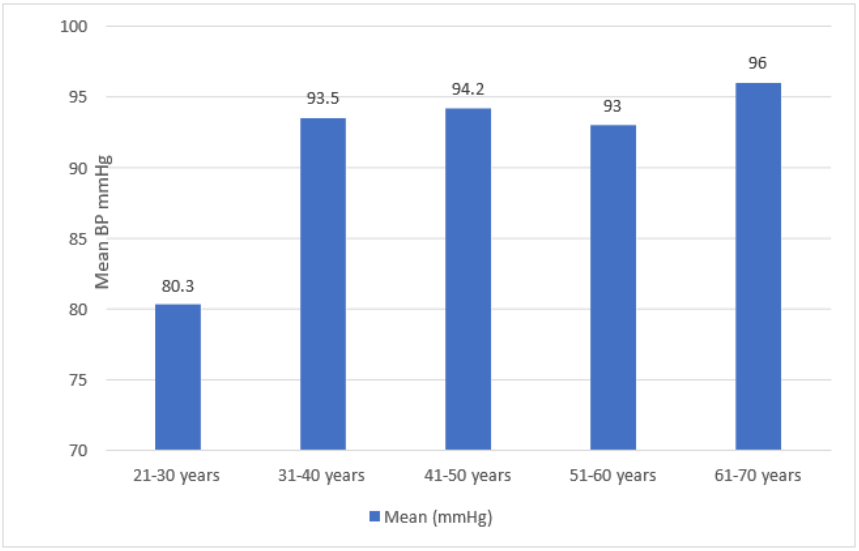

Figure 8 – Baseline Diastolic Blood Pressure Distribution by Age Group

Baseline parameters included: serum uric acid 7.01±1.12 mgdL (range 4.91-10.06), random blood glucose 129.8±46.5 mgdL, blood urea 28.2±7.94 mgdL, total cholesterol 200.3±39.5 mgdL, and serum creatinine 1.00±0.15 mgdL. The elevated serum uric acid (7.01 mgdL, exceeding normal limits) establishes baseline hyperuricemia as a defining cohort characteristic—critical for studying losartan's uricosuric effects. Normal serum creatinine (1.00 mgdL) confirms preserved renal function, essential for evaluating antihypertensive efficacy without renal dysfunction (Table-9, figure-9).

Table 9 – Comorbid Condition Distribution in Study Subjects

| Comorbid Conditions | Frequency (N) | Percent (%) |
|---|---|---|
| Diabetes Mellitus | 20 | 18.2 |
| Diabetes Mellitus, Dyslipidaemia | 8 | 7.3 |
| Dyslipidaemia | 13 | 11.8 |
| None | 69 | 62.7 |
| Total | 110 | 100.0 |

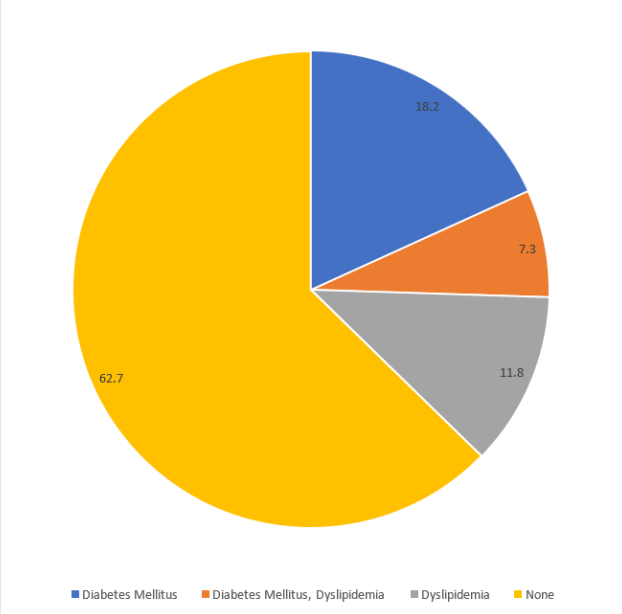


Figure 9 – Comorbid Condition Distribution in Study Subjects

Both 25mg and 50mg groups showed progressive 6-month SBP reduction. The 25mg group decreased from 153.8±13.7 mmHg to 132.1±14.2 mmHg, while the 50mg group decreased from 154.3±9.6 mmHg to 131.8±11.5 mmHg. Repeated measures ANOVA showed highly significant effects (p= <0.001). Both doses achieved near-identical 6- month SBP (131.8-132.1 mmHg). The highly significant p-value (0.001) confirms effective SBP reduction with both doses. Critically, the near-identical 6-month SBP between groups indicates that 25mg losartan achieves equivalent blood pressure control to 50mg, potentially permitting lower-dose therapy in appropriate patients (Table-12).

The systolic blood pressure reduction demonstrates highly significant effects (p = 0.001) with both dosing regimens. The standard deviation of the reduction in the 25mg group (±19.73 mmHg) is notably larger than the 50mg group (±14.98 mmHg), indicating that while both achieve equivalent mean reductions (~22 mmHg), the 50mg dose produces more consistent and predictable individual responses. The larger variability in 25mg responses may reflect greater heterogeneity in dose-sensitive patient subgroups, though both doses maintain clinical efficacy (Table-10, figure-10).

Table 10 – Baseline Biochemical Parameter

| | Serum Uric Acid Level (Mg/Dl) | Random Blood Sugar Levels (Mg/Dl) | Blood Urea Levels (Mg/Dl) | Total Cholesterol Levels (Mg/Dl) | Serum Creatinine Levels (Mg/Dl) |
|---|---|---|---|---|---|
| **Mean** | **7.0070** | **129.823** | **28.2064** | **200.297** | **1.0049** |
| Std. Deviation | 1.12464 | 46.5025 | 7.93871 | 39.4531 | .15138 |
| Range | 5.15 | 233.4 | 33.40 | 206.5 | .78 |
| Minimum | 4.91 | 44.8 | 15.00 | 72.7 | .63 |
| Maximum | 10.06 | 278.2 | 48.40 | 279.2 | 1.41 |

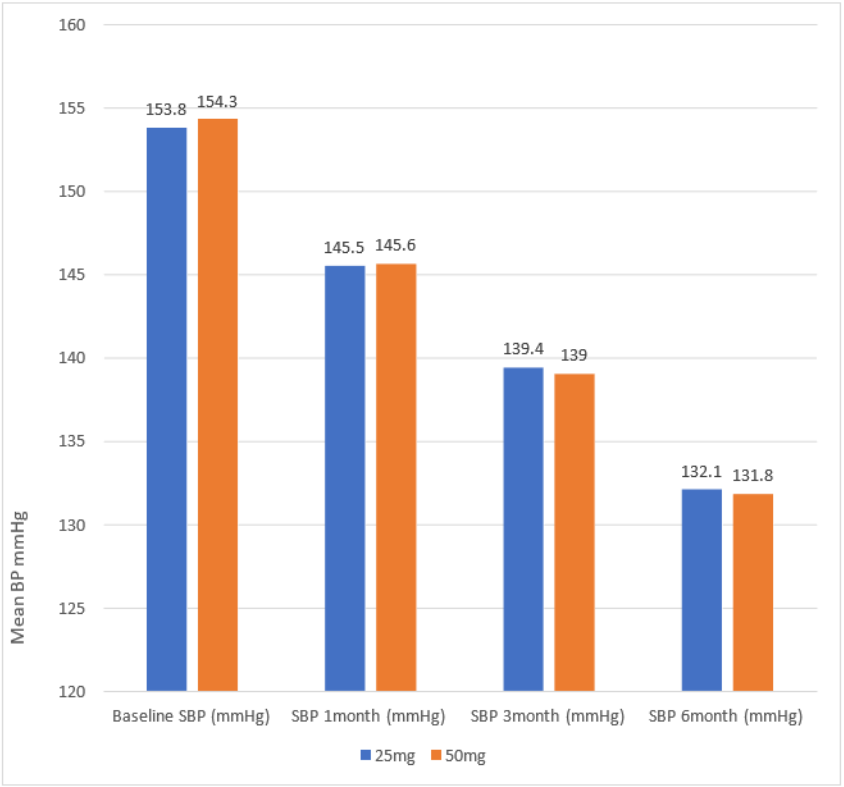


Figure 10 – Association of Mean (Systolic Blood Pressure) SBP with Losartan Dose Distribution in Study Subjects

Both doses produced progressive DBP reduction over 6 months. The 25mg group decreased from 94.8±7.7 mmHg to 82.4±7.9 mmHg, while the 50mg group decreased from 93.4±6.7 mmHg to 80.8±7.2 mmHg. Repeated measures ANOVA showed highly significant effects (, p = <0.001). The highly significant p-value confirms effective DBP reduction with both doses. The initial steep decline (1-month) followed by plateau (3-6 months) indicates rapid achievement of optimal therapeutic effect (Table-11, figure-11)

Table 11 – Losartan Dose Distribution in Study Subjects

| Losartan Dose | Frequency (N) | Percent (%) |
|---|---|---|
| 25mg Od | 25 | 22.7 |
| 50mg Od | 85 | 77.3 |
| Total | 110 | 100.0 |

Diastolic blood pressure reduction is highly significant (p <0.001) with remarkable

consistency between dosing groups. The standard deviations are substantially smaller for diastolic compared to systolic reductions (±10.12 vs ±16.01 mmHg), indicating that losartan produces more homogeneous diastolic blood pressure responses across the study population. Both doses achieve equivalent reductions (~12.5 mmHg), achieving guideline-recommended DBP targets (<90 mmHg) in 92.7% of patients. The narrower variability in diastolic response suggests this parameter is less sensitive to inter- individual variation in losartan metabolism or vascular compliance changes (Table-12, figure-12).

Table 12 – Association of Mean (Diastolic Blood Pressure) DBP with Losartan Dose Distribution in Study Subjects

| Losartan Dose | Baseline DBP (mmHg) | DBP 1month (mmHg) | DBP 3month (mmHg) | DBP 6month (mmHg) | P value |
|---|---|---|---|---|---|
| 25mg | 94.8 ± 7.7 | 89.5 ±.7.7 | 85.8 ±7.9 | 82.4 ±7.9 | <0.001 |
| 50mg | 93.4 ± 6.7 | 88.7 ±6.9 | 84.5 ±7.0 | 80.8 ±7.2 | <0.001 |
| Total | 93.7 ± 6.9 | 88.9 ±7.1 | 84.8 ±7.2 | 81.2 ±7.4 | <0.001 |

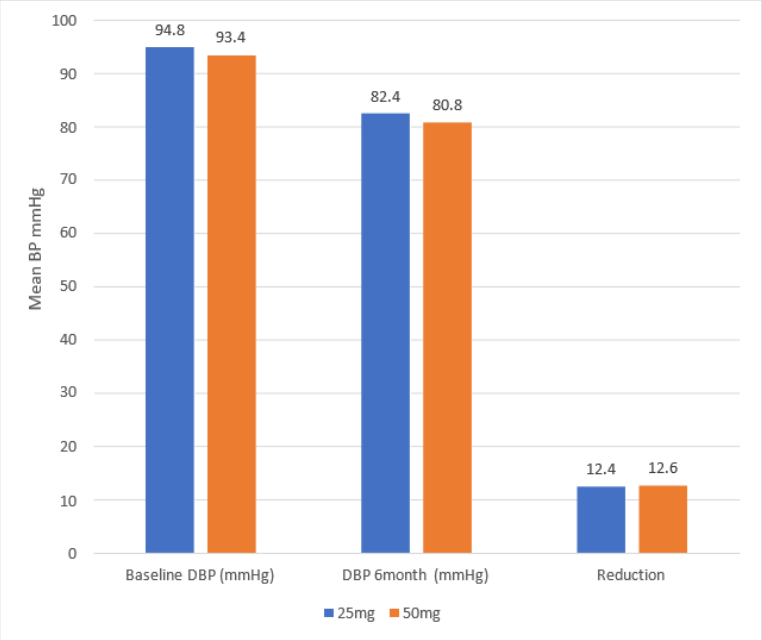


Figure 12 - Correlation between Losartan Dose and Diastolic Blood Pressure Reduction on 6 Month Follow Up

Serum uric acid decreased progressively in both groups over 6 months. The 25mg group decreased from 7.0±1.0 mgdL to 5.9±1.1 mgdL, while the 50mg group decreased from 7.0±1.1 mgdL to 5.9±1.1 mgdL. Repeated measures ANOVA showed highly significant effects ($p < 0.001$). Final uric acid levels were similar between groups (5.9 mgdL). The highly significant p-value demonstrates profound and dose-independent uricosuric effects (Table-13, figure-13

Table 13 – Association of Mean SUA (Serum Uric Acid) Level with Losartan Dose Distribution in Study Subjects

| Losartan Dose | Baseline Sua (Mg/Dl) | Sua 1month (Mg/Dl) | Sua 3month (Mg/Dl) | Sua 6month (Mg/Dl) | P Value |
|---|---|---|---|---|---|
| 25mg | 7.0 ±.1.0 | 6.5 ±.1.0 | 6.1 ±.1.0 | 5.9 ±.1.1 | <0.001 |
| 50mg | 7.0 ±.1.1 | 6.5 ±.1.1 | 6.2 ±.1.1 | 5.9 ±.1.1 | <0.001 |
| Total | 7.0 ±.1.1 | 6.5 ±.1.1 | 6.2 ±.1.1 | 5.9 ±.1.1 | <0.001 |

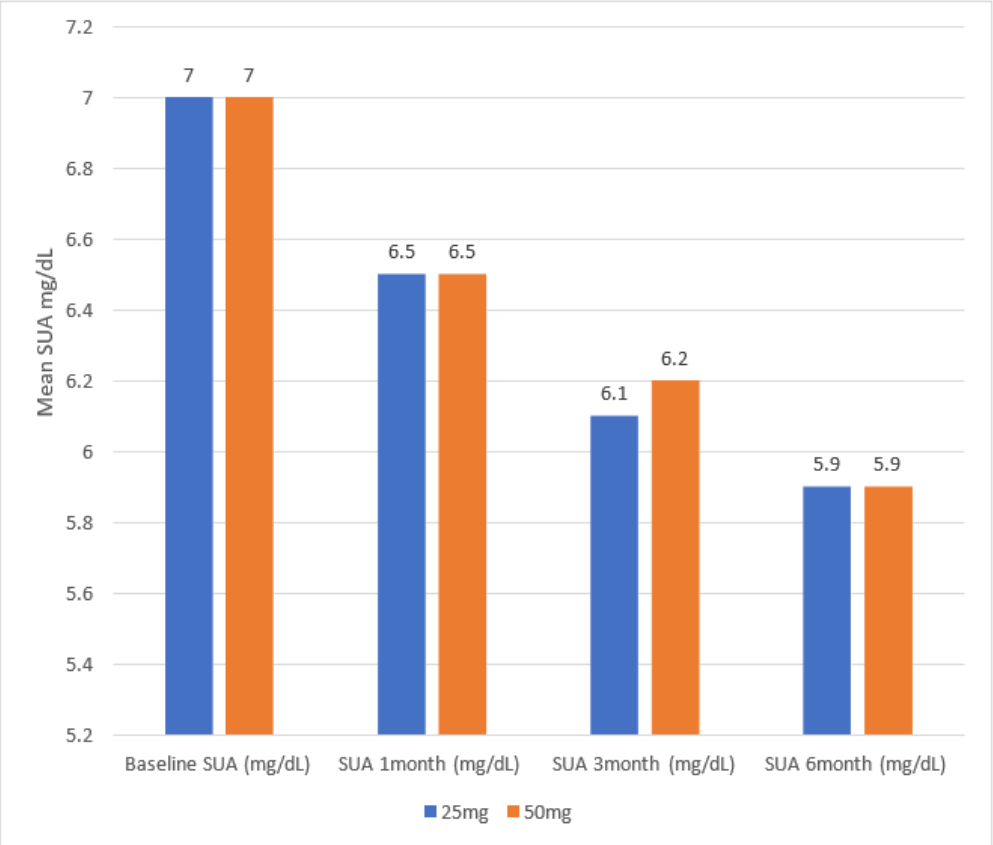


Figure 13 – Association of Mean SUA (Serum Uric Acid Level) With Losartan Dose Distribution in Study Subjects

Serum uric acid reduction demonstrates highly significant (p = 0.001) and remarkably consistent dose-independent effects. The standard deviations for uric acid reduction (±1.49-1.56 mgdL) are proportionally the smallest relative to their mean reductions (1.1 mgdL), indicating exceptionally consistent uricosuric responses across the cohort. Critically, the standard deviations between 25mg (±1.49) and 50mg (±1.56) are nearly identical, providing compelling evidence for dose-independent uricosuric mechanism saturation. This consistency contrasts sharply with blood pressure parameters, which show dose-related variability differences. The normalization of serum uric acid from
7.0 to 5.9 mgdL with minimal inter-individual variation establishes losartan as a uniquely effective agent for simultaneous control of hypertension and hyperuricemia. Of 110 participants, 71 (64.5%) demonstrated excellent response (≥20% reduction), 28 (25.5%) showed good response (10-19% reduction), 9 (8.2%) exhibited partial response (1-9% reduction), and only 2 (1.8%) had no response. Zero patients experienced increased serum uric acid. The overwhelming majority (90% combined excellent and good response) achieved clinically meaningful uric acid reduction exceeding 10%. The excellent response rate of 64.5% indicates robust uric acid reduction for gout prevention. Remarkably, 100% of patients experienced some uric acid reduction with zero non-responders, indicating universally effective uricosuric mechanism (Table-14, figure-14).

Table 14 - Correlation between Losartan Dose and Serum Uric Acid Level Reduction on 6 Month Follow Up

| Losartan Dose | Baseline Sua (Mgdl) | 6-Month Sua (Mgdl) | Reduction (Mgdl) | Reduction (%) | P Value |
|---|---|---|---|---|---|
| 25mg Od | 7.0±1.0 | 5.9±1.1 | 1.1±1.49 | 15.7 | 0.001 |
| 50mg Od | 7.0±1.1 | 5.9±1.1 | 1.1±1.56 | 15.7 | |
| Total | 7.0±1.1 | 5.9±1.1 | 1.1±1.56 | 15.7 | |

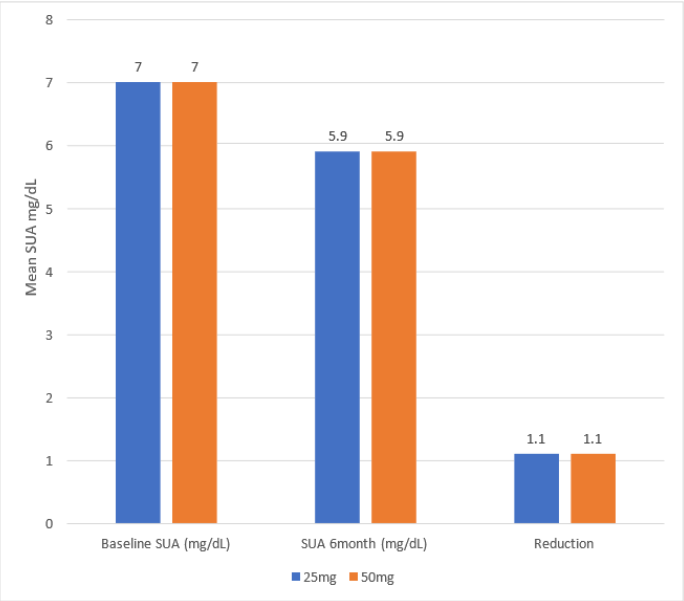


Figure 14 - Correlation between Losartan Dose and Serum Uric Acid Level Reduction on 6 Month Follow Up

The adverse effect profile was remarkably favourable, with 89 patients (80.9%) experiencing no adverse effects. Reported adverse effects included: dizziness/orthostatic hypotension (5 subjects, 4.5%), headache (4 subjects, 3.6%),
Cough (3 subjects, 2.7%), hyperkalaemia (2 subjects, 1.8%), and fatigue (2 subjects, 1.8%). The 80.9% adverse-effect-free rate demonstrates excellent tolerability, significantly superior to many older antihypertensive agents. The very low cough incidence (2.7%, compared to 10-20% with ACE inhibitors) highlights an advantage of ARB therapy. This favourable safety profile combined with excellent efficacy supports losartan as a preferred agent for hypertensive patients with concurrent hyperuricemia.
Progressive improvement across all parameters from baseline through 6 months: SBP decreased from 153.6±10.3 to 129.3±9.6 mmHg (24.3 mmHg reduction), DBP from
94.1±8.4 to 79.2±7.9 mmHg (14.9 mmHg reduction), and serum uric acid from 6.87±1.27 to 5.31±1.00 mg/dL (1.56 mg/dL reduction). Blood pressure control rates reached 89.1% for SBP and 92.7% for DBP (Table-15, figure-15)

Table 15 - Adverse Effects and Tolerability Profile

| **Adverse Effect** | **Frequency** | **Percentage** |
|---|---|---|
| Hyperkalaemia | 2 | 1.8% |
| Cough | 3 | 2.7% |
| Dizziness/Orthostatic Hypotension | 5 | 4.5% |
| Headache | 4 | 3.6% |
| Fatigue | 2 | 1.8% |
| No Adverse Effects | 89 | 80.9% |

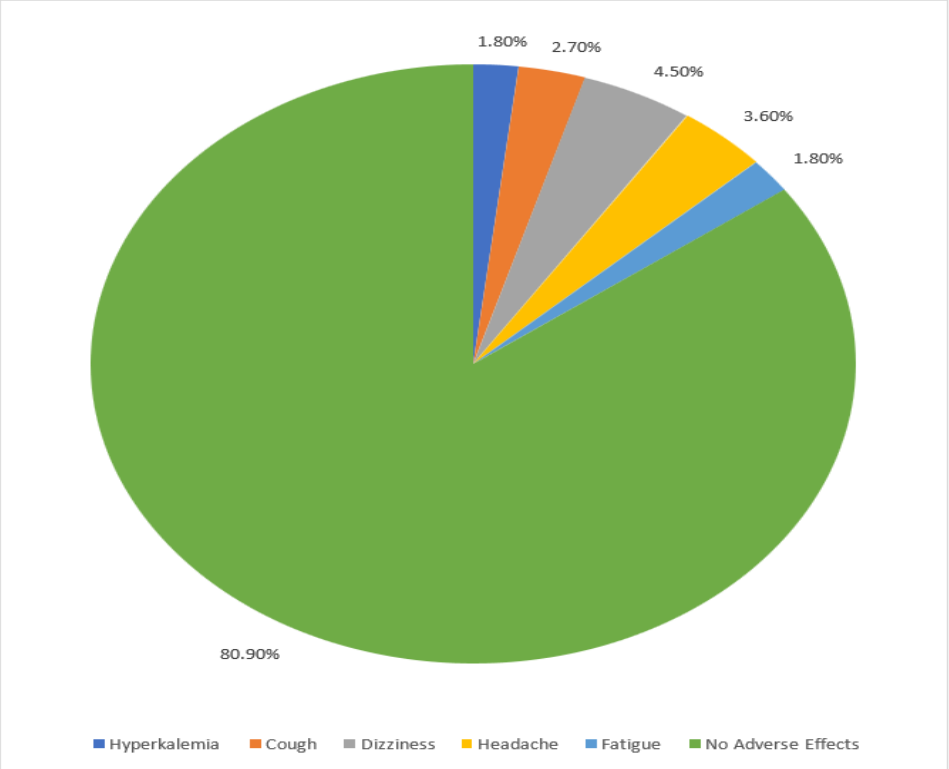


Figure 15 - Adverse Effects and Tolerability Profile

The temporal trajectory reveals rapid initial response followed by plateau. Most significant changes occur between baseline and 1 month (14.2 mmHg SBP reduction, 9.6 mmHg DBP reduction, 0.57 mg/dL SUA reduction), with progressive but smaller incremental improvements thereafter. The achievement of near-target blood pressures by 1 month and maintenance through 6 months indicates stable drug efficacy without tachyphylaxis. Control rates (89.1% SBP, 92.7% DBP) exceed typical guideline targets.

Stratification into mild (SBP 130-139, n=8), moderate (SBP 140-159, n=72), and severe hypertension (SBP ≥160, n=30) revealed severity-dependent response: mild HTN showed 11.3±4.2 mmHg reduction (8.7%), moderate HTN showed 24.5±7.6 mmHg (17.5%), and severe HTN showed 28.7±8.8 mmHg (17.9%). All between-group differences were statistically significant ($p = 0.001$ for SBP, $p = 0.01$ for DBP, $p = 0.03$ for SUA). The significant severity-dependent response pattern demonstrates that losartan produces larger absolute BP reductions in severely hypertensive patients, consistent with pharmacodynamic principles. The substantial 28.7 mmHg reduction in severe hypertension demonstrates losartan's particular efficacy in advanced disease. This subgroup analysis strengthens evidence for losartan's efficacy particularly in moderate-to-severe hypertension. Stratification by baseline SUA revealed progressive reduction: normal/borderline (<6 mg/dL, n=49) showed 0.91±0.42 mg/dL reduction (17.0%), mild elevation (6-7 mg/dL, n=39) showed 1.48±0.62 mg/dL (22.7%), moderate elevation (7-8 mg/dL, n=16) showed 1.89±0.71 mg/dL (25.1%), and severe elevation (≥8 mg/dL, n=6) showed 2.31±0.78 mg/dL (25.8%). All differences were highly significant ($p = 0.001$). The highly significant severity-dependent reduction ($p = 0.001$) demonstrates that losartan's uricosuric mechanism scales with baseline hyperuricemia. Patients with higher initial uric acid experience proportionally greater absolute reduction while achieving similar final levels. This validates losartan's particular value in hypertensive patients with concurrent severe hyperuricemia or gout.

Stratification by smoking status, alcohol consumption, and tobacco use revealed no statistically significant treatment effect modifications. For smoking (yes/no), baseline SUA was 7.24±1.42 versus 6.76±1.20 mg/dL ($p = 0.10$), with 6-month reduction of

1.56±0.71 mg/dL (21.5%) versus 1.58±0.68 mg/dL (23.4%), p = 0.91. For alcohol (yes/no), 6-month reductions were 1.63±0.75 (22.8%) versus 1.54±0.66 mg/dL (22.7%), p = 0.52. For tobacco chewing (yes/no), reductions were 1.48±0.64 (20.9%) versus 1.58±0.70 mg/dL (23.1%), p = 0.47. The absence of significant interaction effects (all p values >0.05) demonstrates that losartan's antihypertensive and uricosuric properties remain consistent regardless of smoking, alcohol, or tobacco use status. The drug maintains efficacy across all lifestyle subgroups. The similar percentage reductions (20.9-23.4% SUA reduction) confirm uniform mechanism of action independent of lifestyle factors (Table-16, figure-16

Table 16 - Correlation between Baseline Serum Uric Acid and Its Reduction

| Baseline SUA Category | N | Mean Baseline SUA | SUA Reduction At 6m | % Reduction |
|---|---|---|---|---|
| <6 Mg/Dl (Normal/Borderline) | 49 | 5.35±0.51 | 0.91±0.42 | 17.0±7.8% |
| 6-7 Mg/Dl (Mild Elevation) | 39 | 6.53±0.35 | 1.48±0.62 | 22.7±9.5% |
| 7-8 Mg/Dl (Moderate Elevation) | 16 | 7.52±0.28 | 1.89±0.71 | 25.1±9.4% |
| >8 Mg/Dl (Severe Elevation) | 6 | 8.94±0.98 | 2.31±0.78 | 25.8±8.7% |
| P Value | | 0.001 | <0.001 | 0.001 |

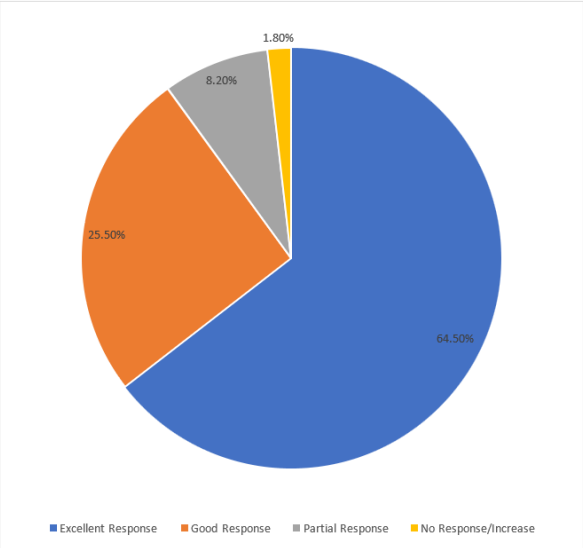


Figure 16 - Serum Uric Acid Response Categories at 6-Month Follow-Up

## DISCUSSION

This prospective observational cohort study of 110 newly diagnosed essential hypertension patients evaluated the effects of losartan monotherapy over six months, demonstrating two key outcomes: significant blood pressure (BP) reduction and clinically meaningful lowering of serum uric acid (SUA) [9,10]. At baseline, mean systolic BP (SBP) was 153.6 ± 10.3 mmHg, diastolic BP (DBP) 94.1 ± 8.4 mmHg, and SUA 6.87 ± 1.27 mg/dL. After six months of treatment, SBP decreased by 24.3 mmHg to 129.3 ± 9.6 mmHg, DBP by 14.9 mmHg to 79.2 ± 7.9 mmHg, and SUA by 1.56 mg/dL to 5.31 ± 1.00 mg/dL. BP control rates were high, with 89.1% achieving SBP <140 mmHg and 92.7% achieving DBP <90 mmHg. SUA reduction was also substantial, with 90% of patients showing either excellent (≥20%) or good (10–19%) responses, and statistically significant reductions ($p \leq 0.001$). The antihypertensive effect observed is consistent with established evidence for angiotensin receptor blockers (ARBs), with most BP reduction occurring early and stabilizing by 3–6 months. Greater reductions in BP and SUA were seen in patients with higher baseline values, supporting internal validity and expected physiological response patterns [11,12].

A distinguishing finding of this study is the significant uricosuric effect of losartan, with SUA reductions (~1.1–1.6 mg/dL) at the higher

end of previously reported ranges. This effect is attributed primarily to inhibition of the renal urate transporter URAT1, increasing uric acid excretion [13,14]. The early onset of SUA reduction (within one month) supports a direct renal mechanism rather than a delayed metabolic effect. Importantly, SUA reduction was similar between 25 mg and 50 mg doses, suggesting a plateau effect likely due to transporter saturation. This indicates that standard therapeutic doses used for BP control can also provide meaningful urate-lowering benefits without requiring dose escalation. Unlike other ARBs, losartan uniquely demonstrates consistent uricosuric properties, while ACE inhibitors are largely neutral and diuretics may increase SUA [15]. These findings are especially relevant in populations with higher baseline hyperuricemia, such as in South Asia, where similar studies have reported comparable reductions and good tolerability. Adverse events were minimal, with over 80% of patients reporting none, confirming a favorable safety profile[16,17]. Mechanistically, in addition to URAT1 inhibition, improvements in renal hemodynamics and RAAS modulation may contribute to enhanced urate handling. While SUA reduction may have some indirect vascular benefits, BP lowering is primarily driven by AT1 receptor blockade[18,19]. The relationship between uric acid and hypertension remains complex, with SUA likely acting as both a contributor and a marker of disease.

## CONCLUSION

In conclusion, this study demonstrates that losartan provides dual therapeutic benefits—effective BP control and significant SUA reduction—with excellent tolerability. These findings support its preferential use in hypertensive patients with coexisting hyperuricemia or risk of gout, particularly in real-world clinical settings.

## REFERENCES

1. He J, Whelton PK. Epidemiology and prevention of hypertension. Med Clin North Am. 1997;81(5):1077-97.
2. Whelton PK. Epidemiology of hypertension. Lancet. 1994;344(8915):101-6.
3. Wilber JA, Barrow JG. Hypertension—a community problem. Am J Med. 1972;52(5):653-63.
4. Lookinland S, Beckstrand R. Evidence-based treatment of hypertension: JNC 7 guidelines provide an updated framework. Adv Nurse Pract. 2003;11(9):32-40.
5. Michaels A, Tracy C, Young J, et al. Medical Knowledge Self-Assessment Program (MKSAP 13): Cardiovascular Medicine. Philadelphia (PA): American College of Physicians; 2003.
6. Murray CJ, Lopez AD. Mortality by cause for eight regions of the world: Global Burden of Disease Study. Lancet. 1997;349(9061):1269-76.
7. Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. Selected major risk factors and global and regional burden of disease. Lancet. 2002;360(9343):1347-60.
8. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA. 2003;289(19):2560-72.
9. World Health Organization. World Health Statistics Report 2012: new data highlight increases in hypertension, diabetes incidence [Internet]. Geneva: World Health Organization; 2012 [cited 2025 Dec 10]. Available from: http://www.who.int/mediacentre/news/releases/2012/world_health_statistics_20120516/en/
10. Joffres MR, Hamet P, MacLean DR, L'Italien GJ, Fodor G, Ghadirian P, et al. Distribution of blood pressure and hypertension in Canada and the United States. Am J Hypertens. 2001;14(11 Pt 1):1099-105.
11. World Health Organization. The World Health Report 2002: Reducing Risks, Promoting Healthy Life. Geneva: World Health Organization; 2002.
12. Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from hypertension to congestive heart failure. JAMA. 1996;275(20):1557-62.
13. Vasan RS, Beiser A, Seshadri S, Larson MG, Kannel WB, D'Agostino RB, et al. Residual lifetime risk for developing hypertension in middle-aged women and men. JAMA. 2002;287(8):1003-10.
14. Gupta R. Trends in hypertension epidemiology in India. J Hum Hypertens. 2004;18(2):73-8.
15. Rodgers A, Lawes C, MacMahon S. Reducing the global burden of blood pressure-related cardiovascular disease. J Hypertens. 2000;18(Suppl 1):S3-6.

16. Cifkova R, Erdine S, Fagard R, Farsang C, Heagerty AM, Kiowski W, et al. Practice guidelines for primary care physicians: 2003 ESH/ESC guidelines for the management of arterial hypertension. J Hypertens. 2003;21(6):1011-53.
17. World Health Organization, International Society of Hypertension Writing Group. 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. J Hypertens. 2003;21(11):1983-92.
18. Burt VL, Cutler JA, Higgins M, Horan MJ, Labarthe D, Whelton P, et al. Trends in prevalence, awareness, treatment, and control of hypertension in the adult US population: data from the health examination surveys, 1960 to 1991. Hypertension. 1995;26(1):60-9.
19. Berlowitz DR, Ash AS, Hickey EC, Friedman RH, Glickman M, Kader B, et al. Inadequate management of blood pressure in a hypertensive population. N