



## Original Research Article

# COMPARE THE ANTIHYPERTENSIVE EFFICACY AND SAFETY OF OLMESARTAN 20MG VERSUS TELMISARTAN 40MG IN STAGE 1 HYPERTENSIVE PATIENTS (JNC VIII) IN A TERTIARY CARE HOSPITAL OF PUNJAB

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**ABSTRACT**

**Background:** Hypertension is a major public health problem and an important risk factor for cardiovascular, cerebrovascular, and renal morbidity and mortality. Early and effective treatment of Stage I hypertension is essential to prevent disease progression and target organ damage. Angiotensin receptor blockers are commonly used in the management of hypertension because of their efficacy, tolerability, and favourable safety profile. Among them, Olmesartan and Telmisartan are widely prescribed, but comparative data regarding their antihypertensive efficacy and safety in Stage I hypertension remain clinically relevant. **Aim:** To determine the antihypertensive efficacy and safety of Olmesartan 20 mg versus Telmisartan 40 mg in patients with Stage I hypertension as per JNC VIII criteria.

**Materials and Methods:** This interventional study was conducted in the Medicine Outpatient Department of Sri Guru Ram Das Charitable Hospital, Amritsar. A total of 110 patients aged 30–80 years with Stage I hypertension were enrolled and randomly divided into two groups of 55 each using a computerized random number generator. Group A received Olmesartan 20 mg once daily, while Group B received Telmisartan 40 mg once daily. Patients were followed for 12 weeks with visits every 2 weeks. Blood pressure parameters including systolic blood pressure, diastolic blood pressure, and mean arterial pressure in sitting and supine positions were recorded. Baseline and final assessment of metabolic, haematological, hepatic, and renal parameters was done. Data were analysed using appropriate statistical methods, and  $p < 0.05$  was considered significant.

**Results:** Both groups were comparable at baseline for most sociodemographic, clinical, and biochemical parameters. Both drugs significantly reduced blood pressure over 12 weeks; however, Olmesartan produced a greater reduction than Telmisartan. Sitting systolic blood pressure decreased from  $150.945 \pm 5.133$  mmHg to  $126.909 \pm 4.519$  mmHg in the Olmesartan group, compared to  $150.400 \pm 5.010$  mmHg to  $134.927 \pm 3.962$  mmHg in the Telmisartan group. Similar superior reductions were observed in supine systolic blood pressure, diastolic blood pressure, and mean arterial pressure with Olmesartan. Telmisartan showed significant improvement in fasting blood sugar, while HbA1c improved significantly in both groups. HDL increased significantly in both groups, and urinary albumin-creatinine ratio decreased significantly in both groups, suggesting renal benefit. No significant adverse effects on liver function, renal function, or haematological parameters were noted in either group.

**Conclusion:** Both Olmesartan and Telmisartan were effective and safe in the management of Stage I hypertension. Olmesartan demonstrated superior antihypertensive efficacy, whereas Telmisartan showed comparatively better metabolic effects. The choice of therapy should therefore be individualized according to the patient's blood pressure control needs and associated metabolic profile.

**Keywords:** Hypertension; Olmesartan; Telmisartan; Antihypertensive efficacy; Safety profile.

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## INTRODUCTION

Hypertension remains one of the most important non-communicable diseases worldwide because of its strong association with cardiovascular, cerebrovascular, and renal morbidity and mortality. Persistent elevation of arterial pressure contributes to structural and functional damage in target organs and substantially increases the long-term risk of coronary artery disease, stroke, heart failure, chronic kidney disease, and premature death. In routine clinical practice, Stage 1 hypertension represents a particularly important therapeutic window because timely intervention at this stage can prevent progression to more severe disease and reduce the burden of complications. Modern hypertension management therefore emphasizes early diagnosis, accurate risk stratification, and prompt initiation of effective, well-tolerated antihypertensive therapy in appropriate patients.<sup>[1]</sup> In India, hypertension is a major public health problem and continues to rise in both urban and rural populations due to demographic transition, dietary changes, physical inactivity, obesity, psychosocial stress, and increasing life expectancy. The disease often remains asymptomatic for long periods, resulting in delayed diagnosis and inadequate control. This silent course leads many patients to present only after development of complications or target-organ damage. Even when diagnosed, control rates are often suboptimal because of poor adherence, irregular follow-up, therapeutic inertia, and limited awareness regarding long-term consequences. These realities highlight the need for effective first-line antihypertensive agents that are safe, convenient, and capable of providing consistent blood pressure reduction in diverse patient populations.<sup>[2]</sup> Current recommendations for the management of adult hypertension support individualized treatment based on baseline blood pressure level, overall cardiovascular risk, coexisting metabolic abnormalities, and the likelihood of sustained adherence. Among the available drug classes, agents acting on the renin-angiotensin-aldosterone system occupy a central role because they provide effective blood pressure reduction along with favourable cardiorenal effects. Angiotensin receptor blockers are especially attractive because they directly block angiotensin II type 1 receptor activity while avoiding some of the adverse effects associated with angiotensin-converting enzyme inhibitors. Their favourable tolerability profile improves treatment persistence and makes them

suitable for long-term use in uncomplicated hypertension as well as in patients with diabetes, renal involvement, or metabolic risk factors.<sup>[3]</sup> Olmesartan and Telmisartan are both widely used angiotensin receptor blockers, yet they differ in pharmacodynamic and pleiotropic characteristics that may influence their clinical performance. Olmesartan is recognized for potent and sustained blockade of the angiotensin II receptor, prolonged blood pressure control, and usefulness as monotherapy or in combination regimens. Telmisartan, in addition to antihypertensive activity, has drawn attention for broader metabolic effects, partly because of its selective peroxisome proliferator-activated receptor- $\gamma$  modulating properties. As a result, these two drugs are often considered attractive options for hypertensive patients who also have obesity, insulin resistance, dyslipidaemia, or early renal risk. Choosing between them may therefore depend not only on the extent of blood pressure reduction but also on safety and effects on associated biochemical parameters.<sup>[4]</sup> The relevance of comparing Olmesartan and Telmisartan is particularly high in Stage 1 hypertension, where both agents are commonly prescribed as initial therapy. At this stage, the ideal drug should achieve reliable blood pressure lowering, maintain hemodynamic stability, and demonstrate a favourable safety profile over repeated follow-up. Since Stage 1 hypertensive patients may remain on therapy for prolonged periods, even small differences in efficacy, metabolic neutrality, renal effects, or tolerability may influence long-term control and adherence. Furthermore, in real-world Indian settings, many patients present with overlapping cardiometabolic risks, making it clinically meaningful to evaluate whether one angiotensin receptor blocker offers any practical advantage over another in terms of both antihypertensive response and laboratory safety parameters.<sup>[5]</sup> Another important aspect of antihypertensive therapy is its influence on associated clinical and biochemical markers such as body mass index, fasting blood glucose, glycated haemoglobin, lipid profile, liver function, renal function, electrolytes, and urinary albumin excretion. These parameters are highly relevant in hypertensive patients because they reflect metabolic status, organ safety, and early evidence of target-organ protection. Drugs that effectively lower blood pressure while preserving hepatic and renal function and improving albuminuria or cardiometabolic profile may offer greater therapeutic value. Therefore, a head-to-head comparison of Olmesartan and Telmisartan is

justified not only from the perspective of blood pressure control but also from the broader standpoint of holistic patient management and long-term risk reduction.<sup>[6]</sup>

## MATERIALS AND METHODS

This interventional study was conducted in the Medicine Outpatient Department of Sri Guru Ram Das Charitable Hospital, attached to Sri Guru Ram Dass University of Health Sciences, Vallah, Amritsar. The study population comprised patients aged 30–80 years of both genders and from all socio-economic classes who were diagnosed with Stage 1 hypertension according to JNC VIII criteria. Each enrolled patient was followed up for a period of 3 months, while the total study duration was 18 months, which included the development of study tools, data collection, analysis, and presentation of findings.

A total of 110 patients fulfilling the eligibility criteria were included in the study, and the final sample size consisted of 55 patients in each group. Inclusion criteria were all patients of either gender aged 30 to 80 years with Stage 1 hypertension, including both newly diagnosed patients and those who had voluntarily discontinued antihypertensive medication for more than 4 weeks. Patients were excluded if they had a history of hypersensitivity to Olmesartan, Telmisartan, or other angiotensin receptor blockers; were pregnant or lactating women; were women of childbearing age using oral contraceptive pills; had significant renal disease with serum creatinine >2 mg/dl; had significant liver disease with SGOT/SGPT more than twice the normal values or cirrhosis; or had a history of stroke, myocardial infarction, cerebral haemorrhage, or hypertensive encephalopathy. Patients with cardiovascular conditions such as secondary hypertension, symptomatic heart failure, significant valvular heart disease, pericardial constriction or effusion, congenital heart disease, or those already on other antihypertensive therapy were also excluded. In addition, patients taking NSAIDs or corticosteroids were not enrolled.

### Methodology

Patients attending the Medicine OPD and satisfying the inclusion criteria were enrolled and randomly allocated into two groups of 55 each using a computerized random number generator. Group A received Tablet Olmesartan 20 mg once daily in the evening, while Group B received Tablet Telmisartan 40 mg once daily in the evening. Medications were dispensed for 2 weeks at a time, and patients were followed up at 2-week intervals for a total period of 3 months. At each follow-up visit, blood pressure, heart rate, mean arterial pressure, and ECG findings were recorded. Rescue medication in the form of Tablet Amlodipine 5 mg was administered if blood pressure rose during treatment. If a patient required rescue medication more than two times during the

study and blood pressure still remained uncontrolled despite therapeutic dosing, the patient was considered to have progressed to Stage II hypertension and was excluded from the study. The study also included patients with diabetes mellitus; among them, 21 patients in Group A and 23 patients in Group B had type 2 diabetes mellitus, while no patients had type 1 diabetes mellitus. Diabetic patients continued their antidiabetic treatment regimens, including metformin alone, metformin with insulin secretagogues, or metformin with insulin secretagogues and pioglitazone. A semi-structured, pretested, and predesigned questionnaire was used for data collection. It was translated into Hindi and Punjabi, pretested, modified as necessary, and retranslated into English. A patient information sheet was provided to all participants, written informed consent was obtained, and detailed medical history and physical examination were carried out. At the first and last visits, liver function tests, lipid profile, hemogram, kidney function tests, chest X-ray, 2D echocardiography, and body mass index were assessed. Any adverse events observed during the study were recorded in the Case Report Form.

### Statistical Analysis

The collected data were entered into Microsoft Excel and analysed using appropriate statistical software. Quantitative variables were expressed as mean  $\pm$  standard deviation, while qualitative variables were presented as percentages and proportions. Both descriptive and inferential statistical methods were applied for data analysis, and conclusions were drawn accordingly.

## RESULTS

### Table 1: Baseline Sociodemographic Distribution of Study Participants

A total of 110 patients with Stage 1 hypertension were enrolled in the study and randomly allocated into two equal groups of 55 participants each. The age distribution of participants in both groups was comparable. In Group A, the majority of patients belonged to the 51–60 years age group (40.00%), followed by 61–70 years (30.91%), 40–50 years (21.82%), and >70 years (7.27%). Similarly, in Group B, most participants were in the 51–60 years age group (43.64%), followed by 61–70 years (29.09%), 40–50 years (16.36%), and >70 years (10.91%). The mean age was  $59.30 \pm 8.75$  years in Group A and  $60.60 \pm 8.65$  years in Group B. The difference in age distribution between the two groups was statistically non-significant ( $p=0.439$ ), indicating that both groups were age matched at baseline. Regarding gender distribution, females constituted a higher proportion in both groups. Group A had 35 females (63.64%) and 20 males (36.36%), whereas Group B had 34 females (61.82%) and 21 males (38.18%). The difference between the groups was statistically non-significant ( $p=0.844$ ), showing comparable gender distribution. Most study

participants belonged to rural areas. In Group A, 74.55% were rural residents and 25.45% were urban residents, while in Group B, 76.36% were rural and 23.64% were urban. This difference was not statistically significant ( $p=0.825$ ). With respect to religion, Sikh participants formed the majority in both groups. In Group A, 87.27% were Sikhs and 12.73% were Hindus, whereas in Group B, 85.45% were Sikhs and 14.55% were Hindus.

**Table 2: Comparison of Baseline Clinical and Biochemical Parameters**

At baseline, most clinical and biochemical parameters were similar between the two treatment groups. Body mass index (BMI) was significantly higher in Group B ( $26.954 \pm 1.378 \text{ kg/m}^2$ ) compared to Group A ( $26.369 \pm 1.385 \text{ kg/m}^2$ ), with a statistically significant difference ( $p=0.028$ ). Fasting blood sugar was  $112.964 \pm 24.792 \text{ mg/dL}$  in Group A and  $115.218 \pm 23.081 \text{ mg/dL}$  in Group B, with no significant difference ( $p=0.623$ ). HbA1c values were also comparable between Group A ( $6.102 \pm 0.790\%$ ) and Group B ( $6.176 \pm 0.675\%$ ), with  $p=0.598$ . Haemoglobin and total leukocyte count did not differ significantly between the two groups ( $p=0.296$  and  $p=0.607$  respectively). Lipid profile parameters including total cholesterol, LDL, HDL, triglycerides, and VLDL were similar in both groups with no statistically significant differences ( $p>0.05$ ). Liver function parameters (SGOT and SGPT) and renal function parameters such as serum urea, blood urea nitrogen, serum creatinine, serum potassium, and urinary albumin creatinine ratio (UACR) were also comparable between the groups ( $p>0.05$ ).

**Table 3: Comparison of Blood Pressure and Mean Arterial Pressure at Various Time Intervals**

At baseline, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) in both sitting and supine positions were comparable between the two groups, with no statistically significant differences ( $p>0.05$ ). After treatment initiation, both drugs produced progressive reductions in blood pressure over the 12-week study period. However, Group A (Olmesartan) demonstrated a greater reduction compared with Group B (Telmisartan). Sitting SBP reduced from  $150.945 \pm 5.133 \text{ mmHg}$  to  $126.909 \pm 4.519 \text{ mmHg}$  in Group A, whereas in Group B it reduced from  $150.400 \pm 5.010 \text{ mmHg}$  to  $134.927 \pm 3.962 \text{ mmHg}$  at 3 months. The difference between groups became statistically significant from the 2nd week onward and remained highly significant thereafter ( $p \leq 0.017$ ,  $p=0.001$  subsequently). Similarly, supine SBP

declined from  $148.436 \pm 4.955 \text{ mmHg}$  to  $126.582 \pm 4.417 \text{ mmHg}$  in Group A, compared to a reduction from  $148.073 \pm 5.456 \text{ mmHg}$  to  $133.309 \pm 3.785 \text{ mmHg}$  in Group B. For diastolic blood pressure, sitting DBP decreased from  $92.691 \pm 2.987 \text{ mmHg}$  to  $79.055 \pm 1.533 \text{ mmHg}$  in Group A, while in Group B it decreased from  $92.327 \pm 2.742 \text{ mmHg}$  to  $83.345 \pm 2.374 \text{ mmHg}$ . Supine DBP also showed a greater decline in Group A than Group B, with statistically significant intergroup differences from the 2nd week onward ( $p=0.001$ ). Mean arterial pressure showed a similar trend. Sitting MAP decreased from  $112.113 \pm 3.159 \text{ mmHg}$  to  $95.020 \pm 1.948 \text{ mmHg}$  in Group A, whereas in Group B it decreased from  $111.691 \pm 3.179 \text{ mmHg}$  to  $100.536 \pm 2.583 \text{ mmHg}$ . Supine MAP reduced from  $109.553 \pm 2.932 \text{ mmHg}$  to  $94.911 \pm 1.940 \text{ mmHg}$  in Group A, compared with  $109.551 \pm 2.977 \text{ mmHg}$  to  $99.753 \pm 2.556 \text{ mmHg}$  in Group B.

**Table 4: Comparison of Metabolic, Haematological, Lipid, Liver, and Renal Parameters from Baseline to 12 Weeks**

Both treatment groups demonstrated significant reductions in BMI after 12 weeks. BMI decreased from  $26.369 \pm 1.385$  to  $25.525 \pm 1.479 \text{ kg/m}^2$  in Group A and from  $26.954 \pm 1.378$  to  $25.195 \pm 1.638 \text{ kg/m}^2$  in Group B ( $p=0.001$  in both groups). However, the final BMI difference between groups was not statistically significant ( $p=0.270$ ). Fasting blood sugar decreased in both groups, but the reduction reached statistical significance only in Group B ( $p=0.001$ ), whereas Group A showed a non-significant decline ( $p=0.054$ ). HbA1c improved significantly in both groups, decreasing from  $6.102 \pm 0.790\%$  to  $5.762 \pm 0.755\%$  in Group A and from  $6.176 \pm 0.675\%$  to  $5.770 \pm 0.546\%$  in Group B. Final HbA1c values were comparable ( $p=0.994$ ). In lipid profile analysis, HDL cholesterol increased significantly in both groups ( $p=0.001$ ). LDL cholesterol and total cholesterol improved significantly only in Group B, whereas Group A showed non-significant changes. Liver function tests (SGOT and SGPT) did not show significant changes in either group, indicating no hepatotoxic effects of either drug. Similarly, renal parameters such as serum urea, BUN, serum creatinine, and serum potassium remained stable in both groups. A statistically significant reduction in UACR was observed in both groups ( $p=0.001$ ), indicating improvement in microalbuminuria and renal protection. Final UACR values were comparable between the groups ( $p=0.774$ ).

**Table 1: Baseline Sociodemographic Distribution of Study Participants**

| Variable  | Category        | Group A No.         | Group A %     | Group B No.         | Group B %     | p-value |
|-----------|-----------------|---------------------|---------------|---------------------|---------------|---------|
| Age group | 40–50 years     | 12                  | 21.82         | 9                   | 16.36         | 0.439   |
|           | 51–60 years     | 22                  | 40.00         | 24                  | 43.64         |         |
|           | 61–70 years     | 17                  | 30.91         | 16                  | 29.09         |         |
|           | >70 years       | 4                   | 7.27          | 6                   | 10.91         |         |
|           | <b>Total</b>    | <b>55</b>           | <b>100.00</b> | <b>55</b>           | <b>100.00</b> |         |
|           | <b>Mean age</b> | <b>59.30 ± 8.75</b> |               | <b>60.60 ± 8.65</b> |               |         |
| Sex       | Female          | 35                  | 63.64         | 34                  | 61.82         | 0.844   |
|           | Male            | 20                  | 36.36         | 21                  | 38.18         |         |

|                    |              |           |               |           |               |       |
|--------------------|--------------|-----------|---------------|-----------|---------------|-------|
|                    | <b>Total</b> | <b>55</b> | <b>100.00</b> | <b>55</b> | <b>100.00</b> |       |
| <b>Living area</b> | Rural        | 41        | 74.55         | 42        | 76.36         | 0.825 |
|                    | Urban        | 14        | 25.45         | 13        | 23.64         |       |
|                    | <b>Total</b> | <b>55</b> | <b>100.00</b> | <b>55</b> | <b>100.00</b> |       |
| <b>Religion</b>    | Hindu        | 7         | 12.73         | 8         | 14.55         | 0.781 |
|                    | Sikh         | 48        | 87.27         | 47        | 85.45         |       |
|                    | <b>Total</b> | <b>55</b> | <b>100.00</b> | <b>55</b> | <b>100.00</b> |       |

**Note:** p-value <0.05 = Significant; p-value <0.001 = Highly significant; p-value >0.05 = Not significant.

**Table 2: Comparison of baseline clinical and biochemical parameters between Olmesartan and Telmisartan groups**

| Baseline variables      | Group A (Olmesartan) | Group B (Telmisartan) | p-value |
|-------------------------|----------------------|-----------------------|---------|
| BMI(Kg/m <sup>2</sup> ) | 26.369 ± 1.385       | 26.954 ± 1.378        | 0.028   |
| FBS(mg/DL)              | 112.964 ± 24.792     | 115.218 ± 23.081      | 0.623   |
| HbA1c(%)                | 6.102 ± 0.790        | 6.176 ± 0.675         | 0.598   |
| Hb                      | 12.858 ± 1.487       | 13.182 ± 1.736        | 0.296   |
| TLC                     | 6996.745 ± 1636.615  | 7164.909 ± 1780.44    | 0.607   |
| TC(mg/dl)               | 169.855 ± 4.378      | 168.891 ± 5.553       | 0.314   |
| LDL(mg/dl)              | 127.703 ± 4.301      | 128.895 ± 3.474       | 0.113   |
| HDL(mg/dl)              | 46.995 ± 3.383       | 47.841 ± 3.808        | 0.221   |
| TG(mg/dl)               | 166.121 ± 5.546      | 165.062 ± 5.780       | 0.329   |
| VLDL(mg/dl)             | 39.866 ± 10.430      | 40.674 ± 9.414        | 0.671   |
| SGOT(U/L)               | 30.158 ± 5.647       | 29.788 ± 5.648        | 0.732   |
| SGPT(U/L)               | 26.184 ± 4.581       | 26.758 ± 6.048        | 0.576   |
| S.Urea (mg%)            | 28.564 ± 4.022       | 28.627 ± 3.890        | 0.934   |
| BUN(mg/dl)              | 12.626 ± 3.166       | 12.436 ± 3.272        | 0.758   |
| S. Creat.(mg%)          | 0.921 ± 0.228        | 0.991 ± 0.421         | 0.281   |
| S. K+(mmol/L)           | 3.797 ± 0.208        | 3.801 ± 0.186         | 0.916   |
| UACR(mg/g)              | 29.984 ± 9.801       | 30.243 ± 11.065       | 0.897   |

**Table 3: Combined comparison of systolic blood pressure, diastolic blood pressure, and mean arterial pressure in sitting and supine positions between Olmesartan and Telmisartan groups at various time intervals**

| Parameter            | Time interval | Group A (Olmesartan) Mean ± SD | Group B (Telmisartan) Mean ± SD | p-value |
|----------------------|---------------|--------------------------------|---------------------------------|---------|
| <b>SBP (Sitting)</b> | Initial       | 150.945 ± 5.1331               | 150.400 ± 5.0096                | 0.578   |
|                      | 2nd week      | 144.182 ± 5.0519               | 146.473 ± 4.4881                | 0.017   |
|                      | 1st month     | 138.182 ± 5.0225               | 142.236 ± 4.2425                | 0.001   |
|                      | 2nd month     | 132.764 ± 5.0551               | 138.436 ± 3.8237                | 0.001   |
|                      | 3rd month     | 126.909 ± 4.5186               | 134.927 ± 3.9621                | 0.001   |
| <b>SBP (Supine)</b>  | Initial       | 148.436 ± 4.9545               | 148.073 ± 5.4564                | 0.721   |
|                      | 2nd week      | 141.636 ± 4.7468               | 144.509 ± 4.8567                | 0.005   |
|                      | 1st month     | 136.255 ± 4.8082               | 140.455 ± 4.3283                | 0.001   |
|                      | 2nd month     | 131.200 ± 4.8320               | 136.945 ± 3.8462                | 0.001   |
|                      | 3rd month     | 126.582 ± 4.4167               | 133.309 ± 3.7853                | 0.001   |
| <b>DBP (Sitting)</b> | Initial       | 92.691 ± 2.9868                | 92.327 ± 2.7424                 | 0.505   |
|                      | 2nd week      | 87.891 ± 2.5652                | 89.600 ± 2.7862                 | 0.002   |
|                      | 1st month     | 84.291 ± 2.2907                | 87.418 ± 2.5726                 | 0.001   |
|                      | 2nd month     | 80.982 ± 1.6721                | 85.091 ± 2.4591                 | 0.001   |
|                      | 3rd month     | 79.055 ± 1.5326                | 83.345 ± 2.3744                 | 0.001   |
| <b>DBP (Supine)</b>  | Initial       | 90.109 ± 2.891                 | 90.291 ± 2.225                  | 0.704   |
|                      | 2nd week      | 85.782 ± 2.767                 | 88.036 ± 2.449                  | 0.001   |
|                      | 1st month     | 82.473 ± 2.372                 | 86.073 ± 2.493                  | 0.001   |
|                      | 2nd month     | 80.582 ± 1.474                 | 84.473 ± 2.552                  | 0.001   |
|                      | 3rd month     | 79.055 ± 1.533                 | 82.982 ± 2.461                  | 0.001   |
| <b>MAP (Sitting)</b> | Initial       | 112.113 ± 3.1593               | 111.691 ± 3.1791                | 0.486   |
|                      | 2nd week      | 106.653 ± 2.7539               | 108.556 ± 3.0306                | 0.001   |
|                      | 1st month     | 102.255 ± 2.5902               | 105.691 ± 2.7933                | 0.001   |
|                      | 2nd month     | 98.240 ± 2.3085                | 102.869 ± 2.5822                | 0.001   |
|                      | 3rd month     | 95.020 ± 1.9478                | 100.536 ± 2.5831                | 0.001   |
| <b>MAP (Supine)</b>  | Initial       | 109.553 ± 2.9319               | 109.551 ± 2.9765                | 0.997   |
|                      | 2nd week      | 104.396 ± 2.7495               | 106.867 ± 2.8267                | 0.001   |
|                      | 1st month     | 100.393 ± 2.4994               | 104.196 ± 2.7159                | 0.001   |
|                      | 2nd month     | 97.458 ± 2.0551                | 101.969 ± 2.6143                | 0.001   |
|                      | 3rd month     | 94.911 ± 1.9403                | 99.753 ± 2.5562                 | 0.001   |

**Table 4: Combined comparison of metabolic, haematological, lipid, liver, and renal parameters between Olmesartan and Telmisartan groups from baseline to 12 weeks**

| Variable                   | Group A (Olmesartan) Baseline Mean±SD | Group A 12 weeks Mean±SD | P     | Group B (Telmisartan) Baseline Mean±SD | Group B 12 weeks Mean±SD | P     | P-value (final visit) |
|----------------------------|---------------------------------------|--------------------------|-------|----------------------------------------|--------------------------|-------|-----------------------|
| BMI (Kg/m <sup>2</sup> )   | 26.369±1.385                          | 25.525±1.479             | 0.001 | 26.954±1.378                           | 25.195±1.638             | 0.001 | 0.270                 |
| FBS (mg/dL)                | 112.964±24.792                        | 103.745±24.841           | 0.054 | 115.218±23.081                         | 108.982±20.362           | 0.001 | 0.229                 |
| HbA1c (%)                  | 6.102±0.790                           | 5.762±0.755              | 0.021 | 6.176±0.675                            | 5.770±0.546              | 0.001 | 0.994                 |
| Hb                         | 12.858±1.487                          | 13.609±1.343             | 0.314 | 13.182±1.736                           | 13.140±1.682             | 0.547 | 0.109                 |
| TLC                        | 6996.745±1636.615                     | 6810.564±1422.775        | 0.355 | 7164.909±1780.44                       | 7076.850±1517.52         | 0.780 | 0.345                 |
| TC (mg/dl)                 | 169.855±4.378                         | 168.795±4.688            | 0.279 | 168.891±5.553                          | 169.927±6.449            | 0.001 | 0.295                 |
| LDL (mg/dl)                | 127.703±4.301                         | 125.880±4.419            | 0.144 | 128.895±3.474                          | 124.931±3.742            | 0.001 | 0.227                 |
| HDL (mg/dl)                | 46.995±3.383                          | 48.804±3.372             | 0.001 | 47.841±3.808                           | 49.533±3.806             | 0.001 | 0.290                 |
| TG (mg/dl)                 | 166.121±5.546                         | 164.610±5.580            | 0.157 | 165.062±5.780                          | 166.096±5.592            | 0.072 | 0.166                 |
| VLDL (mg/dl)               | 39.866±10.430                         | 38.951±10.345            | 0.645 | 40.674±9.414                           | 41.877±6.853             | 0.900 | 0.083                 |
| SGOT (U/L)                 | 30.158±5.647                          | 30.293±5.621             | 0.170 | 29.788±5.648                           | 29.772±5.119             | 0.919 | 0.612                 |
| SGPT (U/L)                 | 26.184±4.581                          | 26.400±4.526             | 0.893 | 26.758±6.048                           | 26.999±5.749             | 0.155 | 0.545                 |
| S.Urea (mg%)               | 28.564±4.022                          | 29.255±3.811             | 0.593 | 28.627±3.890                           | 28.954±3.958             | 0.764 | 0.685                 |
| BUN (mg/dl)                | 12.626±3.166                          | 12.827±3.137             | 0.746 | 12.436±3.272                           | 12.733±3.220             | 0.759 | 0.877                 |
| S. Creat. (mg%)            | 0.921±0.228                           | 0.927±0.227              | 0.015 | 0.991±0.421                            | 1.005±0.420              | 0.861 | 0.228                 |
| S. K <sup>+</sup> (mmol/L) | 3.797±0.208                           | 3.7000±0.361             | 0.086 | 3.801±0.186                            | 3.752±0.185              | 0.156 | 0.344                 |
| UACR (mg/g)                | 29.984±9.801                          | 23.187±5.785             | 0.001 | 30.243±11.065                          | 22.825±7.321             | 0.001 | 0.774                 |

**Note:** p-value <0.05 = Significant; p-value <0.001 = Highly significant; p-value >0.05 = Not significant.

## DISCUSSION

In the present study, both treatment arms were well matched sociodemographically, with most patients belonging to the 51–60 year age group (40.00% in Group A and 43.64% in Group B), mean ages of  $59.30 \pm 8.75$  and  $60.60 \pm 8.65$  years, and a female preponderance in both groups (63.64% and 61.82%, respectively). This pattern is comparable to the study by Singh et al. (2020), in which the maximum number of patients in both groups also belonged to the 51–60 year age group, with near-matched sex distribution of 13 males/17 females in the Olmesartan arm and 14 males/16 females in the Telmisartan arm. Thus, the baseline demographic similarity in our study strengthens the internal validity of the subsequent efficacy comparison between the two drugs.<sup>7</sup> With regard to baseline clinical and biochemical variables, our study showed that the two groups were comparable for fasting blood sugar ( $112.96 \pm 24.79$  vs  $115.22 \pm 23.08$  mg/dL), HbA1c ( $6.10 \pm 0.79$  vs  $6.18 \pm 0.68\%$ ), haemoglobin, TLC, lipid parameters, liver enzymes, renal function, electrolytes, and UACR, while BMI alone was slightly but significantly higher in the Telmisartan group ( $26.95 \pm 1.38$  vs  $26.37 \pm 1.39$  kg/m<sup>2</sup>;  $p=0.028$ ). A similar baseline comparability was reported by Nakayama et al. (2008) in Japanese hypertensive

patients with early type 2 diabetes, where no significant baseline intergroup differences were noted before crossover treatment, and later no meaningful differences were seen in metabolic parameters such as HbA1c and adiponectin between Olmesartan and Telmisartan. This suggests that, apart from BMI, our groups were sufficiently balanced for valid therapeutic comparison.<sup>[8]</sup> The most important finding of our study was the greater antihypertensive efficacy of Olmesartan over Telmisartan. Sitting systolic blood pressure declined from  $150.945 \pm 5.133$  to  $126.909 \pm 4.519$  mmHg in Group A, a fall of 24.04 mmHg, whereas in Group B it declined from  $150.400 \pm 5.010$  to  $134.927 \pm 3.962$  mmHg, a fall of 15.47 mmHg. Likewise, supine systolic blood pressure fell by 21.85 mmHg in the Olmesartan group and by 14.76 mmHg in the Telmisartan group. These findings are in close agreement with Nakayama et al. (2008), who reported that Olmesartan reduced systolic, diastolic, and mean blood pressure by 3.3, 2.7, and 3.1 mmHg more than Telmisartan, respectively, and concluded that Olmesartan exerted a more potent blood pressure-lowering effect. Our study therefore reinforces the evidence that Olmesartan may provide stronger overall systolic blood pressure control than Telmisartan at standard doses.<sup>[8]</sup>

The serial reductions in diastolic blood pressure in our study also favoured Olmesartan, and the superiority became evident early. Sitting diastolic blood pressure decreased from  $92.691 \pm 2.987$  to  $79.055 \pm 1.533$  mmHg in Group A, compared with  $92.327 \pm 2.742$  to  $83.345 \pm 2.374$  mmHg in Group B; similarly, supine diastolic blood pressure declined from  $90.109 \pm 2.891$  to  $79.055 \pm 1.533$  mmHg in Group A versus  $90.291 \pm 2.225$  to  $82.982 \pm 2.461$  mmHg in Group B. These intergroup differences became significant from the second week onward. A very similar trend was documented by Gupta et al. (2016), where systolic blood pressure declined from  $151.13 \pm 6.80$  to  $133.1 \pm 5.60$  mmHg with Olmesartan versus  $150.7 \pm 5.26$  to  $136.67 \pm 3.22$  mmHg with Telmisartan, and diastolic blood pressure declined from  $91.3 \pm 4.29$  to  $80.8 \pm 2.49$  mmHg versus  $90.6 \pm 3.49$  to  $82.07 \pm 2.36$  mmHg, with significance appearing from the second week onward. Thus, our findings are strongly supported by prior short-term North Indian evidence.<sup>9</sup> Mean arterial pressure showed the same directional benefit in our trial. Sitting MAP fell from  $112.113 \pm 3.159$  to  $95.020 \pm 1.948$  mmHg in the Olmesartan group, compared with  $111.691 \pm 3.179$  to  $100.536 \pm 2.583$  mmHg in the Telmisartan group, while supine MAP declined from  $109.553 \pm 2.932$  to  $94.911 \pm 1.940$  mmHg versus  $109.551 \pm 2.977$  to  $99.753 \pm 2.556$  mmHg, respectively. This broader hemodynamic superiority of Olmesartan is consonant with the randomized open-label study by Kalikar et al. (2017), who compared Olmesartan, Telmisartan, and Losartan and concluded that Olmesartan was the most efficacious agent in reducing blood pressure, whereas Telmisartan and Losartan showed lesser antihypertensive efficacy. Hence, our MAP findings fit well with the concept that Olmesartan may produce stronger overall arterial pressure control than Telmisartan in Stage 1 hypertension.<sup>[10]</sup> On metabolic parameters, our study showed that BMI declined significantly in both groups, from  $26.369 \pm 1.385$  to  $25.525 \pm 1.479$  kg/m<sup>2</sup> with Olmesartan and from  $26.954 \pm 1.378$  to  $25.195 \pm 1.638$  kg/m<sup>2</sup> with Telmisartan. Fasting blood sugar decreased in both groups, but statistical significance was achieved only with Telmisartan ( $115.218 \pm 23.081$  to  $108.982 \pm 20.362$  mg/dL;  $p=0.001$ ), while HbA1c improved significantly with both drugs. These results partly mirror the randomized study by de Luis et al. (2010), in which Telmisartan produced a significant fall in glucose by 10.53 mg/dL, insulin by 2.51 mU/L, and HOMA by 1.08, whereas Olmesartan was more notable for improving lipid values. Therefore, our study also suggests a relative glycaemic advantage for Telmisartan, particularly for fasting glucose reduction, even though both drugs improved long-term glycaemic control as reflected by HbA1c.<sup>[11]</sup> Interestingly, our HbA1c findings differed from some previous work. In our study, HbA1c fell significantly in both groups, from  $6.102 \pm 0.790\%$  to  $5.762 \pm 0.755\%$  with Olmesartan and from  $6.176 \pm 0.675\%$  to  $5.770 \pm 0.546\%$  with Telmisartan, with no significant

final between-group difference ( $p=0.994$ ). By contrast, Arao et al. (2013), in a crossover study using high-dose Olmesartan and Telmisartan in hypertensive type 2 diabetics, reported no difference in blood pressure reduction between the drugs, but significant reductions of HbA1c, fasting plasma glucose, and HOMA-IR only in the Olmesartan group, along with a significant increase in HDL-C only with Olmesartan. This discrepancy may be attributable to differences in dose, crossover design, diabetic status of participants, and sample characteristics, but it also highlights that the metabolic pleiotropic profile of these ARBs may vary across populations.<sup>[12]</sup>

Lipid profile changes in our study were modest but informative. HDL cholesterol increased significantly in both groups, from  $46.995 \pm 3.383$  to  $48.804 \pm 3.372$  mg/dL with Olmesartan and from  $47.841 \pm 3.808$  to  $49.533 \pm 3.806$  mg/dL with Telmisartan. Total cholesterol and LDL decreased significantly only in the Telmisartan group, whereas triglycerides and VLDL did not change significantly in either arm. These findings are partly supported by Sasaki et al. (2008), who reported that Telmisartan was more beneficial than Olmesartan for early-morning blood pressure control and for improving glucose and lipid profiles, especially in patients with relatively high baseline HbA1c, total cholesterol, LDL cholesterol, and triglycerides. Thus, while our study did not show a universal lipid superiority of one drug, it does support a relative advantage of Telmisartan for total cholesterol and LDL reduction.<sup>[13]</sup> Renal findings in our study are particularly important. UACR decreased significantly in both groups, from  $29.984 \pm 9.801$  to  $23.187 \pm 5.785$  mg/g with Olmesartan and from  $30.243 \pm 11.065$  to  $22.825 \pm 7.321$  mg/g with Telmisartan, while serum urea, BUN, creatinine, and potassium remained largely stable. These results are consistent with the report by Ikeda et al. (2009), who found that after switching hypertensive patients with type 2 diabetes from other ARBs to Olmesartan, urinary albumin/creatinine ratio significantly decreased, suggesting improved early diabetic nephropathy. Our findings similarly indicate that both Olmesartan and Telmisartan have renoprotective potential, particularly in reducing albuminuria without major deterioration in conventional renal function indices.<sup>[14]</sup> The overall interpretation of our data is that Olmesartan was the stronger antihypertensive drug, while Telmisartan showed some relative metabolic advantages, especially in fasting glucose and selected lipid endpoints; both drugs were safe, with no meaningful adverse effect on hemogram, liver enzymes, serum urea, BUN, or potassium over 12 weeks. This overall pattern is broadly compatible with the larger renal-outcome data of Haller et al. (2011) in the ROADMAP trial, where Olmesartan delayed the onset of microalbuminuria by 23%, with microalbuminuria occurring in 8.2% of Olmesartan-treated patients versus 9.8% in controls, supporting a clinically relevant renoprotective effect beyond

simple blood pressure lowering. Taken together, our short-term results suggest that in Stage I hypertension, Olmesartan may be preferred when stronger blood pressure reduction is the principal objective, whereas Telmisartan may be attractive when metabolic considerations are prominent.<sup>[15]</sup>

## CONCLUSION

Both Olmesartan and Telmisartan were found to be effective and safe in the treatment of Stage I hypertension over the 12-week study period. Although both drugs significantly reduced systolic, diastolic, and mean arterial blood pressure, Olmesartan showed superior antihypertensive efficacy with a faster and greater reduction in blood pressure parameters. Telmisartan, while slightly less potent in blood pressure control, demonstrated additional favourable effects on glycaemic and lipid parameters. Thus, Olmesartan may be preferred when stronger blood pressure reduction is desired, whereas Telmisartan may be a better option in hypertensive patients with associated metabolic abnormalities.

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