

RESEARCH ARTICLE

Comparative study of telmisartan and ramipril as an antihypertensive in mild to moderate hypertension

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ABSTRACT

Background: Hypertension has been termed the silent killer; an asymptomatic chronic disorder that, if undetected and untreated, silently damages the blood vessels, heart, brain, and kidneys. In India, hypertension is emerging as a major health problem and is more prevalent in urban than in rural subjects. Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths in India. **Aims and Objectives:** To compare the efficacy of telmisartan and ramipril as an antihypertensive in mild to moderate hypertension. **Materials and Methods:** This study was a hospital-based prospective, randomized, comparative, observational study conducted over a period of 1-year. For the purpose of this study, equal numbers of mild to moderate hypertensive patients were randomly allocated equally between two groups: one group on telmisartan and the other group on ramipril. Patients were assessed for the blood pressure (BP) reduction during follow-up period of 6-month. **Results:** In both telmisartan and ramipril groups, there was a significant reduction of systolic BP (SBP), diastolic BP (DBP), and mean BP (MBP) from beginning to the end of study ($P < 0.001$). There was a significant difference in reduction of SBP and MBP during 4-12 weeks ($P < 0.001$) between telmisartan and ramipril group but no significant difference in the reduction of SBP and MBP in both drug groups was seen at the end of the study. **Conclusion:** Both telmisartan and ramipril groups were similar and comparable with regards to their SBP and DBP. In both telmisartan and ramipril groups, there was a significant reduction of SBP, DBP, and MBP from beginning to the end of study ($P < 0.001$).


KEY WORDS: Hypertension; Angiotensin-converting Enzyme Inhibitors; Angiotensin Receptors Blockers; Renin-angiotensin System

INTRODUCTION

Hypertension has been termed the silent killer; an asymptomatic chronic disorder that, if untreated, damages the blood vessels,

heart, brain, and kidneys.^[1] In India, hypertension is emerging as a major health problem and is more prevalent in urban than in the rural population. Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary artery disease deaths in India.^[2]

Hypertension remains the most common, risk factor for myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection, and peripheral arterial disease.^[3] Hypertension is a complex disorder that is influenced by genetic and environmental factors as well as their interactions. Genetic factors may determine individual's

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susceptibility to environmental risk factors and risk of developing hypertension. However, the environmental factors must be present to trigger the pathogenesis of the disease in most persons with hypertension. Among environmental risk factors, diet and nutrition play key roles, with intakes of sodium, potassium, fats, fiber, and protein clearly having an effect on blood pressure (BP) and the development of hypertension. Physical inactivity, alcohol consumption, obesity, and stress also play important roles in the development of hypertension.^[4]

Based on clinical trial data, the maximum protection against combined cardiovascular end points is achieved with pressures <135-140 mmHg for systolic BP (SBP) and <80-85 mmHg for diastolic BP (DBP); however, treatment does not reduce cardiovascular disease risk to the level in non-hypertensive individuals.^[5]

Activation of the renin-angiotensin system (RAS) is one of the most important mechanisms contributing to endothelial cell dysfunction, vascular remodeling, and hypertension. The interaction of angiotensin II (AII) with Angiotensin Type 1 (AT1) receptors activates numerous cellular processes that contribute to hypertension and accelerate hypertensive end-organ damage. These include vasoconstriction, generation of reactive oxygen species, vascular inflammation, vascular and cardiac remodeling, and production of aldosterone. There is increasing evidence that aldosterone, AII, and even renin activate multiple signaling pathways that can damage vascular health and cause hypertension.^[3]

Pharmacological therapy for hypertension is employed when non-pharmacological measures could not maintain the BP in an acceptable range.^[6] Angiotensin-converting enzyme (ACE) inhibitors and AII receptor blockers (ARBs) enjoy a popular status among antihypertensive drugs, due to lack of common side effects seen in other antihypertensive drugs and the absence of adverse effects on coexisting conditions. Telmisartan and ramipril are the one of the most commonly used ARB and ACE inhibitors for the treatment of hypertension, respectively.

Hemodynamic and metabolic consequences of ACE inhibition have distinct advantages. They decrease systemic vascular resistance in congestive heart failure; enhance insulin sensitivity in Type 2 diabetes; enhance renal blood flow in renal insufficiency; enhance diminished intraglomerular pressures in diabetic nephropathy, and enhance coronary blood flow in ischemic heart disease, thus, providing distinct benefits on concomitant conditions.^[7] By affecting intrarenal hemodynamics by preferential vasodilatation of efferent versus afferent arterioles with decreased intraglomerular pressure, ACE inhibition decreases proteinuria and provides protection against glomerulosclerosis and renal failure.^[8] Both ARBs and ACE inhibitors block RAS, but they differ

from each other in various aspects. These pharmacological differences are translated into differences in therapeutic efficacy has been an open question.^[9]

Telmisartan has a plasma half-life ($t_{1/2}$) of 24 h so that it provides sustained BP control.^[9,10] It also acts as a selective modulator of peroxisome proliferator-activated receptor gamma (PPAR- γ). PPAR- γ plays an important role in regulation of insulin and glucose metabolism. Thus, telmisartan provides protection against renal and vascular damage caused by the renal and cardiovascular disease.^[11]

Ramipril is a prodrug which is converted to its active form ramiprilat by hepatic esterases. Triphasic elimination kinetics is exhibited by ramiprilat with a long terminal half-life. The first phase is due to extensive distribution to all tissues (with half-life of 2-4 h); the second phase is due to clearance of free ramiprilat from plasma (with half-life of 9-18 h); the third phase is due to dissociation of ramiprilat from tissue ACE (with a half-life of >50 h).^[12]

Mild hypertension has been defined as DBP within 90-99 mmHg and/or SBP within 140-159 mmHg while moderate hypertension has been defined as DBP within 100-109 mmHg and/or SBP within 160-179 mmHg, respectively.^[13]

Most of the studies on similar drugs have been done in the western population, but we are ethnically different from our Caucasian counterpart, therefore, by this study, we want to establish an epidemiological data regarding the antihypertensive effect of these drugs in patients having mild to moderate hypertension at our settings. This study dealt with the comparative study of telmisartan and ramipril as an antihypertensive in mild to moderate hypertension. The idea beyond this study is to get evidence-based appropriate antihypertensive drug for hypertension.

MATERIALS AND METHODS

This study was a hospital-based prospective, randomized, comparative, observational study conducted over a period of 1-year. The subjects of this study were mild to moderate hypertensive patients selected from outpatient department and hypertensive clinic of Department of General Medicine of a tertiary care hospital.

A total of 100 patients were enrolled in the study as per the selection criteria. The inclusion criteria for this study were subjects of either sex of more than 25 years of age who were newly diagnosed patients, previously diagnosed patients of hypertension who were aware that they have hypertension and were not on any antihypertensive medication, and hypertensive patients for less than past 5 years and were on irregular treatment.

The patients were excluded from the study if they had malignant and secondary hypertension, had severe hypertension, i.e., SBP >180 mmHg and DBP >110 mmHg, pregnancy, had serum creatinine level >1.5 mg/dl, known hypersensitivity or intolerance to angiotensin-converting enzyme inhibitor and ARBs, hemodynamically significant valvular or outflow tract obstruction, uncontrolled hypertension on treatment (e.g., BP >160/100 mmHg), significant renal artery disease, hepatic dysfunction, significant gastrointestinal or neurological disorder, uncorrected volume or sodium depletion, simultaneously taking another antihypertensive medication, pregnant and lactating females, and female patients of the child-bearing age group not using medically approved contraceptives, unable to provide written informed consent, and other major non-cardiac illness expected to reduce life expectancy or significant disability interfere with study participation.

Written informed consent was taken from every patient before entry in the trial. Simple random sampling was done for the allocation of the group. For the purpose of this study, equal numbers of patients were randomly allocated equally between two groups.

Drug protocol followed in each group:

Group A (50 patients): Tablet telmisartan 40 mg orally once a day at morning time (between 8 and 10 a.m.).

Group B (50 patients): Tablet ramipril 5 mg orally once a day at morning time (between 8 and 10 a.m.)

Patients were assessed for the changes in the BP with a follow-up of over a period of 24-week. Patients were assessed at the time of screening (1st visit), then after the 1 week run-in period (2nd visit) and then after 1 month (3rd visit), 3rd month (4th visit), and 6th month (last visit). In each assessment visits, both SBP and DBP were measured in the sitting, standing, and lying position using a standardized procedure.

Patients were subjected to thorough history, clinical examination, and biochemical investigations. During screening at first visit, patients were examined completely with due consideration to medical history, family history, socioeconomic history, past history, and addiction history. Patients were examined physically to record the anthropometric measurements, body mass index, and vital signs. Systemic examination including cardiovascular system, respiratory system, central nervous system, and abdominal examination was done. Resting electrocardiogram, X-ray chest, fundus examination, laboratory examination including hemoglobin, total and differential white blood cell count, blood sugar, blood urea, serum creatinine, lipid profile, and urine examination were done. At subsequent visits, suitability of patient was assessed based on the efficacy of

drugs compliance, reporting of any adverse drug reactions, and laboratory values including serum creatinine level to continue with the trial.

Change in BP from baseline to 24 weeks of active treatment (1st, 4th, 12th, and 24th week) was analyzed statistically using SPSS program. Results were expressed as means \pm standard error of the mean. Chi-square test was applied to test the statistical significance. The confidence limit of the study was kept at 95%. Hence, a $P < 0.05$ indicated a statistically significant association.

RESULTS

The antihypertensive effect in a patient receiving telmisartan and ramipril were compared. Both telmisartan and ramipril groups were similar and comparable with regards to their systolic BP and diastolic BP. In both telmisartan and ramipril groups, there was a significant reduction of SBP, DBP, and mean BP (MBP) from beginning to the end of study ($P < 0.001$). (Table 1)

In the telmisartan-treated group, the mean SBP prior to treatment was 163.40 ± 7.31 mmHg. After 1st, 4th, and 12th week of therapy, the mean SBP was 157.24 ± 8.40 , 144.12 ± 10.12 , and 128.32 ± 5.91 mmHg, respectively. At the end of 24 weeks of therapy, the mean SBP was 127.28 ± 5.55 mmHg. In the ramipril-treated group, the mean SBP prior to treatment was 165.0 ± 8.32 mmHg. After 1st, 4th, and 12th week of therapy, the mean SBP was 158.4 ± 11.8 , 151.64 ± 11.23 , and 134.88 ± 10.02 mmHg, respectively. At the end of 24 weeks of therapy, the mean SBP was 129.56 ± 9.64 mmHg. In both telmisartan and ramipril groups, the reduction in SBP was found to be statistically significant after 1st, 4th, and 12th week of therapy when compared with the baseline readings. There was a significant difference in the SBP reduction seen between telmisartan and ramipril during the period of 4th to 12th week. No significant difference in the SBP reduction between telmisartan and ramipril was seen at the end of the study. (Table 2)

In the telmisartan-treated group, the mean DBP prior to treatment was 97.08 ± 4.98 mmHg. After 1st, 4th, and 12th week of therapy, the mean DBP was 92.52 ± 5.81 , 86.72 ± 4.53 , and 82.84 ± 3.45 mmHg, respectively. At the end of 24 weeks of therapy, the mean DBP was 82.56 ± 3.45 mmHg. In the ramipril-treated group, the mean DBP prior to treatment was 98.48 ± 4.34 mmHg. After 1st, 4th, and 12th week of therapy, the mean DBP was 93.64 ± 6.21 , 90.4 ± 5.32 , and 84.68 ± 4.75 mmHg, respectively. At the end of 24 weeks of therapy, the mean DBP was 84.08 ± 4.60 mmHg. In both telmisartan and ramipril groups, the reduction in DBP was found to be statistically significant after 1st, 4th, and 12th week of therapy when compared with the baseline readings. There was a significant difference in the DBP reduction seen

Table 1: Mean SBP, DBP, and MBP in all groups at the beginning and end of study

BP	Mean			
	Telmisartan		Ramipril	
	Initial	End of study	Initial	End of study
SBP	163.40±7.309	127.28±5.551	165.00±8.323	129.56±9.641
	33.529<0.001 (HS)		24.689<0.001 (HS)	
DBP	97.08±4.98	82.56±3.45	98.48±4.34	84.08±4.60
	18.300<0.001 (HS)		19.860<0.001 (HS)	
MBP	119.18±5.14	97.466±3.57	120.65±4.739	99.240±5.84
	27.631<0.001 (HS)		27.008<0.001 (HS)	

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MBP: Mean blood pressure

Table 2: Comparison of mean SBP between treatment groups from baseline to 1st, 4th, 12th weeks and end of the study (24th week)

Duration of treatment	Value	Telmisartan versus ramipril	
Initiation	Mean	163.40±7.309	165.00±8.323
	T value	1.030	
	P value	0.308 (NS)	
1 st week	Mean	157.24±8.407	158.40±11.822
	T value	0.559	
	P value	0.579 (NS)	
4 th week	Mean	144.12±10.121	151.64±11.239
	T value	3.362	
	P value	0.002 (S)	
12 th week	Mean	128.32±5.916	134.88±10.030
	T value	3.701	
	P value	0.001 (S)	
End of study	Mean	127.28±5.551	129.56±9.641
	T value	1.397	
	P value	0.169 (NS)	

SBP: Systolic blood pressure

between telmisartan and ramipril from the 4th week till the end of the study. (Table 3)

In the telmisartan-treated group, the average MBP prior to treatment was 119.18 ± 5.14. After 1st, 4th, and 12th week of therapy, the average MBP was 114.09 ± 5.51, 105.85 ± 5.46, and 98.04 ± 3.58 mmHg, respectively. At the end of 24 weeks of therapy, the average MBP was 97.46 ± 3.57 mmHg. In the ramipril treated group, the average MBP prior to treatment was 120.65 ± 4.739 mmHg. After 1st, 4th, and 12th week of therapy, the average MBP was 115.22 ± 6.87, 110.81 ± 6.10, and 101.02 ± 5.90, respectively. At the end of 24 weeks of therapy, the average MBP was 99.24 ± 5.84 mmHg. In both telmisartan and ramipril groups, the reduction in MBP was found to be statistically significant after 1st, 4th, and 12th week

Table 3: Comparison of mean DBP between treatment group from initiation to 1st, 4th, 12th weeks and end of the study (24th week)

Duration of treatment	Value	Telmisartan versus ramipril	
Initiation	Mean	97.08±4.98	98.48±4.34
	T value	1.522	
	P value	0.135 (NS)	
1 st week	Mean	92.52±5.81	93.64±6.21
	T value	0.934	
	P value	0.355 (NS)	
4 th week	Mean	86.72±4.53	90.4±5.32
	T value	3.735	
	P value	0.001 (S)	
12 th week	Mean	82.84±3.45	84.68±4.757
	T value	2.313	
	P value	0.025 (S)	
End of study	Mean	82.56±3.45	84.08±4.60
	T value	2.019	
	P value	0.049 (S)	

DBP: Diastolic blood pressure

Table 4: Comparison of mean MBP between treatment group from initiation to 1st, 4th, 12th weeks and end of the study (24th week)

Duration of treatment	Value	Telmisartan versus ramipril	
Initiation	Mean	119.18±5.14	120.65±4.739
	T value	1.504	
	P value	0.139 (NS)	
1 st week	Mean	114.09±5.51	115.22±6.87
	T value	0.919	
	P value	0.363 (NS)	
4 th week	Mean	105.85±5.46	110.81±6.10
	T value	4.27	
	P value	0.001 (S)	
12 th week	Mean	98.04±3.58	101.02±5.90
	T value	3.424	
	P value	0.001 (S)	
End of study	Mean	97.466±3.57	99.240±5.84
	T value	1.876	
	P value	0.067 (NS)	

MBP: Mean blood pressure

of therapy when compared with the baseline readings. There was a significant difference in the MBP reductions seen between telmisartan and ramipril during the period of 4th to 12th week. No significant difference in the MBP reduction between telmisartan and ramipril was seen at the end of the study. (Table 4)

DISCUSSION

In our study, we found that telmisartan and ramipril both are equally effective antihypertensive drugs for mild to moderate hypertension. Similar to our study where an ARB telmisartan was compared with ACE inhibitor lisinopril, telmisartan was found to be non-inferior to lisinopril in the treatment of mild to moderate hypertension.^[14] In elderly patients, telmisartan was found to be non-inferior to enalapril for the treatment of mild to moderate hypertension.^[15] No significant difference had been found between ACE inhibitors and ARBs in terms of cardiovascular outcomes or mortality.^[16]

Ramipril was found to significantly reduce the cardiovascular outcomes and mortality in high-risk patients.^[17] Among ARBs, telmisartan was found to be superior to losartan and valsartan in reducing the BP throughout the 24-h period.^[18] Due to the long duration of action, telmisartan provides BP control throughout the whole 24-h period at once a day dosing.^[19]

Our findings are in contrast to the findings of analysis^[20] where telmisartan was found to be more effective antihypertensive than ramipril. As compared to ramipril, telmisartan was found to be superior in reducing BP throughout the 24 h. Telmisartan was also found to be more effective than ramipril during the early morning blood pressure surge (EMBPS). Although in this analysis, telmisartan 80 mg once daily dose was compared with ramipril 5 or 10 mg once daily dose while in our study we compared telmisartan 40 mg once daily dose was compared with ramipril 5 mg once daily dose. In our study, we were not able to assess the effect of ramipril or telmisartan on EMBPS.

Both ARBs and ACE inhibitors block RAS, but they differ from each other in many aspects. Bradykinin, which is a vasodilator and is degraded by ACE, contributes to the antihypertensive effects of ACE inhibitors. This effect is absent in ARBs. Since complete blockade of AII production is not achieved by ACE inhibitors, direct receptor blockade by ARBs are expected to be more successful in producing the desired effect.^[21] ARBs selectively block AT1 receptors without affecting AT2 receptors while ACE inhibitors are associated with decreased activation of both AT1 receptors as well as AT2 receptors. Due to blockade of feedback inhibition, both ARBs and ACE inhibitors cause increased renin release. Increased renin levels by ARBs leads to increased AII levels which cause selective increased activation of AT2 receptors as AT1 receptors as already blocked by ARBs. Whether these differences in the ARBs and ACE inhibitors result in the therapeutic outcomes is not clear.^[12]

From 4th week onward up to the end of the study, there was a significant difference in the DBP reduction between telmisartan and the ramipril group. Similarly, from 4th week onward up to the 12th week, there was a significant difference

in the SBP and MBP reduction between telmisartan and the ramipril group. Further, studies can be planned to find out the rationale behind these findings.

CONCLUSION

Telmisartan and ramipril both are equally effective as antihypertensive agent in mild to moderate hypertension. Although further studies can be planned to find out the rationale behind the greater reduction in DBP from 4th week onward and in SBP and MBP for the period between 4th and 12th week with telmisartan than ramipril.

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