ORIGINAL ARTICLE

A comparative study on the pleiotropic effects of Olmesartan and Telmisartan in hypertensive patients with type 2 diabetes mellitus: A randomized, prospective, open labelled, hospital based study

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Abstract

Background: Hypertension and Diabetes Mellitus (DM) are common non-communicable diseases with increasing incidence worldwide. Angiotensin Receptor Blockers (ARBs) have shown to exhibit various pleiotropic effects. *Aim and Objectives:* To compare the pleiotropic effects of Olmesartan and Telmisartan in hypertensive patients with type 2 DM. *Material and Methods:* A randomized, prospective, open labelled, parallel group, hospital based study was conducted among 66 hypertensive patients with type 2 DM. Patients were randomized into 2 groups (Olmesartan, Telmisartan) of 33 each. Baseline metabolic & renal parameters were recorded and reassessed after 12 weeks. *Results:* Statistically significant reduction in HbA1c (Olmesartan, $p = 0.005$; Telmisartan $p < 0.001$) was seen in both the drugs. Telmisartan showed significant decrease in Total Cholesterol (TC), Low-density Lipoprotein Cholesterol (LDL-C) and statistically significant increase in High-density Lipoprotein Cholesterol (HDL-C) ($p = <0.001$, 0.001, 0.032 respectively). Olmesartan showed significant reduction in Urine Albumin-to-Creatinine Ratio (UACR) ($p = 0.02$). However, when both the groups were compared, none of the parameters showed statistical significance. *Conclusion:* Lipid and glucose parameters improved better with Telmisartan, whereas Olmesartan showed better renoprotection. Physicians can choose preferred ARB depending upon various associate conditions.

Keywords: Hypertension, Diabetes Mellitus, Pleiotropic, Telmisartan, Olmesartan

Introduction

Modernization of lifestyle, leading to adoption of harmful practices such as smoking, unhealthy diet, and reduced physical activity, is leading to overweight/obesity, elevated blood sugar levels, and increased blood pressure [1]. Hypertension (HTN) and Diabetes Mellitus (DM) are common non-communicable diseases with their incidence increasing worldwide. They frequently co-exist in many individuals. HTN is two times more common in patients with DM than those who are not. Patients with DM more commonly present with isolated systolic hypertension. They are also more resistant to treatment [2]. Raised blood pressure and hyperglycaemia are independent risk factors for cardiovascular, cerebrovascular, and renal disease, resulting in increased morbidity and mortality [3-4]. Both serve as potential risk factors for one another, and each pathophysiological disease entity exacerbate the other. The activation of Renin-Angiotensin- Aldosterone System (RAAS) plays a pivotal role in pathogenesis of HTN and DM. It causes vasoconstriction, salt, and water retention; which increases blood pressure. Angiotensin II decreases the pancreatic blood flow, which impairs the insulin secretion. It also causes insulin resistance by interfering with insulin

signalling pathway [5]. In addition, chronic activation of the RAAS leads to progression of atherosclerosis, causing micro and macrovascular complications [6]. Hence early intervention with agents that decrease RAAS activation helps in reducing complications. So, the drugs that block RAAS are the first line drugs, in the management of HTN with T2DM [7]. Angiotensin Receptor Blockers (ARBs) are a unique class of antihypertensive agents that selectively block Angiotensin II type 1 (AT1) receptors, thereby inhibit RAAS and subsequently lowers the blood pressure [8]. ARBs have shown to exhibit pleiotropic effects like antioxidant, antiplatelet, antiatherogenic, antifibrillatory, antiproteinuric and hypouricemic actions [9-10]. However, all ARBs do not show similar effect on metabolic and renal parameters. Olmesartan has shown to delay or prevent microalbuminuria in type 2 diabetes patients [11], whereas Telmisartan improves insulin resistance by partial activation of the Peroxisome Proliferator-Activated Receptor gamma (PPAR-γ) [12-13].

To our best knowledge, only one study was conducted in Indian population comparing the pleiotropic effects of Telmisartan and Olmesartan in hypertensive patients with Metabolic Syndrome (MetS) where Telmisartan was shown to be more effective [14]. In our study, we included newly diagnosed hypertensive patients with T2DM.

The objectives of the study were to study and compare the effects of Olmesartan and Telmisartan on lipid profile, Fasting Blood Sugar (FBS), Glycated Hemoglobin (HbA1c) and renal parameters; and to assess the efficacy of Olmesartan and Telmisartan in reduction of blood pressure.

Material and Methods

This was a randomized, prospective, open labelled, parallel group, hospital based study conducted at GSL Medical College and General Hospital, Rajamahendravaram, Andhra Pradesh. Study was carried out for a period of 6 months from January 2017 to June 2017. Study participants were recruited from those attending Outpatient Department (OPD) of General Medicine. Institutional Ethics Committee approval was obtained. All the study participants were explained about the nature, purpose, procedure of the study and anticipated risks and benefits. Informed consent was taken from each participant prior to the commencement of the study.

Newly diagnosed hypertensive patients (SBP between 140-159 mm of Hg and DBP 90-99 mm of Hg), aged between 18 to 60 years, with associated T2DM of < 1year duration, on Metformin 500 mg OD; were included in the study. Systolic Blood Pressure (SBP) ⩾ 160 mm of Hg and/or Diastolic Blood Pressure (DBP) ⩾ 100 mm of Hg, secondary hypertensive patients, HbA1c > 7, pregnant and lactating mothers, serum creatinine above 2 mg/dl, diabetic retinopathy, diabetic neuropathy, patients with recent MI and severe hepatic disease were excluded.

Demographic data and laboratory investigations [FBS, HbA1c, fasting lipid profile, serum creatinine and Urine Albumin-to-Creatinine Ratio (UACR)] were recorded in case record form. Body Mass Index (BMI) was calculated using the formula, (BMI = weight in kg/height in square meters). Blood pressure was recorded using mercury sphygmomanometer.

Sample size was calculated using statistical superiority design, the formula:

$$N = 2 \times \left(\frac{Z_{1-\frac{\alpha}{2}} + Z_{1-\beta}}{\delta} \right)^2 \times s^2$$

Sample size of 30 was required in each group to get an expected mean difference (δ) of 0.5 in HbA1c% where type 1 error/ α = 0.05; type II error/ β = 0.20; S = 0.7% Standard Deviation (SD) obtained from previous study [15]. An additional three participants were added to each group to account for any potential dropouts or lost-to-follow-up subjects during the study. A total of 66 participants were randomly divided into two groups of 33 each by computer generated list of random numbers. One group received Tab Olmesartan 20 mg and the other received Tab Telmisartan 40 mg. Study participants were instructed to take the drug once a day in the morning after breakfast. Clinical & biochemical parameters were re-evaluated at the end of 12th week. Serious adverse drug reactions if any, reported by the patients or observed by the investigator were recorded.

Statistical analysis was performed by using Statistical Package for Social Sciences (SPSS) version 20.0 (SPSS Inc, Chicago, Illinois, USA) and MS-Excel 2010 (Microsoft corporation, Washington DC, USA). All the descriptive statistics were presented in the form of Mean ± SD and percentages. Unpaired student t-test was used to compare the means of two independent groups i.e., between Telmisartan and Olmesartan; paired t-test to compare the means within the group i.e., before and after treatment. Value of $p < 0.05$ was considered statistically significant.

Results

One-hundred-sixty-eight T2DM patients who were newly diagnosed with hypertension were screened. One-hundred-two patients who did not satisfy the inclusion and exclusion criteria were excluded from the study. A total of 66 participants were randomly divided into two groups of 33 each. Three patients in Olmesartan group and 4 patients in Telmisartan group were lost to follow up. Upon enquiry, two participants in each group relocated, two in the Telmisartan group sought treatment elsewhere, and one in the Olmesartan group did not respond (Figure 1).

The mean age in years of study participants was 47.97 ± 7.38 in Olmesartan group and 47.38 ± 6.44 in Telmisartan group. Mean BMI was 26.5 ± 2.10 kg/m^2 and 26.72 ± 2.26 kg/m^2 in Olmesartan and Telmisartan groups respectively. The baseline demographic and clinical characteristics were depicted in Table 1. At baseline, there was no statistically significant difference between the groups for all the parameters, except for HbA1c (p = 0.012). At the end of the 12th week, the mean decrease in BMI from baseline in the Olmesartan group was 0.8 kg/m², and 1.72 kg/m² in Telmisartan group. These changes were statistically significant in both groups. There was a statistically significant decrease in SBP pressure from baseline, in both the study groups; (Olmesartan: 23 mm of Hg and Telmisartan: 19.79 mm of Hg) Similarly, both the groups exhibited a statistically significant decrease in the mean DBP; (Olmesartan: 15.20 mm of Hg and Telmisartan: 13.58 mm of Hg).

Only Telmisartan showed statistically significant decrease in mean FBS ($p < 0.001$). Significant decrease in mean HbA1c was seen with both the drugs (Olmesartan 0.11%, and Telmisartan 0.3%).

Regarding renal parameters, the Olmesartan group showed a statistically significant decrease in the mean serum creatinine (p = 0.016), which was not observed in the Telmisartan group. Similar results were seen for the UACR, where only Olmesartan (p = 0.02) showed statistically significant decrease in mean values.

Change in lipid parameters from base line to 12th week were shown in Table 2. In Olmesartan group, only HDL-C showed statistically significant increase (p = 0.006), whereas, in Telmisartan group, TC, LDL-C, HDL-C, showed statistical significance (p = < 0.001, 0.001, 0.032 respectively), where TC, LDL-C values were decreased and HDL-C value increased. No serious adverse events were found in both the study groups. At the end of 12th week, when the mean difference of both the groups were compared, none of the parameters showed statistical significance.

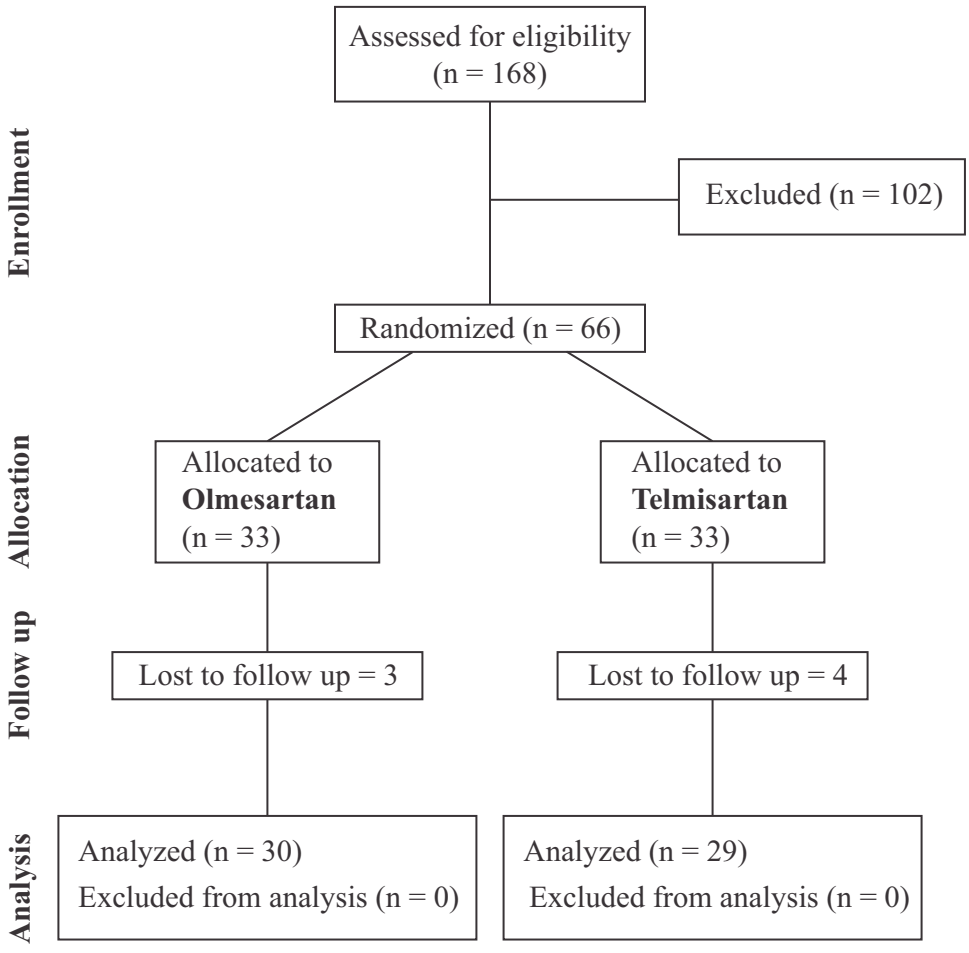

Figure 1: Concert diagram showing the flow of participants through each stage of randomized trial

Table 1: Baseline demographic and clinical characteristics

Variables		Olmesartan (n=30)	Telmisartan (n=29)	*p*
Male n (%)		19 (63.3)	17 (58.6)	
Female n (%)		11 (36.6)	12 (41.4)	
Age (Years) Mean ± SD		47.97 ± 7.38	47.38 ± 6.44	0.746
BMI (kg/m^2)		26.5 ± 2.10	26.72 ± 2.26	0.783
Mean duration of DM in month		7.0 ± 2.37	6.76 ± 1.59	0.650
SBP (mm of Hg)		151 ± 8.03	150.28 ± 7.45	0.721
DBP (mm of Hg)		94.07 ± 6.87	93.45 ± 7.13	0.736
FBS (mg/dl)		118.93 ± 13.62	120.79 ± 13.96	0.607
HbA1c (%)		6.22 ± 0.88	6.74 ± 0.53	0.012
Serum Creatinine (mg/dl)		1.04 ± 0.39	1.01 ± 0.37	0.72
UACR (mg/gm)		30.10 ± 13.24	30.76 ± 14.66	0.857
Lipid Profile	**TC (mg/dl)**	189.16 ± 30.03	200.31 ± 22.09	0.111
	LDL (mg/dl)	113.86 ± 19.88	119.17 ± 14.64	0.249
	HDL (mg/dl)	42.93 ± 6.55	43.44 ± 7.89	0.786
	TG (mg/dl)	159.53 ± 43.48	159.41 ± 45.86	0.992
	VLDL (mg/dl)	38.23 ± 15.70	41.17 ± 16.84	0.491

BMI: Body Mass Index, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure , FBS: Fasting Blood Sugar, HbA1c: Gylcated Hemoglobin, UACR: Urine Albumin-To-Creatinine Ratio, TC: Total Cholesterol, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, TG: Triglycerides, VLDL: Very Low-Density Lipoprotein.

Table 2: Change in the study parameters over a period of 12 weeks

Variable		Olmesartan				Telmisartan				Difference between the two groups at 12 weeks	
		Baseline (Mean ± SD)	12 weeks (Mean ± SD)	Mean difference (95%CI)	*p*	Baseline (Mean ± SD)	12 weeks (Mean ± SD)	Mean difference (95% CI)	*p*	Mean difference (95% CI)	*p*
BMI [Kg/m^2]		26.57 ± 2.10	25.77 ± 2.04	0.8 (0.36 to 1.24)	0.001**	26.7 ± 2.26	25 ± 2.18	1.72 (1.10 to 2.35)	<0.001***	0.77 (-0.34 to 1.87)	0.17
SBP		151 ± 8.03	128 ± 10.89	23 (19.96 to 26.03)	<0.001***	150.28 ± 7.45	130.48 ± 5.72	19.79 (16.88 to 22.7)	<0.001***	-2.48 (-7.04 to 2.07)	0.28
DBP		94.07 ± 6.87	78.87 ± 5.39	15.20 (12.41 to 17.98)	<0.001***	93.45 ± 7.13	79.86 ± 4.60	13.58 (10.90 to 16.26)	<0.001***	-0.99 (-3.62 to 1.63)	0.45
FBS		118.93 ± 13.62	117.30 ± 14.63	1.90 (0.04 to 3.84)	0.055	120.79 ± 13.96	113.90 ± 13.81	6.89 (3.30 to 10.48)	<0.001***	3.13 (-4.28 to 10.56)	0.40
HbA1c		6.22 ± 0.88	6.11 ± 0.82	0.11 (0.036 to 0.184)	0.005**	6.71 ± 0.53	6.41 ± 0.48	0.3 (0.184 to 0.423)	<0.001***	-0.30 (-0.65 to 0.05)	0.094
Sr Creatinine		1.04 ± 0.39	0.93 ± 0.24	0.117 (0.024 to 0.210)	0.016*	1.01 ± 0.37	0.990 ± 0.34	0.01 (-0.07 to 0.09)	0.726	-0.066 (-0.22 to 0.087)	0.391
UACR (mg/gm)		30.10 ± 13.24	24.47 ± 5.34	5.63 (0.83 to 10.435)	0.023*	30.76 ± 14.66	25.66 ± 5.23	5.103 (0.089 to 10.118)	0.046*	-1.189 (-3.95 to 1.571)	0.392
Lipid profile	**TC**	189.16 ± 30.03	188.47 ± 24.90	0.70 (-6.74 to 8.14)	0.849	200.31 ± 22.09	180.10 ± 25.41	20.20 (13.02 to 27.39)	<0.001**	8.36 (-4.75 to 21.48)	0.21
	LDL	113.87 ± 19.88	112.37 ± 19.53	1.50 (-2.38 to 5.38)	0.436	119.17 ± 14.64	113.14 ± 17.25	6.03 (2.82 to 9.24)	0.001**	-0.77 (-10.38 to 8.84)	0.87
	HDL	42.93 ± 6.56	45.83 ± 9.07	-2.90 (-4.91 to -0.88)	0.006**	43.44 ± 7.89	48.72 ± 12.19	-5.27 (-10.05 to -0.50)	0.032*	-2.89 (-8.48 to 2.69)	0.30
	TG	159.53 ± 43.48	160.53 ± 36.92	-1.0 (-7.91 to 5.91)	0.769	159.41 ± 45.86	156.41 ± 34.98	3.0 (-6.16 to 12.16)	0.508	4.11 (-14.64 to 22.88)	0.66
	VLDL	38.23 ± 15.70	38.77 ± 11.04	-0.53 (-4.63 to 3.56)	0.792	41.17 ± 16.84	38.37 ± 12.37	2.79 (-1.54 to 7.12)	0.20	0.39 (-5.72 to 6.49)	0.89

BMI: Body Mass Index, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, FBS: Fasting Blood Sugar, HbA1c: Gylcated Hemoglobin, UACR: Urine Albumin-To-Creatinine Ratio, TC: Total Cholesterol, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, TG: Triglycerides, VLDL: Very Low-Density Lipoprotein.

*$p < 0.05$, ** $p < 0.01$, *** $p < 0.001$*

Discussion

According to the Eighth Joint National Committee (JNC 8) guidelines, angiotensin receptor blockers are one of the first line drugs for hypertension [16]. Both Olmesartan and Telmisartan, showed statistically significant decrease in systolic as well as diastolic blood pressure ($p < 0.001$). When both the drugs were compared, the difference was not statistically significant. Studies conducted by Arao *et al*. and Uday *et al*., showed similar results [17-18]. All ARBs improve insulin resistance by their inhibitory effects on AT1 receptors [19]. In our study there was a statistically significant reduction in HbA1c with both the groups (Olmesartan p=0.005, Telmisartan $p < 0.001$). ARBs have shown to prevent the new-onset diabetes more effectively than other class of anti-hypertensives [18]. The mean reduction in HbA1c was more with Telmisartan group compared to Olmesartan (0.3 vs 0.11) which may be due to the additional PPAR-γ–activating effect of Telmisartan improving insulin sensitivity [20-21]. But the difference was not statistically significant ($p = 0.094$)

Angiotensin II enhances the production of Endothelin-1 (ET-1), which is a potent endothelial growth-promoting and vasoconstrictor peptide. This has been shown to play a key role in promoting proteinuria [21]. ARBs combine with AT1receptors and block the action of angiotensin II, which helps in reducing urinary albumin excretion [23]. Statistically significant decrease in serum creatinine ($p = 0.016$) and UACR ($p = 0.023$) was seen only with Olmesartan group. This is in line with other studies which showed the same [11, 24]. Olmesartan has a double-chained domain consisting of carboxyl and hydroxyl groups, which has high affinity with the AT1 compared to other ARBs; thereby providing better renoprotection [23].

Patients with diabetes and hypertension frequently have dyslipidaemia, which is an important risk factor for cardiovascular disease. In Olmesartan group, HDL-C showed statistically significant increase ($p = 0.006$) after treatment. Statistical significance was not seen with other lipid parameters. This is similar to the study conducted by Arao *et al*. where a significant increase of HDL-C was seen with Olmesartan treatment [17]. However, a study conducted by Fliser *et al*. showed no significant changes in all the lipid parameters after 6 weeks of Olmesartan administration [25]. This might be due to the difference in the study duration. In the Telmisartan group, there was a statistically significant decrease in TC ($p < 0.001$) and LDL-C ($p = 0.001$), while HDL-C ($p = 0.032$) exhibited a statistically significant increase. Various other studies also showed favourable effect of Telmisartan on lipid profile [26-30]. Telmisartan may have hepatic partial PPAR-α agonist activity which increases lipoprotein lipase expression. This study has some potential limitations. It was an open-label study, increasing the risk of bias. The study was also conducted in a small population and the duration of the study was relatively short.

Conclusion

Telmisartan showed more favourable effects on lipid and glucose metabolism. Olmesartan showed better renoprotection. However, both the drugs were effective in decreasing blood pressure. Based

on this study results, clinicians may prefer Telmisartan in hypertensive patients having dyslipidaemias and hyperglycaemia; whereas Olmesartan may be preferred inpatients with abnormal renal parameters.

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