A COMPARATIVE STUDY OF EFFICACY AND SAFETY OF TAMSULOSIN AND SILODOSIN IN TREATMENT OF LOWER URINARY TRACT SYMPTOMS ASSOCIATED WITH BENIGN PROSTATIC HYPERPLASIA

Amulya F. Yaraguppi[1], Ramesh H[2], Ravikumar Jadav[3]

[1]Postgraduate Student, Department of Pharmacology, KIMS, Hubli, Karnataka, India.
[2]Associate Professor, Department of Pharmacology, KIMS, Hubli, Karnataka, India.
[3]Associate Professor, Department of Urology, KIMS, Hubli, Karnataka, India.

ABSTRACT

BACKGROUND

Agents with a high degree of selectivity for α_{1A} receptors have beneficial effects on the symptoms associated with benign prostatic hyperplasia (BPH) and less effect on blood pressure. The α_1 antagonists generally preferred in the management of BPH are Tamsulosin and Silodosin because of their minimal hemodynamic adverse effects. There have been only few comparative studies between these two drugs. Therefore, this study was undertaken to compare efficacy of silodosin and tamsulosin in treatment of lower urinary tract symptoms (LUTS) associated with BPH.

MATERIALS AND METHODS

A total of 70 newly diagnosed patients suffering from LUTS associated with BPH were randomly divided into two groups with 35 patients in each group. Tamsulosin group received Tamsulosin 0.4 mg orally once daily for 12 weeks and Silodosin group received Silodosin 8 mg orally once daily for 12 weeks. Assessment of efficacy was done by assessing improvement in the International Prostate Symptom Score (IPSS), Urinary Flow Rate (Qmax) and assessment of Quality of Life (QOL) at base line, 4 weeks and 12 weeks.

RESULTS

The changes in IPSS total score, Qmax and QOL were significant within the groups but not between the groups

CONCLUSION

Both the drugs are equally effective in treatment of LUTS associated with BPH.

KEY WORDS

Tamsulosin, Silodosin, LUTS, BPH, QOL.

HOW TO CITE THIS ARTICLE: Yaraguppi AF, Ramesh H, Jadav R. A comparative study of efficacy and safety of tamsulosin and silodosin in treatment of lower urinary tract symptoms associated with benign prostatic hyperplasia. J. Evolution Med. Dent. Sci. 2019;8(02):146-151, DOI: 10.14260/jemds/2019/32

BACKGROUND

Benign Prostatic Hyperplasia (BPH) is one of the most common diseases in men, with an increasing prevalence rate with age.[1] It clinically manifests as lower urinary tract symptoms (LUTS). BPH usually starts in men in their 50s, by the age of 60 years, 50% of men have histological evidence of BPH and 80% of men in their 70s suffer from BPH-related LUTS [2]. LUTS can be classified into three categories: voiding symptoms (Hesitancy, weak/slow stream, intermittency, straining, and incomplete voiding), storage symptoms (Frequency, urgency, nocturia, urge incontinence) and postmicturition symptoms (Postvoid dribbling), which may adversely affect the quality of life (QOL).[1] In addition, BPH can also lead to more serious complications such as acute urinary retention, recurrent urinary tract infections, hematuria, bladder calculi, and renal dysfunction. Histologically, BPH is characterized by a progressive increase in the number of epithelial and stromal cells that develop initially in the periurethral area of the prostate gland with increased prostatic smooth muscle tone resulting in urethral constriction and outflow obstruction.[3] Pathophysiologically, BPH related LUTS results not only from fixed mechanical obstruction of the prostatic urethra but also from a dynamic component to the obstruction from prostatic muscle activity.[4] Current strategies for treating men with LUTS associated with BPH depend on the severity of the symptoms and include watchful waiting, pharmacological management, minimally invasive therapies and surgery.[1] Medical therapy is indicated for patients with uncomplicated BPH, those with mild-to-moderate symptoms, awaiting surgery or unwilling or unsuitable to undergo surgery.[5] The definitive management of symptomatic BPH is surgery. The major goals of BPH treatment should include improvement in symptom scores, patient-reported quality of life, lowering the risk of disease progression and need for further surgical interventions.[6] Thus, there is a need for continued research on drug treatment for symptomatic BPH. Over the last decade, the incidence of surgery has declined in almost all countries and the incidence of medical treatment is rising.[5]

The pharmacotherapy of BPH comprises of α_1 antagonists, 5α-reductase inhibitors, phytotherapy, gonadotrophin releasing hormone analogues and androgen receptor blockers.[1] Treatment for BPH aims to relieve two

'Financial or Other Competing Interest': None.
Submission 18-12-2018, Peer Review 02-01-2019,
Acceptance 04-01-2019, Published 14-01-2019.
Corresponding Author:
Dr. Ramesh H,
Associate Professor,
Department of Pharmacology,
KIMS, Hubli, Karnataka,
India.
E-mail: kimsramesh@yahoo.co.in
DOI: 10.14260/jemds/2019/32

types of urinary tract obstruction: mechanical urinary tract obstruction caused by tissue compression due to an enlarged prostate and functional urinary tract obstruction caused by constriction of the urinary tract and prostatic smooth muscle via sympathetic α_1 receptor.[7] Currently α_1 receptor blockers and 5α-reductase inhibitors are two main categories of drugs are used for the treatment of symptomatic BPH. The former addressing the dynamic component by relaxing the smooth muscles of prostate and prostatic urethra, and the latter affecting the static component acts more slowly restricting the hyperplasia. Over the past 20 years, α_1 antagonists have become the primary first-line therapy for LUTS associated with BPH. There are several subtypes of α1-receptors which include α_{1A}, α_{1B}, and α_{1D}. The α_{1A} receptors, predominantly located in the smooth muscles of genitourinary tract, are the primary regulators of smooth muscle tone in the bladder neck and prostate, α_{1B} receptors are present in the vascular smooth muscle and regulate the vascular tone, and α_{1D} subtype mediates contraction of the bladder muscle.[8] Early α_1 antagonists were non-selective for subtypes and were associated with blood pressure-related adverse effects, such as orthostatic hypotension[1] Agents with a high degree of selectivity for α_{1A} receptors have beneficial effects on the symptoms associated with BPH and less effect on blood pressure.[9,10] The α_1 antagonists preferred in the management of BPH are Tamsulosin and Silodosin because of their minimal hemodynamic adverse effects.[11] Tamsulosin has relative selectivity for the α_{1A} and α_{1D} subtypes. Silodosin is a highly selective α_{1A} receptor antagonist. Even though many studies established the efficacy of α_1 antagonists Alfuzosin, Doxazosin, Tamsulosin and Terazosin, there have been only few comparative studies between Silodosin and Tamsulosin. Therefore, this study was undertaken to compare efficacy of Silodosin and Tamsulosin in treatment of LUTS associated with BPH.

MATERIALS AND METHODS

Sample Size[4]

The minimum sample size required was 32 patients in each group. This was calculated to detect a difference of 4 in total IPSS between the groups with 80% power and 0.05 probability of type 1 error, assuming standard deviation of 5 in total IPSS and allowing 20% dropout rate.

Study Design

Randomized, open label, parallel, comparative study.

Place of Study

Department of Urology, KIMS, Hubballi.

Study Period

January 2015 to June 2016.

Inclusion Criteria

1. Age greater than or equal to 50 years.
2. Peak urinary flow rate at least 4 ml/sec but not greater than 15 ml/sec and voided volume is at least 150 ml.
3. International Prostate Symptom Score of 8 or higher associated with moderate to severe symptoms.
4. Serum prostate specific antigen level (PSA) - 1.5 to 4 ng/ml.
5. QOL score ≥3.

Exclusion Criteria

1. Hypersensitivity to the active substance or to any of the excipients.
2. Patients for whom cataract surgery is scheduled.
3. Patients with supine blood pressure less than 90/70 mm of Hg.
4. Moderate or severe renal impairment.
5. Severe hepatic impairment.
6. Concomitant use of other α antagonists or natural/herbal products known to have an effect on LUTS.
7. Concomitant use with potent cytochrome P450 3A4 inhibitors, (possible pharmacokinetic interaction).
8. Prostate cancer.
9. History of prostate or bladder neck surgery.
10. Active urinary tract infection.

Methodology

Ethical clearance was obtained from ethical committee KIMS, Hubballi. A total of 70 patients, newly diagnosed to be suffering from LUTS associated with BPH, found eligible were enrolled in the study. Randomisation of the subjects was done in 1:1 ratio, into two study groups (Silodosin group and Tamsulosin group) by computer generated random numbers with 35 subjects in each group. The severity of LUTS was assessed by the International Prostate Symptom Score (IPSS), based on answers to seven questions regarding urinary symptoms and one question regarding quality of life.

Questionnaire Regarding Urinary Symptoms (Answer Score 0 to 5)-

1. **Incomplete Emptying:** Over the past month, how often have you had the sensation of not emptying your bladder completely after you finished urinating
2. **Frequency:** Over the past month, how often have you had to urinate<2 hours after you finished urinating?
3. **Intermittency:** Over the past month, how often have you found, you stopped and started again several times when you urinated?
4. **Urgency:** Over the past month, how often have you found it difficult to postpone urination?
5. **Weak Stream:** Over the past month, how often have you had a weak urinary stream?
6. **Straining:** Over the past month, how often have you had to push or strain to begin urination? (Scores: 0 - not at all; 1 - less than one in five times; 2 - less than half the time; 3 - about half the time; 4 - more than half the time; 5 - almost always)
7. **Nocturia:** Over the past month, how many times did you get up to urinate from the time you went to bed at night until the time you got up in the morning? (Scores: 0 - none; 1 - one time; 2 - two times; 3 - three times; 4 - four times; 5 - five times) (Total IPSS score: 1-7: mild; 8-19: moderate; 20-35: severe)
8. **Quality of Life:** If you were to spend the rest of your life with your urinary condition just the way it is now, how would you feel about that? (Scores: 0 - delighted; 1 - pleased; 2 - mostly satisfied; 3 - mixed-about equally satisfied and dissatisfied; 4 - mostly dissatisfied; 5 - unhappy; 6 - terrible).[12]

This Study required following Baseline Investigations on the Patients –

1. Urine routine to exclude urinary tract infection
2. Uroflowmetry to measure flow rates
3. Ultrasonography of abdomen and pelvis done for measuring prostate volume and postvoid residual urine
4. Serum PSA to exclude prostate carcinoma.

Assessment of Efficacy

Tamsulosin Group

Patients in this group were treated with Tamsulosin 0.4 mg once daily, orally for 12 weeks.

Silodosin Group

Patients in this group were treated with Silodosin 8 mg once daily, orally for 12 weeks.

Subjects were evaluated at baseline (week 0), week 4, and week 12. The IPSS, uroflowmetry, ultrasonography was done at the base line, at 4 and 8 weeks of the follow up to assess the clinical improvement.

Assessment of efficacy was done by evaluating primary outcome/the change in BPH symptoms was assessed by changes in ipss. 2. Secondary outcome

a. Change in urinary flow rate measured by change from baseline in peak urinary flow rate (Qmax).
b. Improvement in voiding and storage symptoms by change in IPSS voiding and storage sub scores.
c. Change from baseline in QOL due to urinary symptoms.

Statistical Analysis

Demographic data and baseline measurements were used in group differences and were tested using Fisher's exact test & Paired 't' test Independent sample t-test, respectively. The software used was Sigma Stat 3.5. P value < 0.001 was taken as significant.

RESULTS

In this study total of 70 patients were enrolled and divided into two groups (Tamsulosin and Silodosin group) of 35 each. 3 patients in silodosin group and 1 patient in tamsulosin group were lost for follow up. Thus, data of 66 patients – 32 on silodosin and 34 on tamsulosin were analysed-

- There was no relevant statistical difference between the treatment groups in terms of demographic changes as the being 65.25 in the silodosin group and 65.29 in the tamsulosin group (Table 1).
- There was no relevant statistical difference between the treatment groups in terms of duration of symptoms as the being 34.06 in the silodosin group and 33.15 in the tamsulosin group (Table 2).
- The changes in IPSS total score from baseline till the end of 12 weeks were highly significant within the groups (<0.05) but not between the two groups (Table 3).
- The changes in IPSS sub scores were significant in all parameters within the groups (p<0.001), but not between the groups (Table 4).
- The changes in QOL from baseline till end of 12 weeks were highly significant within the groups (p<0.001) but not between the two groups (Table 5).
- The changes in PVRU (Post void residual urine volume) from baseline till end of 12 weeks were highly significant within the groups (p<0.001) but not between the two groups (Table 6)
- There was no statistically significant changes in the prostate volume measured by ultrasonography between the two groups (Table 7).
- The changes in Qmax was significant within the groups(p<0.01) but was not significant in between two groups (Table 8)

Parameter		Silodosin, n = 32	Tamsulosin, n = 34	p-Value
Age (Years)	Range	50 – 82	52 - 80	-
	Mean ± SD	65.25 ± 8.14	65.29 ± 8.53	0.983
Table 1. Comparison of Age Distribution Between the Study Groups				

Parameter	Silodosin, n = 32	Tamsulosin, n = 34	P value
Duration of Symptoms (Months)	4 - 54	3 - 54	-
	34.06 ± 14.51	33.15 ± 13.85	
	42(24 - 42)	36 (30 - 42)	0.502
Table 2. Comparison of Duration of Symptoms Between the Study Groups			

IPSS – Total Score	Silodosin	Tamsulosin	P value Between Groups
Baseline	26.78 ± 5.92	29.00 ± 2.52	0.05
Week 4	15.19 ± 4.45	16.5 ± 2.54	0.143
Week 12	12.19 ± 4.28	13.35 ± 2.64	0.185
% Change (B -12W)	54.48	53.97	
Table 3. Comparison of IPSS - Total Score Changes Between the Study Groups			
IPSS=International Prostate Symptom Score, IQR=Interquartile range, SD=Standard deviation. There was significant decline in IPSS scores over the 12-week study period in both treatment groups (P < 0.001) by two-way analysis of variance).			

IPSS		Silodosin		Tamsulosin		p-Value Between Groups
		Median (IQR)	Mean±SD	Median (IQR)	Mean±SD	
Q1	B	5 (3 - 5)	4.13 ± 1.129	5 (4 - 5)	4.65 ± 0.485	0.088
	4W	2 (1 - 2)	1.91 ± 0.734	2 (2 - 2)	2.00 ± 0.651	0.567
	12W	2 (1 - 2)	1.69 ± 0.693	2 (2 - 2)	2.00 ± 0.650	0.057
Q2	B	4 (3 - 5)	3.94 ± 1.014	4 (4 - 5)	4.26 ± 0.567	0.230
	4W	3 (1 - 4)	2.5 ± 1.244	3 (2 - 3)	2.62 ± 0.697	0.835
	12W	2 (1 – 3.75)	2.38 ± 1.212	2 (2 - 3)	2.21 ± 0.845	0.589
Q3	B	5 (3.25 - 5)	4.16 ± 1.081	4 (4 - 5)	4.32 ± 0.589	0.911
	4W	4 (2.25 - 4)	3.16 ± 1.081	3 (3 - 4)	3.32 ± 0.589	0.900
	12W	3 (1.25 - 3)	2.16 ± 1.081	2 (2 - 3)	2.32 ± 0.590	0.910
Q4	B	5 (4 - 5)	4.25 ± 1.078	5 (4 - 5)	4.50 ± 0.615	0.570
	4W	1 (1 - 2)	1.3 ± 0.592	1 (1 - 2)	1.50 ± 0.564	0.172
	12W	1 (1 - 2)	1.28 ± 0.581	1 (1 - 1)	1.06 ± 0.422	0.089
Q5	B	3 (2 - 4)	3.09 ± 1.118	4 (3 - 4)	3.53 ± 0.662	0.085
	4W	2 (1 - 3)	2.09 ± 1.118	3 (2 - 3)	2.53 ± 0.660	0.085
	12W	1 (0.0 - 2)	1.16 ± 1.019	2 (1 - 2)	1.53 ± 0.662	0.089
Q6	B	3 (2 - 4)	2.84 ± 1.110	3 (2.75 - 4)	3.21 ± 0.914	0.182
	4W	2 (1 - 3)	1.88 ± 1.040	2 (1.75 - 3)	2.21 ± 0.910	0.182
	12W	1 (0.0 - 2)	0.94 ± 0.948	1 (0.75 - 2)	1.21 ± 0.914	0.217
Q7	B	4 (3.25 - 5)	4.34 ± 1.066	5 (4 - 6)	4.82 ± 1.080	0.059
	4W	2 (1.25 - 3)	2.34 ± 1.066	3 (2 - 4)	2.82 ± 1.086	0.059
	12W	3 (2 - 3)	2.59 ± 0.946	3 (2 - 4)	1.058 ± 1.058	0.056

Table 4. Comparison of IPSS - Sub Score Changes Between the Study Groups

IQR=Interquartile range, SD=Standard deviation. There was significant decline in IPSS sub scores over the 12-week study period in both treatment groups (Values depict median (IQR).

QOL		Silodosin	Tamsulosin	p-Value Between Groups
Baseline	Mean ± SD	4.22 ± 0.910	4.35 ± 0.810	
	Median (IQR)	4 (3.25 - 5)	4 (4 - 5)	0.590
Week 4	Mean ± SD	2.22 ± 0.906	2.35 ± 0.812	
	Median (IQR)	2 (1.25 - 3)	2 (2 - 3)	0.600
Week 12	Mean ± SD	1.22 ± 0.900	1.35 ± 0.800	
	Median (IQR)	1 (0.25 - 2)	1 (1 - 2)	0.590
% Change (B – 12W)		71.10	68.97	

Table 5. Comparison of Quality of Life Changes Between the Study Groups

IQR=Interquartile range, SD=Standard deviation. There was significant decline in QOL over the 12-week study period in both treatments.

PVRU		Silodosin	Tamsulosin	P value Between Groups
Baseline	Mean ± SD	46.88 ± 22.781	45.00 ± 20.226	
	Median (IQR)	45 (30 - 60)	45 (30 - 65)	0.884
Week 4	Mean ± SD	23.63 ± 7.594	23.00 ± 6.742	
	Median (IQR)	23 (18 - 28)	23 (18 - 28)	0.880
Week 12	Mean ± SD	15.60 ± 7.590	15.00 ± 6.700	
	Median (IQR)	15 (10 - 20)	15 (10 - 20)	0.880
% Change (B – 12W)		66.72	66.67	

Table 6. Comparison of Post Void Residual Urine Volume Changes Between the Study Groups

IQR=Interquartile range, SD=Standard deviation. There was significant decline in PVRU over the 12-week study period in both treatments.

Prostate Volume		Silodosin	Tamsulosin	P value (Between Groups)
Baseline	Mean ± SD	62.66 ± 16.61	58.08 ± 14.19	
	Median (IQR)	65 (50 - 75)	57.5 (45 - 65)	0.161
Week 4	Mean ± SD	60.78 ± 15.5	58.09 ± 14.20	
	Median (IQR)	60 (50 - 75)	57.5 (45 - 65)	0.304
Week 12	Mean ± SD	61.72 ± 17.16	58.09 ± 14.20	
	Median (IQR)	60 (50 - 75)	57.5 (45 - 65)	0.304
% Change (B – 12W)		1.51	0.00	

Table 7. Comparison of Prostate Volume Changes Between the Study Groups

IQR=Interquartile range, SD=Standard deviation. There was no significant decline in prostate volume over the 12-week study period in both treatment groups.

Qmax	Silodosin	Tamsulosin	P value Between Groups
Baseline	7.297 ± 2.780	7.403 ± 2.132	0.862
Week 4	9.297 ± 2.778	9.621 ± 2.001	0.588
Week 12	9.281 ± 2.733	9.632 ± 2.004	0.552
% Change in Mean (B – 12W)	27.20	30.00	
p-Value Within Group			

Table 8. Comparison of Qmax Changes Between the Study Groups

IQR=Interquartile range, SD=Standard deviation. There was significant decline in Qmax over the 12-week study period in both treatment groups (P < 0.001)

DISCUSSION

In the treatment of LUTS suggestive of BPH, α_1 antagonists are widely recognized as the first-line pharmacotherapy in the treatment of BPH[1] There are several subtypes of α_1-receptors which include α_{1A}, α_{1B}, and α_{1D}. The α_{1A} receptors, predominantly located in the smooth muscles of genitourinary tract, are the primary regulators of smooth muscle tone in the bladder neck and prostate, α_{1B} receptors are present in the vascular smooth muscle and regulate the vascular tone, and α_{1D} subtype mediates contraction of the bladder muscle[8] Early α_1 antagonists were non-selective for subtypes and were associated with blood pressure-related adverse effects, such as orthostatic hypotension. Therefore, agents with high selectivity for α_{1A} receptor should have beneficial effects on LUTS, fewer effects on blood pressure and fewer cardiovascular side effects.[9,10] The α_1 antagonists generally preferred in the management of BPH are Tamsulosin and Silodosin because of their minimal hemodynamic adverse effects.[11] Tamsulosin blocks α_{1A} receptor and α_{1D} receptor with a 10-fold greater affinity than α_{1B} receptor. The affinity of Silodosin towards α_{1A} receptor is about 162 times greater than those towards α_{1B}. A phase 2 study of Silodosin demonstrated an average IPSS and Qmax improvement of 6.5 points and 2.9 mL/sec respectively.[13] The first randomized double blind, placebo-controlled study between Tamsulosin and Silodosin was reported in 2006 in which patients received Silodosin4 mg twice daily, Tamsulosin 0.2 mg once daily, or a placebo for 12 weeks. The changes in the total IPSS from the baseline in the Silodosin, Tamsulosin, and placebo groups were 8.3, 6.8, and 5.3 respectively. The results showed Silodosin was better than the placebo and not inferior to Tamsulosin.[14] Yu et al also demonstrated non-inferiority of Silodosin to Tamsulosin in Asian patients[15]. In our study, the primary efficacy variable was the change in IPSS total score. In concurrence to above studies, the maximum decrease in IPSS occurred in both the study groups at week 4 indicating a similar onset of action and rate of symptomatic improvement with the two α-blockers. During subsequent visits, there was a gradual further decrease in the IPSS indicating that the improvement was sustained throughout the study period without loss of efficacy. At the end of 12 weeks the change in IPSS total score was 54.48% in Silodosin group and 53.97% in Tamsulosin group and no significant difference was found among the two groups. Our study considered change in voiding (incomplete voiding-Q1, intermittency-Q3, weak stream-Q5, straining-Q6) and storage sub scores (frequency-Q2, urgency-Q4, nocturia-Q7) as secondary efficacy variables. In the present study significant changes in all IPSS sub score parameters (<0.05) were observed within the groups but not between the groups. In a recent randomized cross-over comparison of half dose Silodosin with Tamsulosin by Takeshita H et al, subjective improvement in nocturia was noted specifically with silodosin.[16] Similar observation was made in our study where the improvement in mean score of nocturia was 40.32% in silodosin group and 37.14% in tamsulosin group. Silodosin was better by 3.18% when compared to Tamsulosin.

QOL score was one of the secondary efficacy variable in our study. The maximum improvement in QOL was observed after 4 weeks, and it was sustained throughout the study period with little further improvement indicating that the onset of improvement in the QOL corresponds to the decrease in IPSS. The changes in QOL from baseline till end of 12 weeks were highly significant within the groups (<0.05) but not between the two groups. 71.10% and 68.97% change in QOL score was observed in Silodosin and Tamsulosin groups respectively. Other studies have also observed a similar pattern of parallel improvement in the IPSS and QOL score.[3,17] Silodosin significantly improved QOL score compared to Tamsulosin.[9] Qmax was another secondary efficacy variable in our study. We observed that the improvement in Qmax with Silodosin and Tamsulosin were comparable. The changes in Qmax from baseline till end of 12 weeks were significant within the groups but not between the groups. 27.2% and 30% change in Qmax was observed in Silodosin and Tamsulosin groups respectively. Similar observations have been made in other studies with maximum improvement with Tamsulosin and lesser improvement with Silodosin.[3,17] Chapple et al reported that an increase in Qmax was observed in all groups – the adjusted change from baseline to end was 3.77 mL/s for Silodosin, 3.53 mL/s for Tamsulosin, and 2.93 mL/s for placebo, but the changes for Silodosin and Tamsulosin were not statistically significant versus placebo because of a particularly high placebo response. At end-point, the percentage of responders by Qmax were 46.6%, 46.5%, and 40.5% in the Silodosin, Tamsulosin, and placebo treatment groups, respectively. These differences in proportions were also not statistically significant[18] Yu et al have also reported that the changes in Qmax from baseline were comparable between Tamsulosin and Silodosin, and both were not statistically different from respective baseline.[15] Results of a recent meta-analysis in Fusco F et al have shown that Tamsulosin and Silodosin improve Qmax and bladder outlet obstruction index in patients with LUTS/BPH. Lower the Qmax at baseline, higher will be the end of the study improvement.[19] In our study there was a good correlation between the decrease in IPSS and QOL score but the improvement in Qmax did not correlate well with other two treatment outcome measures. Miyakita H et al. reported that a significant decrease in PVRU from baseline was observed only with Silodosin[9] In our study, it was observed that the changes in postvoid residual urine (PVRU) from baseline till end of 12 weeks was highly significant within both the groups (p<0.001) but not between the two groups. Selective α_1-blockers, unlike 5α-reductase inhibitors, are not expected to affect prostate size [14.] As

expected, these did not change significantly in either group. Our findings are in conformity with those in earlier studies [20]. With the results of present study, it appears that both Silodosin and Tamsulosin are comparable in efficacy and neither of them show significant improvement over the other in the parameters studied except for QOL. Thus, either drug may be used in treatment of LUTS associated with BPH with almost equal effectiveness.

CONCLUSION

Silodosin 8 mg, once daily, has similar efficacy in improvement of LUTS (IPSS– total score) associated with BPH as Tamsulosin 0.4 mg, once daily.

REFERENCES

[1] Yoshida M, Kudoh J, Homma Y, et al**.** New clinical evidence of silodosin, an α (1A) selective adrenoceptor antagonist, in the treatment for lower urinary tract symptoms. Int J Urol 2012;19(4):306-16.

[2] Chute CG, Panser LA, Girman CJ, et al. The prevalence of prostatism: a population-based survey of urinary symptoms. The Journal of Urology 1993;150(1):85-9.

[3] Yoshida M, Kudoh J, Homma Y, et al. Safety and efficacy of silodosin for the treatment of benign prostatic hyperplasia. Clin Interv Aging 2011;6:161-72.

[4] Pande S, Hazra A, Kundu AK. Evaluation of silodosin in comparison to tamsulosin in benign prostatic hyperplasia: a randomized controlled trial. Indian Journal of Pharmacology 2014;46(6):601-7.

[5] Salam A, Islam AKMA, Karim KMM, et al. A comparative study of terazosin and tamsulosin in medical treatment of symptomatic benign prostatic hyperplasia. JCMCTA 2009;20(1):34-40.

[6] Djavan B, Dianat SS, Kazzazi A. Effect of combination treatment on patient-related outcome measures in benign prostatic hyperplasia: clinical utility of dutasteride and tamsulosin. Patient Related Outcome Measures 2011;2:71-9.

[7] Yokoyama T, Yamamoto Y, Suzuki T, et al. Intraprostatic botulinum neurotoxin type A injection for benign prostatic hyperplasia: preliminary results with a newly purified neurotoxin. Acta Med Okayama 2012;66(4):291-7.

[8] Rossi M, Roumeguere T. Silodosin in the treatment of benign prostatic hyperplasia. Drug Des Devel Ther 2010;4:291-7.

[9] Miyakita H, Yokoyama E, Onodera Y, et al. Short-term effects of crossover treatment with silodosin and tamsulosin hydrochloride for lower urinary tract symptoms associated with benign prostatic hyperplasia. Int J Urol 2010;17(10): 869-75.

[10] Westfall TC, Westfall DP. Adrenergic agonists and antagonists. In: Goodman & Gilman's The pharmacological basis of therapeutics. 12th edn. New York: McGraw-Hill 2011: p. 277-334.

[11] Nickel JC, Sander S, Moon TD. A meta-analysis of the vascular-related safety profile and efficacy of α-adrenergic blockers for symptoms related to benign prostatic hyperplasia. International Journal of Clinical Practice 2008;62(10):1547-59.

[12] Gerber GS, Brendler CB. Evaluation of the urologic patient: history, physical examination and urinalysis. In: Wein AJ, Novick AC, Partin AW, et al. eds. Campbell-Walsh urology. 10th edn. Philadelphia: Elsevier Saunders 2012: p. 73-98.

[13] Marks LS, Gittelman MC, Hill LA, et al. Rapid efficacy of the highly selective α 1A-adrenoceptor antagonist silodosin in men with signs and symptoms of benign prostatic hyperplasia: pooled results of 2 phase 3 studies. The J of Urol 2009;181(6):2634-40.

[14] Kawabe K, Yoshida M, Homma Y, et al. Silodosin, a new α1A-adrenoceptor-selective antagonist for treating benign prostatic hyperplasia: results of a phase III randomized, placebo-controlled, double-blind study in Japanese men. BJU Int 2006;98(5):1019-24.

[15] Yu HJ, Lin AT, Yang SS, et al. Non-inferiority of silodosin to tamsulosin in treating patients with Lower Urinary Tract Symptoms (LUTS) associated with Benign Prostatic Hyperplasia (BPH). BJU Int 2011;108(11):1843-8.

[16] Takeshita H, Moriyama S, Arai Y, et al. Randomized crossover comparison of the short-term efficacy and safety of single half-dose silodosin and tamsulosin hydrochoride in men with lower urinary tract symptoms secondary to benign prostatic hyperplasia. Lower Urinary Tract Symptoms 2016;8(1):38-43.

[17] Roehrborn CG, Rosen RC. Medical therapy options for aging men with benign prostatic hyperplasia: focus on alfuzosin 10 mg once daily. Clinical Interventions in Aging 2008;3(3):511-24.

[18] Chapple CR, Montorsi F, Tammela TL, et al. Silodosin therapy for lower urinary tract symptoms in men with suspected benign prostatic hyperplasia: results of an international, randomized, double-blind, placebo-and active-controlled clinical trial performed in Europe. European Urology 2011;59(3):342-52.

[19] Fusco F, Palmieri A, Ficarra V, et al. α1-blockers improve benign prostatic obstruction in men with lower urinary tract symptoms: a systematic review and meta-analysis of urodynamic studies. European Urology 2016;69(6):1091-101.

[20] McConnell JD, Roehrborn CG, Bautista OM, et al. The long-term effect of doxazosin, finasteride and combination therapy on the clinical progression of benign prostatic hyperplasia. New England Journal of Medicine 2003;349(25):2387-98.