MARMARA
MEDICAL JOURNAL

Original Article

https://dergipark.org.tr/tr/pub/marumj

The effect of selective alpha-1 receptor antagonist on arterial stiffness parameters in patients with benign prostatic hyperplasia

Cagan YILDIRIM[*,1], Eda UCAR[*,2], Elif Nazlı LALE[*,2], Enes OZEN[*,2], Yavuz YILMAZ[*,2], Yunus Emre GENC[3], Zekeriya DOGAN[1], Yusuf Emre GUREL[1], Tarık Emre SENER[3], Murat SUNBUL[1]

[1] Department of Cardiology, School of Medicine, Marmara University, Istanbul, Türkiye
[2] School of Medicine, Marmara University, Istanbul, Türkiye
[3] Department of Urology, School of Medicine, Marmara University, Istanbul, Türkiye
[*]These five authors have equal contribution to this work.
Corresponding Author: Cagan YILDIRIM
E-mail: caganyildirim94@hotmail.com
Submitted: 06.08.2024 **Accepted:** 18.12.2025

ABSTRACT

Objective: Non-selective adrenergic alpha-1 antagonists have been shown to reduce arterial stiffness. Our aim is to assess the effects of a selective adrenergic alpha-1 antagonist on arterial stiffness, namely silodosin.

Patients and Methods: This prospective study was carried out on 20 patients with recently diagnosed benign prostatic hyperplasia (BPH). International Prostate Symptom Score (IPSS) calculation, arterial stiffness test with an oscillometric device, and uroflowmetry were performed prior to the planned silodosin medical treatment. After initial assessment, patients were given 8 mg of silodosin daily. Initial tests and calculations were repeated after 1 month of follow-up. Comparisons between baseline and control data were performed using the paired sample t-test and the Wilcoxon signed-rank test for normally and non-normally distributed data, respectively.

Results: There was a significant reduction in the IPSS of patients after silodosin treatment ($p < 0.001$). There were statistically insignificant reductions in arterial stiffness and systolic blood pressure, and an increase in maximum flow rate ($p = 0.314$, $p = 0.608$, and $p = 0.053$, respectively).

Conclusion: Silodosin is an effective drug to relieve BPH symptoms but does not have a statistically significant effect on neither blood pressure nor arterial stiffness.

Keywords: Adrenergic alpha-1 receptor antagonists, Arterial stiffness, Benign prostate hyperplasia, Silodosin

1. INTRODUCTION

Benign prostatic hyperplasia (BPH), which is defined as non-malignant growth of stromal and epithelial cells in the transition zone of the prostate, is one of the most common causes of lower urinary tract symptoms (LUTS) in men in the increasingly life-prolonging population [1]. The incidence of BPH increases with age. It is estimated that BPH affects up to 75% of men older than 50 years old and more than 80% of men older than 70 years old. Furthermore, the histological prevalence of BPH in autopsy studies is approximately 50-60% for men in their 60s and increases up to 90% for those aged 70 and over [1, 2]. The treatment of BPH includes regular follow-ups and medical or surgical treatment. Medical treatment is the first step for any patient without an indication for surgery [3]. There are two important agents used in the medical treatment of BPH. These are alpha-1 adrenoreceptor blockers, known as alpha-blockers, and 5-alpha-reductase inhibitors [4].

Alpha-1 blockers exert their action by binding to alpha receptors in smooth muscle or blood vessels, altering the action of the sympathetic nervous system. Alpha-1 adrenoreceptor blockers are divided into two groups: selective and non-selective. Non-selective alpha blockers cause vasodilation by blocking all types of alpha receptors (alpha-1 and alpha-2). Their blocking activity reduces vascular resistance in arterioles thus lowering the blood pressure. In clinical practice, nonselective alpha 1-blockers are not used in the first-line therapy due to adverse effects such as postural hypotension, fatigue, and dizziness [5]. Selective alpha blockers exert their action just on the alpha-1 or alpha-2 receptors and cause vasodilation and relaxation of smooth muscle in the related tissue [6]. Silodosin is one of the most selective agents among alpha blockers and therefore it is being used as one of the first-line agents in the treatment of symptomatic BPH, since it has a more favorable adverse effect profile [7-9].

How to cite this article: Yildirim C, Ucar E, Lale EN, et al. The effect of selective alpha-1 receptor antagonist on arterial stiffness parameters in patients with benign prostatic hyperplasia. Marmara Med J 2025;39(1): 19-23. doi: 10.5472/marumj.1872753

© 2026 Marmara University Press, All Rights Reserved
ISSN: 1309-9469

http://doi.org/10.5472/marumj.1872753
Marmara Med J 2026;39(1): 19-23

Content of this journal is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

Arterial stiffness refers to the thickening of the arterial wall due to collagen deposits [10]. Thus, arterial stiffness leads to a decrease in distensibility and loss of the capacity of arteries to absorb the pressure changes caused by the heart's pumping action [11]. It is a strong independent predictor of cardiovascular risk. Stiff arteries are a natural part of aging, but can also be caused by a variety of conditions, including diabetes, high blood pressure, and chronic kidney disease [12].

Arterial stiffness has many measures, such as pulse pressure, pulse wave velocity (PWV), and augmentation index (AIx). These measurements can be made invasively and noninvasively. Noninvasive methods are the most commonly used methods for the measurement of arterial stiffness. The velocity of the pulse wave passing through the arterial wall is directly proportional to the arterial stiffness, and the PWV is a direct and reliable measure of arterial stiffness. PWV can be measured by dividing distance by the difference in the arrival time of the waveforms between the two different arterial points. There are many types of PWV depending on the location of the arteries being measured. The most commonly used measurements are carotid-femoral PWV (cfPWV) and brachial-ankle PWV (baPWV) [13].

The relationship between arterial stiffness and non-selective alpha blockers has been investigated thoroughly in past studies. Our study aims to evaluate the relationship between arterial stiffness and the clinically used selective alpha blocker, namely silodosin.

2. PATIENTS and METHODS

Study population

The study was conducted in the Cardiology and Urology Departments of Marmara University Hospital. Ethical approval was obtained from the Marmara University School of Medicine Ethical Committee (Approval number: 03.12.2021.1382, date: 03.12.2021). Written and signed informed consents were obtained from subjects prior to the study. Thirty-two patients with recently diagnosed BPH were included into the study. Inclusion criteria were described as silodosin naïve male patients aged over 50 years with a recent diagnosis of BPH. For the diagnosis of BPH, the following criteria were considered: International Prostate Symptom Score (IPSS) more than 7, maximum flow rate (Qmax) ranging between 4-15 ml/sec with a minimum voided volume of 150 ml on uroflowmetry, and a prostate volume over 30 cm^2 in ultrasonography [14]. Exclusion criteria were determined to be a prior use of silodosin, alpha-blocker use in the last 2 weeks before arterial stiffness measurement, prostate cancer or prostate cancer suspicion, past pelvic surgeries including prostate-related surgeries or past pelvic radiotherapies, known neurological disorders, uncontrolled diabetes, urethral stenosis, or irregular use of silodosin throughout the study, and loss to follow-up. The presence of hypertension, diabetes mellitus, and hyperlipidemia was noted after diagnoses had been confirmed according to international guidelines [15-17]. Silodosin (8 mg a day) treatment was assigned to the subjects for a month. Subjects received their treatment plan early in the morning for 30 consecutive days. Arterial stiffness and related parameters were measured before initiation of silodosin treatment and remeasured 30 days after the initial measurements. Additionally, blood pressures of the patients were measured with 24-hour ambulatory blood pressure monitoring at both before initiation of treatment and 30 days after the initial measurements.

Assessment of arterial stiffness parameters

Arterial stiffness test was performed with the patient in the supine position in a quiet, temperature-controlled room (22–24°C) in the early morning, prior to which patients had refrained from eating, or drinking alcohol, coffee, or tea for at least 12 hours. Measurements were performed using an oscillometric device, (Mobil-O-Graph, Stolberg, Germany), which detects signals from the brachial artery with cuff pressure up to 35 mm Hg higher than systolic pressure. The basis of this technique is the contraction of the myocardium generating a pulse wave (early systolic peak) that travels down the aorta. The wave is reflected from the aortic wall at the distal branching point, generating a second reflected wave (late systolic peak), the morphology of which depends upon the stiffness of the large artery. PWV and AIx, adjusted for a heart rate of 75 bpm, were recalculated according to current guidelines, using the amplitude and time difference of the first and second wave [18].

Statistical analysis

Statistical analysis was performed with SPSS statistical package for Windows (version 20.0; SPSS Inc., Chicago, IL, USA). Whether the distribution of continuous variables was normal was determined by the Shapiro-Wilk test. Values displaying normal distribution were expressed as mean±SD and number of patients. Values not displaying normal distribution were expressed as median (interquartile range). Statistical comparison of quantitative data was performed with paired sample t-test for continuous variables displaying normal distribution. The Wilcoxon signed-rank test was used for continuous variables not displaying normal distribution. A value of $p<0.05$ was considered statistically significant.

3. RESULTS

During follow-up, twelve subjects who did not follow the treatment plan regularly were excluded from the study. The study population consisted of the remaining 20 patients with BPH. Mean age was 66.2 ± 7.5 years. Baseline clinical characteristics are shown in Table I. Comparison of cardiac hemodynamic parameters evaluated using an oscillometric device and urological parameters including IPSS and Qmax is shown in Table II. All cardiac hemodynamic parameters, were within normal range. Baseline cardiac hemodynamic parameters including blood pressure levels, heart rate, and pulse pressure were similar after the treatment. Arterial stiffness parameters, including PWV and AIx, were evaluated by the oscillometric method before and after treatment in the study population. Post-treatment measurements of PWV and AIx were similar to baseline measurements (9.7±1.4 m/s vs 9.9 ± 1.4 m/s,

http://doi.org/10.5472/marumj.1872753
Marmara Med J 2026;39(1): 19-23

p=0.314 and 23.8±12.6% vs 24.6±12.1%, p=0.859, respectively, (Figure-1)). IPSS values decreased significantly after silodosin therapy when compared to baseline values (p<0.001). Although post-treatment Qmax values were higher than pre-treatment values, they did not reach statistical significance (p=0.053).

Table I. *Clinical characteristics of study population (n=20)*

Age (year)	66.2±7.5
Height (cm)	169.3±6.7
Weight (kg)	80.8±14.6
Smoking (n-%)	4 (20)
Presence of hypertension (n-%)	8 (40)
Presence of diabetes mellitus (n-%)	6 (30)
Presence of hyperlipidemia (n-%)	6 (30)

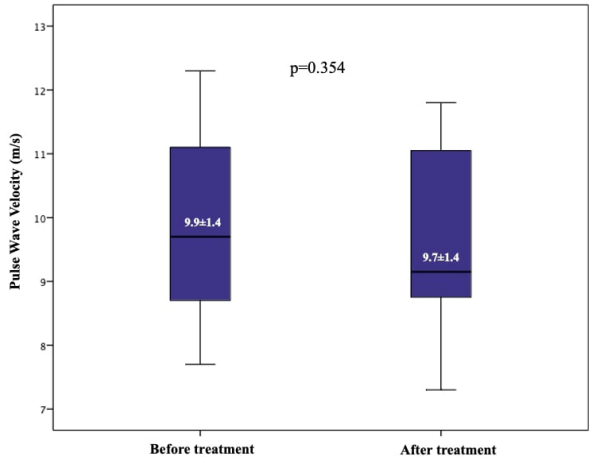

Figure 1. *Pulse wave velocity measurements with oscillometric device before and after silodosin treatment.*

Table II. *Comparison of cardiac hemodynamic measurements and urological parameters before and after treatment*

	Before treatment	After treatment	p
Systolic blood pressure (mmHg)	135.1±22.8	133.5±21.4	0.608
Diastolic blood pressure (mmHg)	85.0±11.6	83.0±13.6	0.363
Mean blood pressure (mmHg)	107.4±15.4	106.2±15.9	0.605
Heart rate (beat/minute)	80.3±14.5	78.8±17.8	0.666
Pulse pressure (mmHg)	49.6±18.1	50.5±14.5	0.701
Pulse wave velocity (m/s)	9.9±1.4	9.7±1.4	0.314
Augmentation index (%)	24.6±12.1	23.8±12.6	0.859
IPSS	17.2±6.3	13.5±5.5	< 0.001
Maximum flow rate (ml/s)	11.3±4.9	13.0±5.1	0.053

IPSS: International Prostate Symptom Score

4. DISCUSSION

In this prospective study, we aimed to determine the effect of silodosin treatment on arterial stiffness parameters in addition to validating its effectivity on BPH symptoms. Alpha-blocker therapies are used as symptomatic treatment for patients with outflow obstruction of the bladder due to BPH. Studies have shown that these agents significantly increase flow rates (Qmax) in the post-treatment period compared to pre-treatment values, and also significantly reduce IPSS values and improve the quality of life of patients [19-22]. In this study, we demonstrated that silodosin provides symptomatic improvement in patients by significantly reducing IPSS values, similar to the literature [7, 9]. On the other hand, even though there was a numerical improvement in urine flow rates, it did not reach statistical significance. This may be due to the small sample size.

Cardiovascular diseases (CVD) are the leading cause of mortality in the world population. Due to the high mortality of CVDs, studies on factors that will enable early detection of CVDs have been increased in the last two decades. Hypertension, atherosclerosis, and finally arterial stiffness are found to have crucial roles in the pathogenesis of CVDs besides age, obesity, genetics, and lifestyle. Arterial stiffness is mostly associated with the larger arteries and is increased in many CVDs, such as aging, hypertension, atherosclerosis, and diabetes mellitus as well as in many non-CVDs such as rheumatoid arthritis [12].

Arterial stiffness is both a cause and a result of hypertension. Arterial stiffness caused by hypertension leads to the development of inward remodeling in arteries, resulting in increased blood pressure itself, thus creating a feedback loop between arterial stiffness and hypertension [23]. Arterial stiffness is also associated with diabetes mellitus, and increased arterial stiffness is found even in pre-diabetic populations [24]. In the Framingham Heart Study, which included 2,232 people in a time span of 7.8 years, it was found that high aortic PWV was associated with a 48% increased risk for CVD [25]. Arterial stiffness is an independent determinant of cardiovascular events and mortality in healthy individuals, being a different indicator from classical cardiovascular risk factors.

The relationship between arterial stiffness, which enables us to evaluate the CVD risk of individuals in a short time with easy-to-apply methods, and antihypertensive treatment agents has been the subject of research in recent years. As a result of these studies, it was found that calcium channel blockers (CCBs), angiotensin II receptor blockers (ARBs), and angiotensin-converting enzyme (ACE) inhibitors have a better effect on arterial stiffness than diuretics and beta-blockers in people with arterial hypertension and cardiovascular risk factors [26].

Although the effect of many antihypertensive drugs on arterial stiffness has been investigated exclusively, there are only scarce data showing the direct effect of alpha-1 blockers on arterial stiffness. Wykretowicz et al., found that add-on therapy with 4 mg of doxazosin for 16 weeks to their existing antihypertensive therapy reduced arterial stiffness in 20 patients with insufficiently controlled mild-to-moderate essential hypertension [27]. Komai et al., reported that 12 months of low-dose doxazosin

http://doi.org/10.5472/marumj.1872753
Marmara Med J 2026;39(1): 19-23

treatment improved proximal arterial stiffness in their study of 15 hypertensive Asians [28].

No study has yet investigated the effect of another frequently used selective alpha-1 blocker, silodosin, on arterial stiffness in humans. Although silodosin is a prostate-specific alpha-1 blocker, it is known that it may cause orthostatic hypotension in some cases [29]. Considering the relationship between hypertension and arterial stiffness, we investigated the effects of silodosin on both blood pressure and arterial stiffness parameters.

In our study, a dose of 8 mg silodosin was administered once daily to patients diagnosed with BPH, and PWV measurements were taken before treatment initiation and after an average of four weeks of treatment. We did not find any significant difference regarding both blood pressure and arterial stiffness parameters. This finding suggests silodosin does not have an effect on arterial stiffness, which can be explained by maintained baseline blood pressure levels. On the other hand, arterial stiffness parameters of our patients showed a trend of decrease with the usage of silodosin, which may suggest there may have been a significant difference in a larger population due to our study being underpowered.

Limitations

Main limitation of our study is the small sample size, resulting in a calculated power of 0.09. Another limitation is the relatively short follow-up duration, which may have been inadequate to detect long-term changes.

Conclusion

We conclude that silodosin is an effective drug to relieve BPH symptoms but does not have a statistically significant effect on either blood pressure or arterial stiffness. However, with our study being underpowered, future studies with larger sample sizes and extended follow-ups may further clarify the relationship between arterial stiffness and silodosin.

Compliance with Ethical Standards

Ethical approval: Ethical approval was obtained from the Marmara University School of Medicine Ethical Committee (Approval number: 03.12.2021.1382, date: 03.12.2021). Written and signed informed consents were obtained from subjects prior to the study.

Conflict of interest: The authors declare that there is no conflict of interest.

Financial support: This study received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Author contribution: CY, EU, ENL, EO, YY, YEGenc, ZD and YEGurel: Concept, EU, ENL, EO, YY and MS: Design, data collection and processing, CY, ZD, YEGurel, TES and MS: Analysis or interpretation, CY, EU, ENL, EO, YY and YEGenc: Literature search, CY, EU, ENL, EO, YY and MS: Writing the manuscript. All authors have reviewed and approved the final version of the manuscript prior to submission.

REFERENCES

[1] Roehrborn CG. Benign prostatic hyperplasia: an overview. Rev Urol 2005;7 Suppl 9(Suppl 9):S3-s14.

[2] Egan KB. The Epidemiology of benign prostatic hyperplasia associated with lower urinary tract symptoms: Prevalence and incident rates. Urol Clin North Am 2016;43:289-97. doi:10.1016/j.ucl.2016.04.001

[3] Bishr M, Boehm K, Trudeau V, et al. Medical management of benign prostatic hyperplasia: Results from a population-based study. Can Urol Assoc J 2016;10:55-9. doi:10.5489/cuaj.3058

[4] Dhingra N, Bhagwat D. Benign prostatic hyperplasia: An overview of existing treatment. Indian J Pharmacol 2011;43:6-12. doi:10.4103/0253-7613.75657

[5] Nishimune A, Suzuki F, Yoshiki H, Morishima S, Muramatsu I. Alpha 1-adrenoceptor pharmacome: alpha 1L-adrenoceptor and alpha 1A-adrenoceptor in the lower urinary tract. Int J Urol 2010;17:31-7. doi:10.1111/j.1442-2042.2009.02368.x

[6] Cihan A, Kazaz İ O, Yıldırım Ö, et al. Changing aspects of male sexual functions accompanying treatment of benign prostatic hyperplasia with silodosin 8 mg per day. J Sex Med 2020;17:1094-100. doi:10.1016/j.jsxm.2020.02.023

[7] Schilit S, Benzeroual KE. Silodosin: a selective alpha1A-adrenergic receptor antagonist for the treatment of benign prostatic hyperplasia. Clin Ther 2009;31:2489-502. doi:10.1016/j.clinthera.2009.11.024

[8] Miernik A, Gratzke C. Current Treatment for benign prostatic hyperplasia. Dtsch Arztebl Int 2020;117:843-54. doi:10.3238/arztebl.2020.0843

[9] Jung JH, Kim J, MacDonald R, Reddy B, Kim MH, Dahm P. Silodosin for the treatment of lower urinary tract symptoms in men with benign prostatic hyperplasia. Cochrane Database Syst Rev 2017;11:Cd012615. doi:10.1002/14651858.CD012615.pub2

[10] Shirwany NA, Zou MH. Arterial stiffness: a brief review. Acta Pharmacol Sin 2010;31:1267-76. doi:10.1038/aps.2010.123

[11] Avolio A. Arterial stiffness. Pulse (Basel) 2013;1:14-28. doi:10.1159/000348620

[12] Zieman SJ, Melenovsky V, Kass DA. Mechanisms, pathophysiology, and therapy of arterial stiffness. Arterioscler Thromb Vasc Biol 2005;25:932-43. doi:110.1161/01.Atv.0000160548.78317.29

[13] Lee GP, Kim HL. Incremental value of the measures of arterial stiffness in cardiovascular risk assessment. Rev Cardiovasc Med 2022;23:6. doi:10.31083/j.rcm2301006

[14] Ko YH, Chae JY, Jeong SM, et al. Clinical implications of residual urine in Korean benign prostatic hyperplasia (BPH) patients: A prognostic factor for BPH-related clinical events. Int Neurourol J 2010;14:238-44. doi:10.5213/inj.2010.14.4.238

[15] McEvoy JW, McCarthy CP, Bruno RM, et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. Eur Heart J 2024;45:3912-4018. doi:10.1093/eurheartj/ehae178

[16] Marx N, Federici M, Schütt K, et al. 2023 ESC Guidelines for the management of cardiovascular disease in patients

with diabetes. Eur Heart J 2023;44:4043-140. doi:10.1093/eurheartj/ehad192

[17] Mach F, Koskinas KC, Roeters van Lennep JE, et al. 2025 Focused update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Eur Heart J 2025;46:4359-78. doi:10.1093/eurheartj/ehaf190

[18] Van Bortel LM, Duprez D, Starmans-Kool MJ, et al. Clinical applications of arterial stiffness, Task Force III: recommendations for user procedures. Am J Hypertens 2002;15:445-52. doi:10.1016/s0895-7061(01)02326-3

[19] Novara G, Chapple CR, Montorsi F. A pooled analysis of individual patient data from registrational trials of silodosin in the treatment of non-neurogenic male lower urinary tract symptoms (LUTS) suggestive of benign prostatic hyperplasia (BPH). BJU Int Sep 2014;114:427-33. doi:10.1111/bju.12712

[20] Kawabe K, Yoshida M, Homma Y. Silodosin, a new alpha1A-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;98:1019-24. doi:10.1111/j.1464-410X.2006.06448.x

[21] Wu YJ, Dong Q, Liu LR, Wei Q. A meta-analysis of efficacy and safety of the new α1A-adrenoceptor-selective antagonist silodosin for treating lower urinary tract symptoms associated with BPH. Prostate Cancer Prostatic Dis 2013;16:79-84. doi:10.1038/pcan.2012.36

[22] Manohar CMS, Nagabhushana M, Karthikeyan VS, Sanjay RP, Kamath AJ, Keshavamurthy R. Safety and efficacy of tamsulosin, alfuzosin or silodosin as monotherapy for LUTS in BPH – a double-blind randomized trial. Cent European J Urol 2017;70:148-53. doi:10.5173/ceju.2017.924

[23] Boutouyrie P, Chowienczyk P, Humphrey JD, Mitchell GF. Arterial stiffness and cardiovascular risk in hypertension. Circ Res 2021;128:864-86. doi:10.1161/circresaha.121.318061

[24] Prenner SB, Chirinos JA. Arterial stiffness in diabetes mellitus. Atherosclerosis 2015;238:370-9. doi:10.1016/j.atherosclerosis.2014.12.023

[25] Mitchell GF, Hwang SJ, Vasan RS, et al. Arterial stiffness and cardiovascular events: the Framingham Heart Study. Circulation 2010;121:505-11. doi:10.1161/circulationaha.109.886655

[26] Schettini IVG, Rios DRA, Figueiredo RC. Effect of different classes of antihypertensive drugs on arterial stiffness. Curr Hypertens Rep 2023;25:61-70. doi:10.1007/s11906-023-01238-4

[27] Wykretowicz A, Guzik P, Wysocki H. Doxazosin in the current treatment of hypertension. Expert Opin Pharmacother 2008;9:625-33. doi:10.1517/14656566.9.4.625

[28] Komai N, Ohishi M, Moriguchi A, et al. Low-dose doxazosin improved aortic stiffness and endothelial dysfunction as measured by noninvasive evaluation. Hypertens Res 2002;25:5-10. doi:10.1291/hypres.25.5

[29] Yoshida M, Kudoh J, Homma Y, Kawabe K. Safety and efficacy of silodosin for the treatment of benign prostatic hyperplasia. Clin Interv Aging 2011;6:161-72. doi:10.2147/cia.S13803