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INNOVATIVE TREATMENT STRATEGIES FOR PATIENTS WITH OVERACTIVE BLADDER AND URGE INCONTINENCE NOT CAUSED BY OBSTRUCTION

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Abstract

Overactive bladder (OAB) is observed in approximately 25% of patients with various urinary tract disorders, with urge urinary incontinence present in about one-third of these cases. The objective of this study was to enhance treatment outcomes in women with OAB, urge urinary incontinence, and non-obstructive urination by employing a combined pharmacological approach using an α1-adrenergic blocker and an M-anticholinergic agent. Additionally, the study aimed to investigate the correlation between clinical symptoms and urodynamic parameters, as well as assess changes in bladder mucosal microcirculation before and after therapy.

The study involved 40 women aged 20 to 72 years, with disease durations ranging from one to 26 years. The treatment regimen included a daily combination of tamsulosin (0.4 mg at night) and oxybutynin (5 mg in the morning) over a period of one month. Post-treatment assessments using uroflowmetry and cystometry revealed reduced urination time, increased urine volume per void, and improved maximum urinary flow rate. Cystometric bladder capacity also increased, while involuntary detrusor contractions during bladder filling whether spontaneous or provoked were significantly reduced or completely absent. Ultrasound evaluation showed a reduction in post-void residual urine volume.

Following treatment, a positive clinical response was observed in 29 patients (73%), with urinary incontinence persisting in only 6 patients (15%).

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Introduction

Urinary disorders remain a significant challenge in modern urogynecology. Numerous studies have reported a growing prevalence of these conditions among women [1, 2]. The clinical presentation of urinary dysfunction is diverse, ranging in severity from urinary incontinence to acute urinary retention, reflecting a wide spectrum of underlying pathologies. These disorders generally result from either obstructive urination, irritative symptoms, or a combination of both.

Overactive bladder (OAB) is a chronic syndrome characterized by urinary symptoms. According to the European Association of Urology, OAB often accompanied by urge urinary incontinence is defined by increased urinary frequency with or without the urgent need to void [3, 4]. OAB affects approximately 20% of individuals with urinary dysfunction, with urge incontinence occurring in about one-third of these cases [5]. The condition is most prevalent among women aged 20 to 50, a demographic typically in their working years. Although OAB is not life-threatening, it significantly impairs mental health and contributes to social, psychological, professional, familial, and sexual difficulties, ultimately diminishing a woman's quality of life.

The principal urodynamic indicator of OAB is involuntary detrusor contractions detected during the bladder filling phase via cystometry [6]. These contractions raise intravesical pressure, leading to a compelling urge to urinate, suggesting a primary impairment in the bladder's storage function [7].

Two prevailing theories explain the pathogenesis of OAB: the neurogenic and the myogenic hypotheses. The neurogenic theory attributes OAB to heightened afferent nerve activity or weakened central/peripheral neural control of bladder storage. This is supported by evidence that functional bladder outlet obstruction (such as detrusor-sphincter dyssynergia) and certain neurological disorders activate bladder C-fiber receptors, triggering involuntary detrusor contractions. In contrast, the myogenic theory suggests that structural and functional alterations in detrusor muscle cells disrupt intercellular signaling, leading to the

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synchronized contraction of multiple muscle fibers and resulting in detrusor overactivity.

A variety of factors contribute to OAB. While some patients experience idiopathic detrusor overactivity, others develop the condition due to neurological disorders, including Alzheimer's disease, stroke, brain tumors, multiple sclerosis, Parkinson's disease, spinal cord injuries, and vertebrogenic pathologies like spinal stenosis [8]. In many cases, urgency symptoms are also linked to recurrent inflammation, hormonal imbalances, and nerve dysfunction, often accompanied by microcirculatory impairments in the bladder and adjacent tissues [9]. Reduced blood flow can lead to hypoxia and metabolic disruption, playing a critical role in OAB pathogenesis.

Despite ongoing efforts, effective treatment strategies for managing OAB in women remain limited. Anticholinergic drugs, which inhibit muscarinic M-receptors on smooth muscle cells and suppress involuntary detrusor activity, are the mainstay of therapy. However, their systemic side effects often limit long-term use. In cases of obstructive urination, anticholinergic treatment must be carefully monitored or avoided altogether due to the risk of urinary retention. The introduction of α1-adrenergic blockers has provided a novel approach to treating various urinary dysfunctions, including OAB [10]. These medications have demonstrated significant efficacy in patients with urodynamically confirmed functional bladder outlet obstruction [11]. Research has shown that increased α1-adrenergic receptor activity in the bladder neck and proximal urethra contributes to smooth muscle spasms and dynamic obstruction [12]. By blocking these receptors, α1-blockers promote detrusor relaxation during bladder filling and enhance bladder capacity. This results in a notable improvement in lower urinary tract function, manifested by reduced urinary frequency and a normalized urination pattern. Some researchers, including D. Yu. Pushkar et al. [13], have also suggested that α1-blockers exert beneficial effects on the bladder's vascular system.

Oxybutynin, a widely used M-anticholinergic agent, illustrates the broader therapeutic potential of targeting biochemical pathways to regulate cellular and organ function [14]. Although traditionally associated with its antimuscarinic

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action on the detrusor, recent studies have suggested possible vascular effects of M-anticholinergic drugs, including those on the heart and urinary bladder.

In recent years, the role of microcirculation in disease processes and recovery has gained attention. The study of regional microcirculation is increasingly recognized as crucial for understanding tissue repair, morphological restoration, and functional recovery after various pathological conditions or treatments [16]. Detecting microcirculatory abnormalities is vital for diagnosis, assessing disease severity, monitoring therapy, and predicting outcomes.

The aim of this study was to improve treatment outcomes in women with OAB and urge urinary incontinence by using a combination of an α1-adrenergic blocker and oxybutynin (5 mg). The study also sought to examine the relationship between clinical symptoms and urodynamic findings including urgency and obstructive urination and to assess changes in bladder mucosal microcirculation before and after treatment.

Materials and methods

The study was conducted at the *Republican Specialized Scientific and Practical Medical Center of Urology*, a state institution located in Tashkent, Uzbekistan. A total of 40 women diagnosed with overactive bladder (OAB) and non-obstructive urination disorders were enrolled. Participants ranged in age from 20 to 72 years, with a mean age of 51.6 years. The duration of symptoms varied between one and 20 years.

Notably, 11 patients (28%) had a history of gynecological surgery, with 8 of these (20%) having undergone interventions for stress urinary incontinence. Neurological comorbidities were identified in 28 patients (70%), primarily involving cervical and lumbar spine disorders. A comprehensive urological examination revealed no evidence of lower urinary tract organic pathology or urethral stricture in any of the participants.

The treatment protocol included a combination of tamsulosin (0.4 mg at bedtime), an α1-adrenergic blocker, and oxybutynin (5 mg each morning), an M-anticholinergic agent. The treatment was administered daily over the course of one month. According to published data, both medications have minimal impact

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on systolic/diastolic blood pressure and heart rate, thus ensuring cardiovascular safety.

The diagnostic assessment included:

A voiding diary

Routine urological evaluation (urinalysis, complete blood count)

Urodynamic studies (uroflowmetry and cystometry)

Ultrasound examination of the bladder with measurement of post-void residual urine volume

Urodynamic testing, particularly uroflowmetry and cystometry, served as the primary diagnostic method for assessing bladder storage and voiding function. Uroflowmetry was considered valid if the bladder volume at the time of voiding was between 150 and 350 ml. Automated analysis of the uroflow curves was performed, generating both graphical and quantitative data. The following parameters were analyzed:

TQ – voiding time. **Qmax** – maximum urine flow rate. **Vcomp** – voided urine volume

Reference values were based on norms established by E.L. Vishnevsky et al. [17]. Cystometry was used to assess bladder filling characteristics, including physiological and maximum bladder capacity, detrusor stability, and pressure fluctuations. Particular attention was given to involuntary detrusor contractions during the filling phase (spontaneous or provoked), which are indicative of detrusor overactivity and cannot be suppressed by voluntary effort.

To evaluate the microcirculatory status of the bladder mucosa, laser Doppler flowmetry (LDF) was employed. This modern, non-invasive method is based on detecting frequency shifts in laser light reflected by moving blood components, primarily erythrocytes. Using a laser microcirculation analyzer, two key assessments were performed:

1-**Basal perfusion parameters**, including the mean microcirculatory index (M), standard deviation (σ), and coefficient of variation.

2-**Amplitude-frequency spectrum analysis** of perfusion oscillations, which provides insight into vascular tone and the function of various perfusion control mechanisms across specific frequency ranges

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A control group of 10 women without symptoms of OAB, urge incontinence, or obstructive voiding was used for comparison. Blood flow characteristics in the control group, as assessed via uroflowmetry, were considered representative of normal peripheral hemodynamics in the bladder wall.

Results and discussion

A clinical improvement was observed in 29 patients (73%) following treatment, as confirmed by entries in the urination diary (Table 1).

Table 1. Changes in Clinical Symptoms Before and After Treatment.

Symptom	Episodes before treatment	Number of episodes following treatment
Pollakiuria	17,6±4,1	9,9±1,0 p>0.001
Nocturia	5,6±0,4	2,4±0,1 p>0.001
Imperative urges	8,7±1,0	2,0±0,3 p>0.001
Feeling of incomplete emptying of the bladder	5,0±0,8	2,3±0,1 p>0.001
Weakening of the urine stream	6,1±0,4	3,1±0,3 p>0.001
Intermittent urination	5,2±0,6	3,3±0,2 p>0.001

The most commonly reported side effects associated with the combination therapy were dizziness, general weakness, hypotension, nausea, and, in some cases, flushing and palpitations. These symptoms were generally transient and tended to diminish over time. Importantly, no patients discontinued treatment due to adverse effects.

Ultrasound evaluation revealed that, prior to treatment, 15 patients (37.5%) had post-void residual (PVR) urine volumes ranging from 50 to 270 ml (mean: 105 ml). Following therapy, residual urine was detected in only 6 patients (15%), with volumes ranging from 65 to 95 ml.

A reduction in the maximum urine flow rate (Qmax), prolonged urination time (TQ), and the presence of a wave-like uroflowmetry curve are typically indicative of bladder outlet obstruction, reduced detrusor contractility, or underlying neurogenic dysfunction. In this study, the uroflowgrams of all patients exhibited

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a characteristic zigzag pattern, reflecting features consistent with detrusor overactivity.

Quantitative uroflowmetry parameters recorded before and after treatment are summarized in Table 2.

Table 2. Changes in Uroflowmetry Indicators Pre- and Post-Treatment (M±m).

Index	Before treatment	After treatment	Norm
TQ,c	49.7±0.4	21.7±0.2 p>0.001	8.77±0.66
Qmax ml/c	6,9±0,7	26,2±0,1 p>0.001	29,91±2,18
Vcom ml	67,2±3,2	155,4±1,9 p>0.001	138,11±11,63

Urodynamic disturbances identified via uroflowmetry were characterized by a reduction in the bladder's evacuation function, reflected in prolonged urination time and decreased urinary flow rate. Additionally, impaired storage function was indicated by a lower volume of urine excreted prior to treatment. Following the therapeutic intervention, urination time was reduced, while both the voided volume and the maximum flow rate increased. Notably, the uroflowmetry curve appeared less irregular and zigzag-shaped in all cases, indicating a normalization of the urination pattern.

Cystometric analysis revealed that all 40 patients (100%) exhibited decreased cystometric bladder capacity, ranging from 50 to 130 ml (mean: 110 ml), pointing to compromised storage function. Detrusor overactivity was present in all participants, manifested by involuntary contractions during the bladder filling phase (either spontaneous or provoked), which could not be voluntarily suppressed. Post-treatment evaluations showed an increase in cystometric capacity, with values ranging from 75 to 210 ml (mean: 180 ml). Additionally, detrusor stability was restored in 9 patients (23%), and in the remaining cases, the frequency of involuntary detrusor contractions significantly declined.

Microcirculatory analysis prior to treatment demonstrated decreased tissue perfusion, as evidenced by a reduction in the coefficient of variation, myogenic and neurogenic tone, shunt index, and microcirculation efficiency index. After treatment, improvements were observed in neurogenic tone in the precapillary

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segment, the shunt index, and microcirculation efficiency index, along with increases in the overall microcirculation index and coefficient of variation. These changes suggest enhanced vascular function and improved mucosal condition of the bladder.

Amplitude-frequency spectrum analysis of the bladder wall mucosa post-treatment revealed a statistically significant decrease in the amplitude of pulse and slow oscillations, along with an increase in the amplitude of fast oscillations. There was a marked improvement in active microcirculatory mechanisms and a reduction in passive regulatory mechanisms and vascular tone. These findings indicate a notable reduction in venous stasis within the bladder wall and an overall enhancement of bladder wall perfusion.

Conclusion

Based on the results of our study, the combined use of the α1-adrenergic blocker tamsulosin (0.4 mg at night) and the M-anticholinergic agent oxybutynin (5 mg in the morning), administered daily over the course of one month, proved to be effective in treating women with overactive bladder (OAB), urge urinary incontinence, and non-obstructive urination. This therapeutic regimen led to normalization of bladder function during both the storage and voiding phases, along with improvements in microcirculation within the bladder mucosa, as confirmed by specialized diagnostic assessments. A positive clinical response was observed in 29 patients (73%), while urge urinary incontinence persisted in only 6 patients (15%), as documented in urination diaries.

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