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Multimodal Rehabilitation Combining -Wide-Focused Li-ESWT- and Behavioral Therapy for Coexisting Underactive Bladder, Mild BPH, and Erectile Dysfunction: A Case Report

[1, 2] Farida Arisanti*, [3]Anjar Setiadi, [4] Winge W. Arline, [5] Rinesa Larasati
[1] Physical Medicine and Rehabilitation Department, Faculty of Medicine, Universitas Padjadjaran/Dr. Hasan Sadikin General Hospital, Bandung, West Java, Indonesia*; email: farida.arisanti@unpad.ac.id
[2] Slamet 39, Neurorehabilitation Center, Bandung, Indonesia; email: farida.arisanti@unpad.ac.id
[3] Slamet 39, Neurorehabilitation Center, Bandung, Indonesia; email: anjarsetiadi1988@gmail.com
[4] Slamet 39, Neurorehabilitation Center, Bandung, Indonesia; email: wwarline2204@gmail.com
[5] Physical Medicine and Rehabilitation Department, Faculty of Medicine, Universitas Padjadjaran/Dr. Hasan Sadikin General Hospital, Bandung, West Java, Indonesia; email: larasatirinesa@gmail.com
*Correspondence

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Abstract
Introduction: *Managing coexisting detrusor underactivity (UAB), benign prostatic hyperplasia (BPH), and erectile dysfunction (ED) remains challenging when symptoms persist despite maximal medical therapy.* ***Objective:*** *To evaluate the clinical outcomes of a multimodal neurorehabilitation approach combining -wide-focused low-intensity extracorporeal shockwave therapy- (Li-ESWT), fluid management, and relaxation-focused pelvic floor muscle training in a patient with refractory UAB, mild BPH, and ED.* ***Method:*** *A 66-year-old male with cardiometabolic risk presented with persistent lower urinary tract symptoms and ED despite treatment with Dutasteride/Tamsulosin and Bethanechol. Baseline ultrasound showed mild prostatic enlargement (35.4 g), voided volume of 298 mL, peak flow rate (Qmax) of 21.4 mL/s, and post-void residual (PVR) of 100-150 mL. The patient underwent a 12-week multimodal rehabilitation program.* ***Result and Discussion:*** *PVR decreased from 100–150 mL to 40–50 mL, reducing clean intermittent catheterization (CIC) from 3–4 to 1–2 times daily. Functional voided volume increased to 380 mL, nocturnal dribbling resolved, and Qmax remained non-obstructive at 18.2 mL/s, indicating improved detrusor contractile function and quality of life.* ***Conclusions:*** *Multimodal neurorehabilitation incorporating Li-ESWT, behavioral modification, and pelvic floor training may provide a promising non-invasive treatment for refractory UAB, BPH, and ED*

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Introduction

Aging men frequently experience concurrent lower urinary tract symptoms (LUTS)—including detrusor underactivity (UAB) and overactive bladder (OAB)—alongside vasculogenic erectile dysfunction (ED). These interrelated conditions share a core pathophysiology driven by cardiometabolic risk factors: atherosclerosis-induced chronic pelvic ischemia (CPI), oxidative stress, endothelial dysfunction, and peripheral autonomic neuropathy (Baboudjian et al., 2024). This chronic ischemic state severely impairs penile artery relaxation and triggers detrimental bladder tissue remodeling (De Leonardis et al., 2022; Sarychev and Savchenko, 2021). Furthermore, the physical burden of pelvic floor dysfunction often induces psychological distress and anxiety, creating a detrimental cycle of pelvic floor hypertonicity that severely reduces the patient's quality of life (Grimes and Stratton, 2023).

Standard pharmacological treatments frequently reach a therapeutic plateau because they merely manage symptoms without reversing the underlying ischemic tissue degradation. For instance, combining alpha-blockers with anticholinergics yields mixed evidence for long-term urologic improvement (Pang et al., 2021), while muscarinic agonists like bethanechol stimulate temporary detrusor contractions but fail to induce the structural muscle remodeling necessary for true UAB management (Gaitonde et al., 2019; Galluzzi et al., 2025). Polypharmacy further complicates recovery; concurrent medications for central anxiety, such as benzodiazepines (e.g., Alprazolam), can inadvertently impair coordinated sphincter relaxation, exacerbating nocturnal distress and dribbling. Consequently, effective management of refractory ED and LUTS necessitates a comprehensive, multimodal strategy (Abreu-Mendes et al., 2020).

To overcome these pharmacological limitations, integrating "bottom-up" biophysical therapies with "top-down" behavioral modifications offers a highly synergistic solution. -Wide-focused low-intensity extracorporeal shockwave therapy (Li-ESWT)- induces cellular mechanotransduction, activating pathways that express Vascular Endothelial Growth Factor (VEGF) and endothelial Nitric Oxide Synthase (eNOS) to aggressively restore microvascular networks and peripheral nerves in both penile and bladder tissues (Frairia and Berta, 2011). When this structural tissue regeneration is coupled with lifestyle modifications, fluid regulation, and relaxation-focused pelvic floor muscle training (PFMT), the combined approach effectively minimizes detrusor over-distension and reduces central sympathetic overactivity (Lazaros et al., 2023). Ultimately, this holistic multimodal intervention successfully bridges the gap between mechanical tissue repair and coordinated neuromuscular control.

Case Presentation

Patient Demographics and Medical History A 66-year-old male presented to the neurorehabilitation center with persistent mixed lower urinary tract symptoms (LUTS) and concurrent vasculogenic erectile dysfunction (ED). The patient possessed a complex medical history marked by cardiovascular risk factors, hypertension, and a cardiometabolic profile. Despite undergoing optimal pharmacological management, the patient exhibited persistent detrusor weakness and incomplete bladder emptying. His active medication regimen included Duodart (Dutasteride/Tamsulosin) for benign prostatic hyperplasia (BPH), Urotone (Bethanechol chloride) to manage detrusor underactivity (UAB), and Alprazolam (0.5 mg) for central anxiety.

Baseline Anatomical and Radiological Profile To establish the anatomical baseline, a transabdominal urological ultrasound was performed on January 7, 2025. The imaging revealed only a mild prostatic enlargement with an estimated volume of 35.4 grams (4.5×3.6×4.1 cm). Crucially, the urinary bladder demonstrated a completely normal, non-thickened wall contour. This specific anatomical presentation confirmed that the patient was suffering from primary myogenic detrusor weakness (underactive bladder) rather than a secondary decompensation driven by severe mechanical bladder outlet obstruction.

Functional and Behavioral Assessments Subjectively, the patient reported a mild Overactive Bladder Symptom Score (OABSS total score: 3), which was primarily driven by episodes of nocturnal urgency. The baseline 24-hour voiding diaries highlighted a severe, unregulated daily fluid intake exceeding 4,000 mL/day, accompanied by frequent and distressing episodes of uncontrolled nocturnal dribbling ranging between 40 mL and 120 mL per episode.

Objective functional baseline parameters were recorded via uroflowmetry on February 5, 2025. The evaluation confirmed incomplete bladder emptying, revealing a restricted functional Voided Volume of 298 mL and a Post-Void Residual (PVR) urine volume ranging 100-150 mL, which was why the patient required Clean Intermittent Catheterization (CIC) 3–4 times daily at baseline. While the peak flow rate (Qmax) was recorded at 21.4 mL/s—likely augmented by the ongoing Dutasteride/Tamsulosin therapy—the detrusor contraction initiation was suboptimal, evidenced by a prolonged Time to Max Flow (TQmax) of 10.8 seconds.

Methods

Study Design and Ethical Considerations

This study presents a single-case report of a 66-year-old male undergoing a 3-month multimodal neurorehabilitation program designed to treat coexisting detrusor underactivity (UAB), benign prostatic hyperplasia (BPH), and erectile dysfunction (ED).

Multimodal Intervention Protocol

The comprehensive rehabilitation protocol combined "bottom-up" biophysical tissue regeneration with "top-down" neuro-behavioral modifications over 12 weeks. The intervention frequency was scheduled once weekly for the first 2 months (Weeks 1–8) and escalated to twice weekly during the third month (Weeks 9–12).

Biophysical Therapy (-Wide-Focused Li-ESWT-): Low-intensity extracorporeal shockwave therapy was administered using the Likawave device, delivering a total of 6,000 pulses per session without anesthesia. The pulses were distributed anatomically as follows:

Suprapubic (Bladder Wall): 2,000 pulses at Energy Setting 5, 4 Hz.
Bilateral Crura Penis: 1,000 pulses (500 per side) at Energy Setting 5, 4 Hz.
Corpus Penis (Posterior, Media, Anterior): 3,000 pulses total (distributed across zones) at Energy Setting 4, 4 Hz.

Behavioral Therapy & Fluid Management: Based on initial voiding logs showing severe unregulated polyuria (>4,000 mL/day), strict restriction was enforced to target a physiological fluid intake of 2,500–3,000 mL/day. Total fluid cessation was mandated 3 hours prior to sleep.

Pelvic Floor Muscle Training (PFMT): The patient performed 3 sets daily of controlled exercises, consisting of 10 quick flicks (1-second hold) and 10 sustained contractions (5-second hold). Crucially, each contraction was followed by a mandatory 10-second complete muscle relaxation phase to mitigate autonomic hypertonicity.

Central Relaxation Therapy: To counteract somatic anxiety and sympathetic overactivity, the patient practiced structured diaphragmatic breathing and progressive muscle relaxation (PMR) for 15 minutes, twice daily.

Table 1
Systematized Multimodal Intervention Protocol (Likawave + Behavioral Therapy)

Phase / Modality	Targeted Anatomical Zone	Wave Type	Energy Setting	Frequency	Pulse Volume / Parameter	Clinical Schedule & Protocol Details
Li-ESWT Likawave	Suprapubic (Bladder Wall)	Wide Focus	Energy 5	4 Hz	2.000 pulses	Weeks 1–8: 1x / week; Weeks 9–12: 2x / week
	Bilateral Crura Penis	Wide Focus	Energy 5	4 Hz	500 / side (1.000 total)	Weeks 1–8: 1x / week; Weeks 9–12: 2x / week
	Corpus Penis Posterior (L/R)	Wide Focus	Energy 4	4 Hz	500 / side (1.000 total)	Weeks 1–8: 1x / week; Weeks 9–12: 2x / week
	Corpus Penis Media (L/R)	Wide Focus	Energy 4	4 Hz	500 / side (1.000 total)	Weeks 1–8: 1x / week; Weeks 9–12: 2x / week
	Corpus Penis Anterior	Wide Focus	Energy 4	4 Hz	500 pulses	Weeks 1–8: 1x / week; Weeks 9–12: 2x / week
Behavioral Therapy	Systemic Intake Regulation	N/A	N/A	N/A	Total Restriction	Restricted to 2,000–2,500 mL/day; complete fluid cessation 3 hours before sleep.
	Pelvic Floor Muscle Complex	N/A	N/A	N/A	Controlled Sets	3 sets/day: 10 quick flicks (1s), 10 sustained holds (5s) followed by 10s mandatory complete relaxation.
	Central Nervous System	N/A	N/A	N/A	15 mins	Diaphragmatic breathing and progressive muscle relaxation (PMR) 2x / day.

Results and Discussion

1. Result

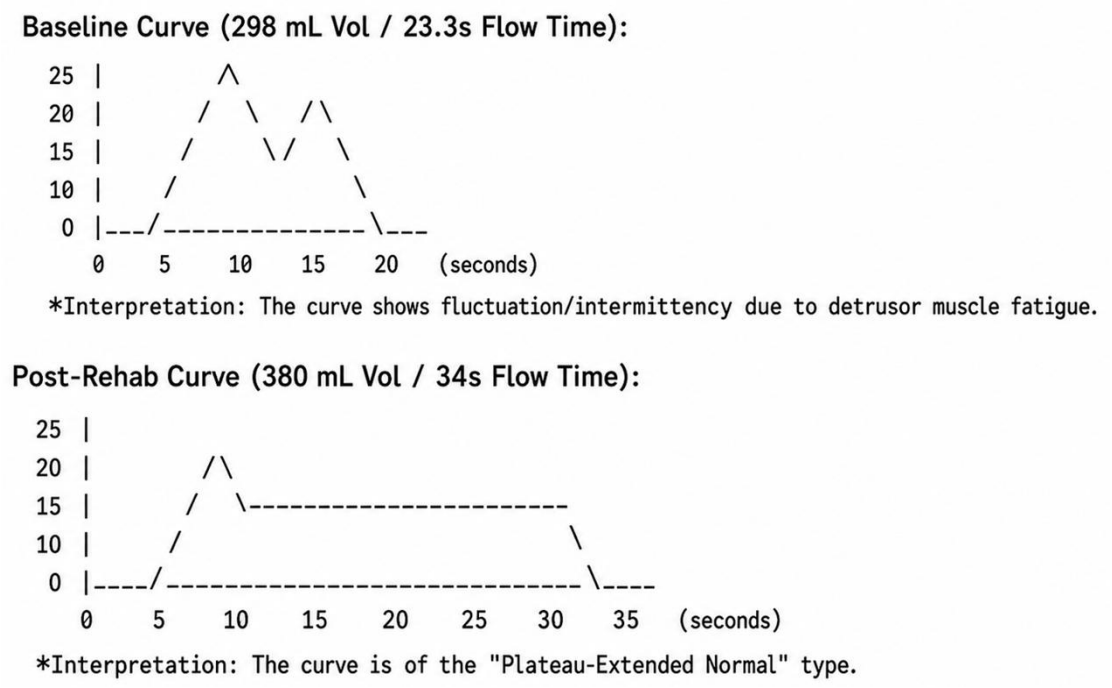
Baseline Anatomical and Functional Evaluation

A baseline transvesical and renal ultrasound performed on January 7, 2025, revealed mild prostatic enlargement (35.4 g; 4.5 x 3.6 x 4.1 cm) but demonstrated a normal, non-thickened urinary bladder wall contour, confirming a primary myogenic failure (UAB) rather than severe mechanical outlet obstruction. Subjectively, the patient exhibited a baseline Overactive Bladder Symptom Score (OABSS) of 3, driven by nocturnal urgency, alongside marked erectile dissatisfaction. Baseline 24-hour voiding diaries showed unregulated fluid intake (>4,000 mL/day) and frequent nocturnal dribbling (40–120 mL/episode). Objective baseline uroflowmetry on February 5, 2025, showed a Voided Volume of 298 mL, a Peak Flow Rate (Qmax) of 21.4 mL/s, a prolonged Time to Max Flow (TQmax) of 10.8 seconds, and a significant Post-Void Residual (PVR) urine volume of 100-150 mL.

Post-Intervention Clinical Outcomes

Following the 3-month multimodal protocol, reassessments in December 2025 revealed substantial functional improvements across all primary endpoints. Notably, the average Post-Void Residual (PVR) urine volume decreased substantially from a baseline of 100–150 mL to 40–50 mL per void, and this reduction in retained urine allowed the patient's reliance on Clean Intermittent Catheterization (CIC) to be decreased from 3–4 times daily to only 1–2 times daily. Final 24-hour voiding charts confirmed stabilized fluid intake and the complete resolution of nocturnal dribbling distress. Subjective score trends demonstrated a marked improvement in both the OABSS and the Index of Erectile Function (IFED).

Post-treatment uroflowmetry documented an expanded functional bladder capacity, with the functional Voided Volume increasing by 27.5% to 380 mL (up from 298 mL). The velocity of detrusor contraction optimized, as shown by a shortened TQmax of 10.0 seconds (down from 10.8 seconds). The peak flow rate adjusted to a stable, non-obstructive functional parameter of 18.2 mL/s over a smooth, continuous 34-second flow curve, confirming superior detrusor-sphincter coordination without an increase in outflow resistance or any adverse events.



Picture 1, 2. Summary of Functional Parameter Results

Table 2
Pre- and post-intervention adjustments clearly, the objective clinical data shifts

Functional Parameter	Baseline (Feb 2025)	Post-Intervention (Dec 2025)	Clinical Interpretation
Post-Void Residual (PVR)	100-150 mL	40-50 mL	Improved detrusor emptying and reduced retained urine
CIC Frequency	3-4 times daily	1-2 times daily	Reduced catheterization burden
Voided Volume	298 mL	380 mL	+27.5% Expansion of bladder capacity
Peak Flow Rate (Qmax)	21.4 mL/s	18.2 mL/s	Stabilized functional curve without obstruction
Time to Max Flow (TQmax)	10.8 seconds	10.0 seconds	Swifter detrusor contraction initiation
Nocturnal Dribbling	40–120 mL/episode	Resolved	Complete symptomatic resolution
Daily Fluid Intake	>4.000 mL/day	2.500–3.000 mL/day	Stabilized fluid homeostasis

2. Discussion

The management of coexisting detrusor underactivity (UAB), benign prostatic hyperplasia (BPH), and vasculogenic erectile dysfunction (ED) in elderly patients with cardiometabolic profiles represents a major therapeutic challenge. The structural and anatomical insights provided by the baseline urological ultrasound are crucial for unraveling this case's clinical paradox. The imaging revealed only a mild prostatic enlargement (35.4 g) coupled with a completely normal, non-thickened urinary bladder wall contour. In conventional bladder outlet obstruction (BOO) secondary to BPH,

chronic mechanical resistance typically induces compensatory detrusor muscle hypertrophy, macroscopically visible as bladder wall thickening (detrusor wall thickness/DWT) observed with ultrasound examination.(Bhattarai et al., 2024)

The absence of detrusor wall thickening in this patient, despite an elevated baseline post-void residual (PVR), directly demonstrates a primary myogenic contractility failure, classifying the condition as underactive bladder rather than pure mechanical obstruction. (Saleh et al., 2020)

Recent studies suggest that increased suburothelial inflammation and altered sensory protein expressions in the bladder mucosa are prominent in UAB patients, contributing to impaired detrusor contractility. The condition is often seen in elderly patients with chronic medical or neurological diseases, further complicating the diagnosis and management. (Jiang and Kuo, 2019)

This primary myogenic deficit explains why conventional pharmacological therapies reached a clinical plateau. Although the patient was optimized on Duodart (Dutasteride/Tamsulosin) to reduce urethral resistance—which accounts for the high baseline peak flow rate (Qmax) of 21.4 mL/s in the bladder "pump" remained structurally weak, requiring oral parasympathomimetics (Urotone/Bethanechol chloride) to chemically force detrusor contractions. However, oral cholinergics merely stimulate available muscarinic receptors without reversing the underlying chronic pelvic ischemia, endothelial dysfunction, or microvascular rarefaction driven by the patient's hypertensive and cardiometabolic background (Gaitonde et al., 2019; Galluzzi et al., 2025). Furthermore, the long-term administration of benzodiazepines (Alprazolam 0.5 mg) for central anxiety can inadvertently induce generalized muscle relaxation and impair coordinated pelvic floor activity, further hindering efficient bladder evacuation and exacerbating nocturnal distress like incontinence (Tsakiris et al., 2008).

The profound functional recovery observed after the 3-month intervention demonstrates a powerful synergy between biophysical tissue regeneration and neuro-behavioral modulation. On a tissue level, Li-ESWT delivered via the Likawave device served as a powerful regenerative agent. Unlike focused shockwaves that target a singular point, wide-focus acoustic energy distributes mechanotransduction forces over a larger surface area of the suprapubic bladder wall and penile tissues. This mechanical shear stress upregulates the PI3K/Akt signaling pathway, promoting the release of angiogenic factors like Vascular Endothelial Growth Factor (VEGF) and endothelial Nitric Oxide Synthase (eNOS) (Frairia and Berta, 2011). This biological cascade restores microvascular networks, enhances blood perfusion to the ischemic detrusor muscle, and recruits endogenous progenitor cells to repair damaged cavernous smooth muscle structures, directly addressing both UAB and vasculogenic ED simultaneously. In the context of erectile dysfunction, Li-ESWT improves erectile function by enhancing neovascularization and tissue regeneration, which could similarly benefit pelvic floor muscle function (Porst, 2021).

Therapeutic shockwaves exert mechanical stress via direct amplitude impact and the violent collapse of cavitation bubbles. This cavitation preferentially targets and disrupts vascular endothelium rather than solid tissues, thereby initiating neoangiogenesis through chemokine-mediated stem cell activation, VEGF release, and endothelial progenitor cell recruitment (Fode et al., 2017).

Crucially, this biophysical repair was structurally guided by the behavioral framework. Restricting daily fluid intake to 2,000–2,500 mL and enforcing complete

cessation three hours prior to sleep effectively protected the newly regenerating detrusor muscle fibers from chronic mechanical over-distension, allowing the bladder to operate within a physiological volume range. Concurrently, because traditional high-tone pelvic floor exercises can exacerbate outflow resistance in patients with mixed bladder phenotypes, the pelvic floor muscle training (PFMT) in this protocol heavily prioritized the complete muscle relaxation phase combined with diaphragmatic breathing. This relaxation focus down-regulated systemic sympathetic overactivity, a clinical necessity aligned with the patient's Alprazolam profile thereby decreasing guarding reflexes and reducing functional bladder outlet hypertonicity (Cox and Rovner, 2019; Lazaros et al., 2023).

The integration of these mechanisms is clearly reflected in the post-treatment uroflowmetry curve. The patient's functional bladder capacity successfully expanded, with the voided volume increasing by 27.5% to 380 mL, while maintaining a stable, non-obstructive functional Qmax of 18.2 mL/s and shortening the Time to Max Flow (TQmax) to 10 seconds. Rather than showing a volatile or hesitant pattern, the post-rehabilitation curve assumed a smooth, extended plateau configuration. This specific morphology proves that a structurally rejuvenated detrusor muscle that repaired by Li-ESWT was able to sustain a constant, efficient contraction against a fully relaxed, non-obstructive pelvic floor and patent urethra which optimized by behavioral training and alpha-blockade. Li-ESWT has been shown to regenerate urethral sphincter tissue and pelvic floor muscles, which are crucial for maintaining urinary continence and efficient contraction of the pelvic floor. The therapy activates tissue-resident stem cells and modulates cellular signaling pathways, contributing to the regeneration of musculature in the urethra and pelvic floor (Lin and Lue, 2024).

While these multi-component outcomes are highly encouraging, this study is inherently limited by its single-case design (n=1), preventing the direct isolation of the individual statistical weight of Li-ESWT versus behavioral modifications. Consequently, future prospective randomized controlled trials with larger sample cohorts and extended follow-up parameters are required to firmly establish standardized wide-focus protocols and confirm generalizability for clinical guidelines in neuro-urology.

Conclusions

The present case suggests that combining Li-ESWT with behavioral modification and relaxation-based pelvic floor muscle training may offer a feasible non-invasive treatment for patients with medically refractory urinary and sexual dysfunction. By integrating regenerative, vascular, and neuromuscular rehabilitation, this multimodal approach addressed several pathophysiological mechanisms simultaneously rather than focusing on a single therapeutic target.

Recovery of bladder contractility was demonstrated by a substantial reduction in post-void residual (PVR) urine volume, from approximately 100–150 mL at baseline to 40–50 mL after treatment. As bladder emptying improved, the patient's reliance on clean intermittent catheterization (CIC) also decreased, with catheterization frequency falling from 3–4 to 1–2 times daily. In parallel, Li-ESWT may have contributed to improved penile vascular function, resulting in clinically meaningful recovery of erectile performance after conventional pharmacological therapy had failed to produce additional benefit.

Other favorable outcomes included an increase in functional voided volume to 380 mL, complete resolution of nocturnal dribbling, and maintenance of a non-obstructive peak urinary flow rate (Qmax) of 18.2 mL/s. These functional improvements were accompanied by greater independence in daily activities and a clear improvement in overall quality of life.

Although these findings are encouraging, they should be interpreted cautiously because they are based on a single patient. Larger prospective studies and randomized controlled trials are required to determine the reproducibility of these results, establish optimal treatment protocols, and further define the role of multimodal neuro-urological rehabilitation in patients with combined lower urinary tract and sexual dysfunction.

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