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*Original article*

# Efficacy and Safety of Low-Intensity Shockwave Therapy for Vasculogenic Erectile Dysfunction

**Mohamed Elzwawi[1,2], Salem Swieb[1,2]*, Moneer Badish[1,2]**

[1]*Department of Surgery, Faculty of Medicine, Misurata University, Misurata, Libya*
[2]*Al-Safwa Hospital, Misurata, Libya*
***Corresponding email.*** *s.swieb@med.misuratau.edu.ly*

**Abstract**
Vasculogenic erectile dysfunction (ED) is highly prevalent and frequently refractory to first-line therapies such as phosphodiesterase type 5 inhibitors (PDE5-Is). Low-intensity shockwave therapy (Li-SWT) has emerged as a regenerative modality that promotes angiogenesis and endothelial repair, offering potential long-term benefits. This study evaluated the efficacy and safety of Li-SWT in patients with vasculogenic ED unresponsive or partially responsive to PDE5-Is, and explored predictors of treatment response. In this prospective, open-label, single-arm trial, patients diagnosed with vasculogenic erectile dysfunction (ED) were enrolled between 2023 and 2024. Participants underwent 4 to 6 weekly sessions of low-intensity shockwave therapy (Li-SWT) using the Piezowave2 device, which delivered 4,000 shocks per session, complemented by a daily dosage of tadalafil at 5 mg. Efficacy was evaluated using the International Index of Erectile Function-5 (IIEF-5) and the Erection Hardness Score (EHS) at baseline and following the treatment. Twenty patients participated in this study, with a mean age of 55.9 years (±13.5). The mean baseline scores for the IIEF-5 and EHS were 10.9 (±4.5) and 2.1 (±0.8), respectively. Post-treatment, the IIEF-5 increased to 14.9 (±6.1), representing a mean difference of +3.95 (95% CI 1.56–6.34, p=0.002), and the EHS rose to 2.65 (±1.31), with a mean difference of +0.60 (95% CI 0.10–1.10, p=0.022). Six patients (30%) achieved “no ED,” while 40% displayed no clinically meaningful response. Responders (40%) were significantly younger (46.6 vs. 61.8 years, p=0.005), exhibited higher baseline IIEF-5 scores (14.1 vs. 8.8, p=0.003), and had fewer instances of hypertension (12.5% vs. 58.3%, p=0.047). Additionally, age showed a negative correlation with treatment response (r = −0.624, p=0.003). Li-SWT shows promise as a safe and moderately effective treatment for vasculogenic erectile dysfunction, especially in younger patients with mild to moderate baseline severity. Despite encouraging results, the lack of a control group, concurrent PDE5-I use, and small sample size limit generalizability.
**Keywords.** Erectile Dysfunction, Vasculogenic ED, Low-intensity Shockwave Therapy.

## Introduction

Erectile dysfunction (ED) is defined as the persistent inability to achieve or maintain an erection sufficient for satisfactory sexual activity. The term ED has replaced the older term "impotence" [1]. Erection requires a complex process involving sexual stimulation, the central nervous system, and a peripheral neuro-vascular pathway, involving hormones, which has to occur [2]. Six integrated vascular processes result in a penile erection: flaccidity, filling phase, tumescence, full erection, rigidity, and detumescence [3]. Vasculogenic ED is the subtype concerned with filling failure (arteriogenic) or venous leakage (venogenic) or a combination of both [4]. Low-intensity shockwave therapy (Li-SWT) is relatively novel for ED but has been used in other medical fields, including orthopedics, urology, cardiovascular, and gastrointestinal disorders, since the late 1970s [5]. As a non-invasive regenerative procedure, Li-SWT utilizes low-energy acoustic waves to induce angiogenesis and restore penile vascular function [6].

This is the first trial in Libya to evaluate Li-SWT in patients with vasculogenic ED who show poor response to phosphodiesterase type 5 (PDE5) inhibitors. There is a clinical need for alternatives to invasive treatments. Therefore, rigorous evaluation of Li-SWT is required to establish its safety, efficacy, and predictors of treatment response. Global statistics show statistics estimate that ED will affect approximately 322 million by 2025 [7]. Among men younger than 40 years old, the prevalence of ED ranges from 1% to 10%, mostly attributed to psychogenic etiology [8,9]. Specifically, the studies report that the prevalence among patients aged 29–30 is about 5.1% [8,9]. In contrast, ED prevalence increases substantially after age 40, which affects about 40.56% of men, with organic ED forming the majority [10]. Age-specific prevalence rates are approximately 14.8% in men aged 40–59, 44% in those aged 60–69, and more than 50% of patients over 70, with most of them considered to be of vasculogenic origin, since it represents the largest portion of organic ED [11,12].

Vasculogenic ED is a term inclusive of arterial causes, which relate to impaired arterial inflow, and venous causes, which relate to abnormality of corporal smooth muscle structure, or a combination of both, termed arteriovenogenic ED [13-15]. ED prevalence is particularly high among patients with vascular comorbidities, including coronary artery disease (CAD), hypertension (HTN), cerebrovascular disease, and peripheral arterial disease (PAD) [16-19]. This association is largely explained by atherosclerosis, where gradual arterial plaque buildup impairs penile blood flow [20]. Macrophages induce the formation of plaques in blood vessels, contribute to vascular injury, and impair penile perfusion [21]. Furthermore,


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Abnormal lipid metabolism and monocyte–macrophage interactions further accelerate atherosclerosis, which is strongly linked to ED development [22]. Reduced blood flow leads to relative hypoxia, which further leads to thickening and an increase in collagen and fiber content of vascular smooth muscles, which narrows the vessel lumen, causing ED [11]. Other conditions beyond atherosclerosis may also promote fibrosis of the cavernosal tissue, causing failure of the veno-occlusive mechanism, and subsequently, venous leakage occurs [23].

The International Index of Erectile Function-5 (IIEF-5) is a validated self-reported questionnaire that is considered the gold standard for ED treatment outcome measurement, regardless of the type of investigated treatment [24]. It consists of five items assessing erectile function over the past four weeks; each item can get a score ranging from one to five, making the overall range of 5-25 [25]. Severity is categorized as follows: severe ED in scores of 5-7, moderate ED for 8-11 scores, mild to moderate ED in 12-16 scores, mild ED in 17-21 scores, and no erectile dysfunction is denoted in 22-25 scores [25]. The self-reported Erection Hardness Score (EHS) is another commonly self-reported tool with a single-item [26]. It uses a four-point scale to measure the hardness of an erection: EHS1 = increase in size of penis, but no hardness; EHS2 = increase in size and slight increase in hardness, but insufficient for intercourse; EHS3 = increase in hardness sufficient for sexual intercourse, but not fully hard and rigid; EHS4 = fully rigid erection [26].

Laboratory testing alone cannot confirm the diagnosis of ED, yet it provides an opportunity to identify comorbid conditions that might be critical and influence management [27]. If there are no medical records in the past 12 months, lipid profile and hemoglobin A1c should be measured [28]. Hormonal evaluation should include total fasting testosterone in the early morning [28]. Prostate-specific antigen, pituitary hormone prolactin, and luteinizing hormone are not routinely conducted unless there are other indicative findings [28]. Color Doppler duplex ultrasound (CDDU), performed after intracavernosal vasoactive drug injection, is the first-line diagnostic tool for vascular arterial erectile dysfunction. It combines high-resolution imaging with pulse-Doppler assessment of flow, requiring the usage of values of peak systolic velocity (PSV), end diastolic velocity (EDV), resistance index (RI), and cavernous artery diameter to assess function. Variability may arise from measurement site, proximity, laterality, and the injection procedure itself, and incomplete relaxation of smooth muscles from anxiety or inadequate dosing can produce false positives [10]. Selective penile angiography is the choice when greater details of anatomical structures are required, due to its ability to visualize pelvic and penile vasculature, facilitating detection of traumatic arterial injury, stenosis, anatomic variants, and collateral networks [10]. Magnetic resonance arteriography (MRA) is a less expensive alternative that provides a high-resolution three-dimensional option that helps with proximal iliac and pudendal vessels yet is limited in evaluating distal pudendal and penile branches [10]. Advances such as virtual endoscopic reconstruction with multi-slice CT angiography (CTA) and 3D-CT cavernosography have improved noninvasive visualization of penile anatomy and venous drainage [10].

Dynamic infusion cavernosometry and cavernosography (DICC) combines cavernosometry (functional pressure/flow testing) and cavernosography (direct imaging of venous reflux), typically reserved for patients suspected of venous ED or for preoperative mapping of venous fistulae, and it records induced flow, maintained flow, and pressure decay. Ultrasound elastography is a sensitive technique for assessing tissue stiffness and has recently been refined to allow quantitative measurement [10]. Shear waves travel faster in stiffer tissue and slower in softer tissue [10]. Shear wave elastography (SWE) has recently been used to assess penile tissue stiffness in both healthy individuals and ED patients. Studies suggest it can objectively evaluate erection hardness and erectile function [10]. Despite having variable treatment options for ED, all of them are palliative in nature. Li-SWT is unique in that it aims to restore reperfusion and angiogenesis, aiming to restore the ability to achieve spontaneous erection [29]. The mechanism of action is thought to mimic fluid shear stress, stimulating vascular endothelial growth factor (VEGF) and other local factor expression to enhance local angiogenesis, particularly at low energy settings [30]. In vitro studies demonstrated that Li-SWT increased levels of VEGF and endothelial nitric oxide synthase (eNOS), and that caveolin-1 and ß1-integrin, constitutive proteins, are necessary for angiogenesis [30].

Since there are still questions regarding the reliability of evidence, the American Urological Association (AUA) stated that provided data are not sufficient to consider Li-SWT as standard treatment for ED patients and that it should be investigational with/without PDE5 inhibitors response [31]. The European Association of Urology has been more permissive, enlisting it in the treatment algorithm specifically for vasculogenic ED after PDE5 inhibitors [28]. However, the recommendation is based on weak evidence due to variability in trial designs and outcomes. The purpose of this open-label, single-arm, prospective clinical trial was to evaluate the safety and efficacy of Li-SWT in the treatment of vasculogenic ED. The study also explored associations with comorbidities and other factors influencing treatment efficacy at Al-Safwa Hospital.


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## Methods

### *Study Design and Patients*

In this open-label, single-arm, prospective study, patients with ED were enrolled between January 2023 and December 2024 at the Department of Urology, Al-Safwa Hospital, Misrata, Libya. All participants underwent physical examination and detailed medical history documentation. All patients provided written informed consent after being informed about Li-SWT based on contemporary literature regarding its use in the ED.

### *Inclusion and Exclusion Criteria*

#### *Inclusion criteria*

Adult married men (≥18 years) with known vasculogenic ED for at least one year duration, patient with baseline IIEF-5 score < 22 and refractory or partially responsive to PDE5 inhibitor treatment.

#### Exclusion criteria

Patient with a previous history of diagnosis of severe neurological ED, history of radical pelvic surgery, history of penile prosthesis and previous diagnosis of severe Peyronie's disease.

### *Li-SWT Protocol*

The treatment was performed using Piezowave2 of (Richard Wolf) with tadalafil 5 mg (Cialis®, Eli Lilly) once daily throughout the treatment course. A single experienced urologist performed all treatments. Each session, lasting approximately 8 minutes, was performed once per week for 4-6 consecutive weeks in the outpatient setting. In each treatment session, a total of 4000 shockwaves is divided into 2000 shockwaves each applied to the corpora cavernosa and the crus of the penis. Shockwaves were applied at an energy flux density of 0.2 mJ/mm² and a frequency of 8 Hz. By the end of the treatment course, patients had received a total of 16,000 shocks (4 sessions) or 24,000 shocks (6 sessions). This corresponded to a cumulative treatment time of approximately 33 or 50 minutes, with a total applied energy of 3,200 or 4,800 mJ. The variation of session number accounts for the varied condition of patients on initial assessment.

### *Assessment of Li-SWT Efficacy Using Erectile Function (EF) Indices*

Treatment response was assessed using two validated, self-reported questionnaires of erectile function: International Index of Erectile Function (IIEF), a patient-reported measure of erectile dysfunction and other sexual issues, and Erection Hardness Score (EHS). EF assessments were performed at baseline (pre-treatment) and post-treatment.

For treatment success, a "Responder" was defined as a patient achieving both: 1) an IIEF-5 score ≥ 17, and 2) an improvement of ≥4 points from baseline.

### *Data collection*

The full medical record included demographics of age and marital status, special habits of medical importance, history of comorbidities, findings of the clinical assessment, and important values of investigational (Laboratory and radiology) tests. Additionally, the assessment of Li-SWT efficacy and safety outcome collection is described above.

### *Statistical Analysis*

All statistical analyses were performed using Jamovi software (version 2.3.28). Descriptive statistics for all variables were conducted, with continuous data presented as mean ± standard deviation (SD), and categorical data summarized using frequencies and percentages. For comparative analyses, independent t-tests were used to compare means between groups when data were normally distributed, while the Mann-Whitney U test was applied for non-normally distributed variables. Statistical significance was defined as $p < 0.05$. Additionally, correlation analysis using Pearson's r was performed to explore the association between baseline variables and post-treatment IIEF-5 scores.

## Results

### *Characteristics of the Included Population*

A total of 20 patients were included in this study. The mean age was 55.9 (±13.5) with a range of 27.0 - 75.0 years. Most of the patients were smokers (85.0%), while 25.0% were regular alcohol consumers. The most prevalent comorbidity was Diabetes Mellitus (DM) affecting 12 patients (60%), followed by HTN 8 (40.0%), and other conditions combined (30%). The details are shown in (Table 1).

Regarding clinical assessment and investigation, the mean duration of ED for the population was 6.85 years, and 15 patients reported use of PDE5 inhibitors. The mean IIEF5 and EHS were 10.9 (range 5.0 – 18.0) and 2.1 (range 1.0 – 3.0), respectively. Laboratory testing showed that serum testosterone had a


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mean of 300 ng/dL with a range of 150 – 400 ng/dL. Penile Doppler revealed venous leakage in 3 patients (15%), while 17 (85%) had normal findings (Table 1).

***Table 1. Characteristics of Included Population (n = 20).***

| Variable | Value |
|---|---|
| **Age** | |
| Mean (±SD) | 55.9 (±13.5) |
| Range | 27.0 - 75.0 |
| **Habits of medical importance, n (%)** | |
| Smoking | 17 (85.0%) |
| Alcohol | 5 (25.0%) |
| **Comorbidities** | |
| DM | 12 (60.0%) |
| HTN | 8 (40.0%) |
| Heart Disease (CHD/IHD) | 3 (15.0%) |
| Others | 3 (15.0%) |
| Duration of ED (Years, mean (±SD)) | 6.85 (±4.02) |
| History of PDE5 inhibitors usage, n (%) | 15 (75.0%) |
| **IIEF-5** | |
| Mean (±SD) | 10.9 (±4.5) |
| Range | 5.0 - 18.0 |
| **EHS** | |
| Mean (±SD) | 2.1 (±0.8) |
| Range | 1.0 - 3.0 |
| Morning erection, n (%) | 11 (55.0%) |
| **S. testosterone** | |
| Mean (±SD) | 300 (±90) |
| Range | 150 - 400 |
| **S. prolactin** | |
| 0 | 17 (85.0%) |
| 7 | 1 (5.0%) |
| 8 | 2 (10.0%) |
| **Penile Doppler findings** | |
| Venous leakage | 3 (15.0%) |
| Normal | 17 (85%) |

***Li-SWT Significant Improvement of Erectile Function***

***Post-treatment Outcomes***

The entire population (n = 20) completed the treatment course. Mean IIEF-5 increased to 14.85 ± 6.14, and mean EHS increased to 2.65 ± 1.31. Patient satisfaction was reported by 11 patients (55%). Ten patients (50%) reported morning erections, and 11 (55%) continued tadalafil use (Table 2).

***Table 2. Post-treatment Outcomes.***

| Variable | Value |
|---|---|
| Total sessions per patient, n (%) | |
| Four sessions | 10 (50.0%) |
| Six sessions | 10 (50.0%) |
| IIEF-5 (Mean (±SD)) | 14.85 (±6.14) |
| EHS (Mean (±SD)) | 2.65 (±1.31) |
| Pt. satisfaction, n (%) | |
| Dissatisfied | 9 (45.0%) |
| Satisfied | 11 (55.0%) |
| Morning erection, n (%) | 10 (50.0%) |
| Continued tadalafil, n (%) | 11 (55.0%) |

***Significant Erectile Function Improvement Compared with Baseline***

As shown in (Figure 1), patients in the mild to moderate and moderate ED groups demonstrated improvement, with six patients (30%) achieving proper IIEF-5 scores to be categorized as "No ED".


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Although substantial improvement was observed, the severe group remained unchanged, with six (30%) patients.

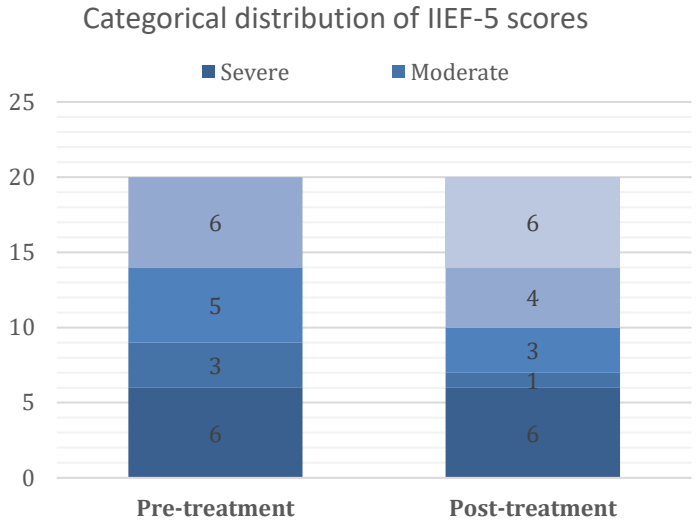


***Figure 1. Comparison of pre- and post-treatment IIEF-5 scores.***

(Table 3) shows that the mean IIEF-5 score significantly increased by +3.95 (95% CI 1.56–6.34, p=0.002). Also, the EHS had a significant improvement, with a mean change of +0.60 (95% CI 0.10 to 1.10, p = 0.022. In contrast, morning erection with the same value in both phases and a p-value of 1.000 showed no change, probably due to minimal conscious suppression or distraction (e.g. stress, anxiety, or environmental factors) which occur during sleep.

***Table 3. Analysis of pre- and post-treatment means of erectile function outcomes.***

| Outcome Measure | Pre-Treatment Mean ± SD | Post-Treatment Mean ± SD | Mean Difference (95% CI) | p-value |
|---|---|---|---|---|
| IIEF-5 | 10.90 ± 4.52 | 14.85 ± 6.14 | +3.95 (1.56 to 6.34) | p = 0.002 |
| EHS | 2.05 ± 0.89 | 2.65 ± 1.31 | +0.60 (0.10 to 1.10) | p = 0.022 |
| Patients with Morning Erections | 10 (55%) | 10 (55%) | 0 | p = 1.000 |

***Risk Factors and Predictors of Treatment Response***

***Responders and Non-responders***

Only eight out of 20 fulfilled criteria to be termed a responder, and 12 were non-responders, based on the total score. The mean age of the responder group compared to non-responders was markedly lower, expressed in a p-value of 0.005. A significant difference in baseline IIEF-5 score between the two groups, (14.1 ± 2.50) for responders and (8.8 ± 4.3) for non-responders. Hypertension was more prevalent among non-responders (58% vs 12.5%, p=0.047), suggesting it may reduce treatment efficacy. No statistically significant difference was observed between the two groups in venous leakage or DM (Table 4).

***Table 4. Comparison of risk factors between responders and non-responders.***

| Variable | Responders (n=8) | Non-Responders (n=12) | p-value |
|---|---|---|---|
| Age (years), Mean ± SD | 46.6 ± 10.8 | 61.8 ± 10.5 | p = 0.005 |
| Venous Leakage, n (%) | 0 (0%) | 3 (25%) | p = 0.244 |
| Baseline IIEF-5, Mean ± SD | 14.1 ± 2.5 | 8.8 ± 4.3 | p = 0.003 |
| DM, n (%) | 3 (37.5%) | 9 (75%) | p = 0.170 |
| HTN, n (%) | 1 (12.5%) | 7 (58.3%) | p = 0.047 |

***Predictors of IIEF-5 score***

Age demonstrated a strong negative correlation, thus older age predicts less improvement ($r = -0.624$, $p = 0.003$). Baseline IIEF-5 score showed a moderate negative correlation, which means a worse baseline ED predicts greater improvement ($r = -0.531$, $p = 0.016$). Finally, ED duration had a modest negative correlation, not enough to reach statistical significance ($r = -0.378$, $p = 0.101$).


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***Table 5. Correlation analysis (Pearson's r) of treatment predictors.***

| Variable | Correlation Coefficient (r) | p-value |
|---|---|---|
| Age vs. IIEF-5 | -0.624 | p = 0.003 |
| Baseline IIEF-5 vs. IIEF-5 | -0.531 | p = 0.016 |
| ED Duration vs. IIEF-5 | -0.378 | p = 0.101 |

## Discussion

This study achieved its primary aim of assessing efficacy and safety outcomes of Li-SWT in the treatment of vasculogenic ED by using two disease-specific scoring systems widely used to quantify treatment response. For a secondary outcome, analyses of risk factors were performed for a secondarily outcome of detecting negative and positive predictors of the pre-specified primary outcomes. Li-SWT demonstrated efficacy mainly in patients with mild to moderate ED. Analysis also provided evidence that younger age is a positive predictor affecting treatment response. Out of all the comorbidities recorded, only HTN showed a meaningful clinical implication.

Since Li-SWT targets local tissue to activate biological cascades that involve the expression of VEGF and eNOS, ultimately leading to angiogenesis, the condition of local tissue is the determinant of the magnitude of this reaction [30]. Fibrosis and loss of smooth muscle cells have always been a part of vasculogenic ED, whether it be arteriogenic or venogenic, particularly illustrated by prior observations in three cases of venous leakage [23]. Severely damaged condition of the erectile tissue can resist repetitive stimulations of SWT. Similar findings in the literature indicate that individuals with mild to moderate ED tend to show better responses than those with severe ED, regardless of study design [32,33]. With age, there is a progressive decrease in functional corporal smooth muscle. The mechanism(s) underlying this aging-related loss are believed to be due mainly to an apoptotic process that is primarily triggered by oxidative stress [34]. Aging is a distinct contributor to erectile dysfunction, responsible for another subtype of ED [34]. Therefore, when vasculogenic ED coexists with older age, prognosis is generally poorer [34].

The role of oxidative stress in explaining the pathogenic effects of different factors continues with HTN [35]. Endothelial dysfunction is a common link between HTN and ED [36]. During hypertension, the sustained release of pro-contractile factors impairs the balance between vasoconstrictors and vasodilators, leading to detrimental impairment of vascular and erectile structures. This pro-hypertensive state enhances the reactive oxygen species, with no internal antioxidant countering mechanisms [35]. Vascular and hypertensive anomalies support the development of ED. Another mechanism for oxidative stress is the innate immune system sustaining a low-grade inflammatory state, thus inducing oxidative stress [35]. There is also an additional effect of antihypertensive drugs on EF [36].

### *Strengths and Limitations*

This is the first study from Libya providing preliminary clinical data on Li-SWT treatment for the vasculogenic ED population with poor response to PDE5 inhibitors. Employment of internationally validated assessment tools (IIEF-5 and EHS) ensures reliable outcome measurement. The deployed standard treatment protocol was elaborated in all details of the treatment course, including the energy flux density, duration, frequency, and shock count mentioned concerning individual sessions and totals at the end of treatment. These details help reproducibility and allow comparisons with future studies [37]. The findings of the appropriate correlation analysis we conducted further aid policymakers through valuable clinical insights to develop helpful screening programs. The major limitation in this study is the absence of a control group; having a control group to compare outcomes with makes measurement more reliable. Being an open label may have affected patient reporting of outcomes, subsequently affecting the overall evidence. Although the protocol had high standards, concurrent administration of tadalafil 5mg limits the ability to solely attribute findings to Li-SWT. With 20 patients in this sample size, many clinically significant differences and risk factors could not undergo testing and analysis to contribute to the evidence generated; the existing evidence is vulnerable to selection bias and lack of generalizability. Finally, the short follow-up duration limits the conclusion about a long-term aspect to monitor the effect duration, as included in some of the previous literature [38].

## Conclusion

This prospective study demonstrated that LiSWT is both effective and safe in improving erectile function in patients with vasculogenic erectile dysfunction. Patients experienced significant improvement in erectile function scores, with benefits maintained during the six-month follow-up period. Importantly, no major adverse effects were reported, supporting the favorable safety profile of this therapy. These findings suggest that LiSWT may serve as a promising non-invasive treatment option for men with vasculogenic erectile dysfunction, particularly for those who are unresponsive or intolerant to conventional pharmacologic therapies. Nevertheless, the single-arm design and relatively small sample size limit the generalizability of the results, underscoring the need for larger, randomized controlled trials with extended


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follow-up to confirm efficacy, define patient selection criteria, and establish standardized treatment protocols.

***Conflict of interest***. Nil

## References

1. Mazzilli F. Erectile dysfunction: causes, diagnosis and treatment: an update. J Clin Med. 2022;11(21):6429. DOI: 10.3390/jcm11216429.
2. Salonia A, Briganti A, Dehò F, Naspro R, Scapaticci E, Scattoni V, et al. Pathophysiology of erectile dysfunction. Int J Androl. 2003;26(3):129-36. DOI: 10.1046/j.1365-2605.2003.00402.x.
3. Dean RC, Lue TF. Physiology of penile erection and pathophysiology of erectile dysfunction. Urol Clin North Am. 2005;32(4):379-95, v. DOI: 10.1016/j.ucl.2005.08.007.
4. Ostfeld RJ, Allen KE, Aspry K, Brandt EJ, Spitz A, Liberman J, et al. Vasculogenic erectile dysfunction: the impact of diet and lifestyle. Am J Med. 2021;134(3):310-6. DOI: 10.1016/j.amjmed.2020.10.007.
5. Porst H. Review of the current status of low intensity extracorporeal shockwave therapy (Li-ESWT) in erectile dysfunction (ED), Peyronie's disease (PD), and sexual rehabilitation after radical prostatectomy with special focus on technical aspects of the different marketed ESWT devices including personal experiences in 350 patients. Sex Med Rev. 2021;9(1):93-122. DOI: 10.1016/j.sxmr.2020.04.003.
6. Yahata K, Kanno H, Ozawa H, Yamaya S, Tateda S, Ito K, et al. Low-energy extracorporeal shock wave therapy for promotion of vascular endothelial growth factor expression and angiogenesis and improvement of locomotor and sensory functions after spinal cord injury. J Neurosurg Spine. 2016;25(6):745-55. DOI: 10.3171/2016.5.SPINE15923.
7. Ismail EA, El-Sakka AI. Innovative trends and perspectives for erectile dysfunction treatment: a systematic review. Arab J Urol. 2016;14(2):84-93. DOI: 10.1016/j.aju.2016.01.002.
8. Calzo JP, Austin SB, Charlton BM, Missmer SA, Kathrins M, Gaskins AJ, et al. Erectile dysfunction in a sample of sexually active young adult men from a US cohort: demographic, metabolic and mental health correlates. J Urol. 2021;205(2):539-44. DOI: 10.1097/JU.0000000000001363.
9. Nguyen HMT, Gabrielson AT, Hellstrom WJG. Erectile dysfunction in young men-a review of the prevalence and risk factors. Sex Med Rev. 2017;5(4):508-20. DOI: 10.1016/j.sxmr.2017.05.004.
10. Ma M, Yu B, Qin F, Yuan J. Current approaches to the diagnosis of vascular erectile dysfunction. Transl Androl Urol. 2020;9(2):709-21. DOI: 10.21037/tau.2020.01.04.
11. Pang K, Pan D, Xu H, Ma Y, Wang J, Xu P, et al. Advances in physical diagnosis and treatment of male erectile dysfunction. Front Physiol. 2023;13:1096741. DOI: 10.3389/fphys.2022.1096741.
12. Uddin SMI, Mirbolouk M, Dardari Z, Feldman DI, Cainzos-Achirica M, DeFilippis AP, et al. Erectile dysfunction as an independent predictor of future cardiovascular events: the Multi-Ethnic Study of Atherosclerosis. Circulation. 2018;138(5):540-2. DOI: 10.1161/CIRCULATIONAHA.118.033990.
13. Mulhall J, Barnas J, Aviv N, Anderson M, Parker M. Sildenafil citrate response correlates with the nature and the severity of penile vascular insufficiency. J Sex Med. 2005;2(1):104-8. DOI: 10.1111/j.1743-6109.2005.20110.x.
14. Hannan JL, Blaser MC, Pang JJ, Adams SM, Pang SC, Adams MA. Impact of hypertension, aging, and antihypertensive treatment on the morphology of the pudendal artery. J Sex Med. 2011;8(4):1027-38. DOI: 10.1111/j.1743-6109.2010.02178.x.
15. Yafi FA, Jenkins L, Albersen M, Corona G, Isidori AM, Goldfarb S, et al. Erectile dysfunction. Nat Rev Dis Primers. 2016;2:16003. DOI: 10.1038/nrdp.2016.3.
16. Azab SS, El Din Hosni H, El Far TA, Ismail NN, El Bakdady YK, Mohamed AF. The predictive value of arteriogenic erectile dysfunction for coronary artery disease in men. J Sex Med. 2018;15(6):880-7. DOI: 10.1016/j.jsxm.2018.03.008.
17. Koroglu G, Kaya-Sezginer E, Yilmaz-Oral D, Gur S. Management of erectile dysfunction: an under-recognition of hypertension. Curr Pharm Des. 2018;24(30):3506-19. DOI: 10.2174/1381612824666180813125813.
18. Ponholzer A, Gutjahr G, Temml C, Madersbacher S. Is erectile dysfunction a predictor of cardiovascular events or stroke? A prospective study using a validated questionnaire. Int J Impot Res. 2010;22(1):25-9. DOI: 10.1038/ijir.2009.37.
19. Polonsky TS, Taillon LA, Sheth H, Min JK, Archer SL, Ward RP. The association between erectile dysfunction and peripheral arterial disease as determined by screening ankle-brachial index testing. Atherosclerosis. 2009;207(2):440-4. DOI: 10.1016/j.atherosclerosis.2009.06.006.
20. Ibrahim A, Ali M, Kiernan TJ, Stack AG. Erectile dysfunction and ischaemic heart disease. Eur Cardiol. 2018;13(2):98-103. DOI: 10.15420/ecr.2018:8:2.
21. Marchio P, Guerra-Ojeda S, Vila JM, Aldasoro M, Victor VM, Mauricio MD. Targeting early atherosclerosis: a focus on oxidative stress and inflammation. Oxid Med Cell Longev. 2019;2019:8563845. DOI: 10.1155/2019/8563845.
22. Randrup E, Baum N, Feibus A. Erectile dysfunction and cardiovascular disease. Postgrad Med. 2015;127(2):166-72. DOI: 10.1080/00325481.2015.1015928.
23. Eardley I. Pathophysiology of erectile dysfunction. Br J Diabetes Vasc Dis. 2002;2(4):272-6. DOI: 10.1177/14746514020020040401.
24. Yule MA, Davison J, Brotto LA. The International Index of Erectile Function: a methodological critique and suggestions for improvement. J Sex Marital Ther. 2011;37(4):255-69. DOI: 10.1080/0092623X.2011.582931.
25. Neijenhuijs KI, Holtmaat K, Aaronson NK, Holzner B, Terwee CB, Cuijpers P, et al. The International Index of Erectile Function (IIEF)-a systematic review of measurement properties. J Sex Med. 2019;16(7):1078-91. DOI: 10.1016/j.jsxm.2019.04.010.


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26. Mulhall JP, Goldstein I, Bushmakin AG, Cappelleri JC, Hvidsten K. Validation of the erection hardness score. J Sex Med. 2007;4(6):1626-34. DOI: 10.1111/j.1743-6109.2007.00600.x.
27. Ghanem HM, Salonia A, Martin-Morales A. SOP: physical examination and laboratory testing for men with erectile dysfunction. J Sex Med. 2013;10(1):108-10. DOI: 10.1111/j.1743-6109.2012.02810.x.
28. Salonia A, Capogrosso P, Boeri L, Cocci A, Corona G, Dinkelman-Smit M, et al. European Association of Urology guidelines on male sexual and reproductive health: 2025 update on male hypogonadism, erectile dysfunction, premature ejaculation, and Peyronie's disease. Eur Urol. 2025;67(1):78-108. DOI: 10.1016/j.eururo.2024.09.019.
29. Gruenwald I, Appel B, Kitrey ND, Vardi Y. Shockwave treatment of erectile dysfunction. Ther Adv Urol. 2013;5(2):95-9. DOI: 10.1177/1756287212470696.
30. de Oliveira PS, Ziegelmann MJ. Low-intensity shock wave therapy for the treatment of vasculogenic erectile dysfunction: a narrative review of technical considerations and treatment outcomes. Transl Androl Urol. 2021;10(6):2617-28. DOI: 10.21037/tau-20-1298.
31. Burnett AL, Nehra A, Breau RH, Culkin DJ, Faraday MM, Hakim LS, et al. Erectile dysfunction: AUA guideline. J Urol. 2018;200(3):633-41. DOI: 10.1016/j.juro.2018.05.004.
32. Adeldaeim HM, Abouyoussif T, El Gebaly O, Assem A, Wahab MMA, Rashad H, et al. Prognostic indicators for successful low-intensity extracorporeal shock wave therapy treatment of erectile dysfunction. Urology. 2021;149:133-9. DOI: 10.1016/j.urology.2020.12.024.
33. Lu Z, Lin G, Reed-Maldonado A, Wang C, Lee YC, Lue TF. Low-intensity extracorporeal shock wave treatment improves erectile function: a systematic review and meta-analysis. Eur Urol. 2017;71(2):223-33. DOI: 10.1016/j.eururo.2016.05.050.
34. Ferrini MG, Gonzalez-Cadavid NF, Rajfer J. Aging related erectile dysfunction-potential mechanism to halt or delay its onset. Transl Androl Urol. 2017;6(1):20-7. DOI: 10.21037/tau.2016.11.18.
35. de Oliveira AA, Nunes KP. Hypertension and erectile dysfunction: breaking down the challenges. Am J Hypertens. 2021;34(2):134-42. DOI: 10.1093/ajh/hpaa148.
36. Javaroni V, Neves MF. Erectile dysfunction and hypertension: impact on cardiovascular risk and treatment. Int J Hypertens. 2012;2012:627278. DOI: 10.1155/2012/627278.
37. Wang M, Ma H, Shi Y, Ni H, Qin C, Ji C. Single-arm clinical trials: design, ethics, principles. BMJ Support Palliat Care. 2024;15(1):46-54. DOI: 10.1136/bmjspcare-2022-004048.
38. Fatima K, Abbas Z, Un-Noor A, Aaqil SI, Amir R, Nawaz F, et al. Efficacy of low-intensity extracorporeal shock wave therapy for erectile dysfunction: updated meta-analysis of randomized trials. Future Sci OA. 2025;11(1):2511438. DOI: 10.2144/fsoa-2024-0224.


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