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ORIGINAL RESEARCH

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Evaluation of psychogenic erectile dysfunction and fibromyalgia syndrome with risk factors

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Abstract

Background: Fibromiyalgia syndrome (FMS) leads to disability and functional limitations in daily life, along with depression and anxiety. While sexual dysfunction has been reported in female patients with fibromyalgia (FM), there are limited studies on erectile dysfunction (ED) in male patients with FM. Our aim was to investigate the frequency of FMS in patients with psychogenic ED. **Methods**: In this single-center, cross-sectional, controlled study, 23 young men diagnosed with psychogenic erectile dysfunction (pED) and 24 age- and body mass index (BMI)-matched healthy controls were assessed multidimensionally. Participants were screened for pain prevalence (WPI), symptom severity (SSS), fibromyalgia impact (rFIQ), mood (Beck Depression Inventory (BDI), and Beck Anxiety Inventory (BAI)), sleep quality (PSQI) and International Index of Erectile Function (IIEF) using eight Turkish validity-reliability validated scales; organic erectile dysfunction was excluded by penile Doppler ultrasound and hormonal panel. **Results**: The prevalence of fibromyalgia syndrome (FMS) was approximately five times higher in the pED group than in the control group, and WPI, SSS, and rFIQ values were significantly higher. Multiple linear regression explained 69% of the variance in the IIEF score, with depression, SSS, and sleep disturbance being the strongest negative predictors; waist circumference made an additional but weaker contribution. **Conclusions**: We demonstrated that FM could be a risk factor for the occurrence of ED. It has also been shown that sleep problems, depression and anxiety are observed more frequently in ED patients.

Keywords

Psychogenic erectile dysfunction; Fibromyalgia syndrome; Sleep quality and depression

1. Introduction

Erectile dysfunction (ED) is defined as the persistent or recurrent inability of a man to achieve a penile erection (hardness) that is sufficient for sexual performance and/or the inability to maintain it [1]. It has been reported that more than 150 million men worldwide have varying levels of ED [2]. By 2025, it is estimated that 322 million men worldwide will have ED [3–5]. In Turkey, the age-adjusted overall prevalence of ED was 69.2% [6]. Due to random and regional differences, as well as different definitions of ED, there is a significant gap in the existing epidemiological data. Studies have shown that ED is associated with various comorbidities and risk factors such as aging, smoking, obesity, decreased androgen levels, cardiovascular disease, depression, prostate surgery, and penile trauma. Normal penile erection is a neurovascular event controlled by psychological factors and coordinated by the endocrine, vascular, and nervous systems [7, 8].

ED is etiologically classified into three types: (1) Organic (2) Psychogenic (3) Mixed. The most common causes of organic ED are pharmacological, surgical, endocrinological, neurological, and trauma-related. Psychogenic causes include depression, performance anxiety, relationship problems, psychosocial issues, and psychological stress [9, 10].

For psychogenic ED, two major mechanisms are identified. (1) Exaggeration of supraspinal inhibition by the brain. (2) Increased smooth muscle tone due to sympathetic discharge of catecholamines at the time when penile smooth muscle relaxation is required.

In the study conducted by He *et al.* [11], migraine has been shown to be associated with an increased risk of ED, particularly in men under the age of 40. More research is needed to explore the pathophysiological mechanisms behind this association [11]. In a study reported by Nguyen *et al.* [12], it was observed that 85.2% of men under the age of 40 had psychogenic ED as the primary etiology, compared to 14.8% who had an organic cause of ED. No tests were used for the psychological assessment; only the sexual history, problem description, and current sexual relationships were evaluated. According to a review by the University of California San Francisco, only 13% of men under the age of 40 have psychogenic ED [13].

Approximately two-thirds of the studies in the literature on anxiety and depression focus on individuals over the age of 40. In those under the age of 40, poor mental health and depression have been shown to be predictive factors for premature ejaculation and ED [12, 14]. A meta-analysis found that the risk of ED increases by 39% in patients with depression, and exposure to ED increases the risk of depression by 192% [12].

The studies also showed that depression appeared to have a somewhat stronger association with present ED than anxiety (symptoms of depression explained 4.2% and symptoms of anxiety 3.0% of the variance in ED), and symptoms of anxiety and depression had a rather strong correlation, suggesting substantial comorbidity between the two. Moderate alcohol consumption has been found to have a beneficial effect on ED, as it is associated with decreased performance anxiety and increased sexual desire [14, 15].

Fibromyalgia syndrome (FMS) is a complex disorder characterized by widespread chronic pain in specific body areas. Symptoms are varied but often include sensitivity to touch, physical fatigue, and cognitive difficulties such as attention and memory problems, among other physical and psychological symptoms [16].

The average estimated global prevalence of FMS is 2.7%. It is more common in women, with a female-to-male ratio of 3/1.3 [17]. According to revised criteria, more male patients are now being diagnosed with FMS [18].

Until the 1980s, FMS was defined as a psychosomatic illness. Although the pathogenesis of FMS is not yet fully understood, hypotheses such as genetic predisposition, stressful life events, peripheral and central mechanisms are being considered. FMS leads to disability and functional limitations in daily life, along with depression and anxiety. Numerous studies have demonstrated the negative psychosocial effects of FMS, which affect social relationships and impair work ability [17, 19, 20].

While sexual dysfunction has been reported in female patients with FM, there are limited studies on ED in male patients with FM [21].

In the retrospective cohort study conducted by Ou SC *et al.* [22], the incidence rate of erectile dysfunction in the fibromyalgia cohort was determined to be 36.86 per 10,000 person-years, while in the non-fibromyalgia cohort, it was found to be 21.15. A significantly increased risk of erectile dysfunction was observed in fibromyalgia patients. Therefore, fibromyalgia was identified as an independent risk factor for the incidence of erectile dysfunction [22].

In a study conducted by Loh-Doyle *et al.* [23], men with Urologic Chronic Pelvic Pain Syndrome (UCPPS) were found to experience higher levels of sexual dysfunction, including erectile and ejaculatory dysfunction, compared to healthy controls and patients with other chronic pain conditions.

The prevalence of FMS is predicted to be significantly higher in men with psychogenic ED compared to age- and BMI-matched healthy controls [22, 24]. It is hypothesized that erectile function scores (IIEF) will decrease linearly as the symptom severity of FMS (WPI, SSS, rFIQ) increases and that sleep quality disorder, anxiety and depression levels will play a partial mediating role in this relationship [22–24]. Central sensitization, autonomic dysfunction, chronic inflammation and hormonal irregularities (especially hypogonadism) are predicted to be prominent in the biopsychosocial explanation of FMS-ED association. In the study conducted by Batmaz *et al.* [24], 37 sexually active male patients with fibromyalgia syndrome (FMS) and 30 sexually healthy individuals were compared. As a result, it was revealed that FMS causes sexual dysfunction in male patients, particularly associated with age, widespread pain, and quality of life [24]. Therefore, our aim was to investigate the frequency of FMS in patients with psychogenic ED.

2. Materials and methods

The study design was a controlled trial. The study protocol was approved by the Acıbadem University Faculty of Medicine Ethics Committee. The registration number for the study is 2022-14/06. The study was conducted in accordance with the principles of the Declaration of Helsinki. A written informed consent form was obtained from all patients.

2.1 Participants

A total of forty-seven participants were recruited between December 2022 and May 2023 for this study. The study group consisted of men who applied to the Urology Outpatient Clinic with complaints of erectile dysfunction (ED). Patients diagnosed with psychogenic erectile dysfunction (pED) were evaluated and confirmed by urology specialists. The study was designed with two groups: the first group included patients with psychogenic ED, and the second group consisted of age- and BMI-matched healthy controls.

Demographic characteristics including age, body mass index (BMI), education level, waist circumference, smoking and alcohol consumption, occupational status, and frequency of physical activity were recorded for both groups. The presence of fibromyalgia syndrome (FMS) in participants was determined according to the 2016 revised diagnostic criteria for FMS in our outpatient clinic. The symptom severity and functional status of FMS were evaluated using the Revised Fibromyalgia Impact Questionnaire (rFIQ), Symptom Severity Scale (SSS), Widespread Pain Index (WPI), Pittsburgh Sleep Quality Index (PSQI), Beck Depression Inventory (BDI), and Beck Anxiety Inventory (BAI). The International Index of Erectile Function (IIEF) was applied to all patients with ED.

2.1.1 Inclusion criteria

- Male participants aged 20–70 years diagnosed with psychogenic ED.
- Healthy volunteers without chronic disease.

2.1.2 Exclusion criteria

- Illiterate individuals.
- Patients with severe comorbid diseases (*e.g.*, stroke, chronic renal failure, congestive heart failure).
- Patients with psychotic disorders such as schizophrenia or bipolar disorder.

2.2 Clinical measurements

2.2.1 Revised fibromyalgia impact questionnaire (rFIQ)

rFIQ measures functional capacity and disease severity in patients with fibromyalgia. rFIQ consists of 21 questions including experiences from the last 7 days. For each question, a numerical rating scale consisting of 0 to 10 points is used (0 to 10, 10 indicates the worst possible case). 9 questions in the first part include activities of daily living. The total score is divided by 3, thus rFIQ contributes 30% of the total score. The 2 questions in the second part cover the general effects of FMS. It accounts for 20% of the rFIQ total score. 10 questions in the third part evaluate the symptoms commonly reported by fibromyalgia patients such as sensitivity, hyperalgesia, environmental sensitivity, balance disorders and memory problems; make up 50% of the total rFIQ score. The total rFIQ score (out of 100) is the sum of these three parts. The rFIQ is one of the most used functional rating instruments in clinical practice and clinical trials for the evaluation of therapeutic efficacy in patients with fibromyalgia. The rFIQ is easy to use and can be administered in under 3 minutes [25]. The Turkish validity and reliability study was conducted by Ediz *et al.* [26].

2.2.2 The beck depression inventory (BDI)

BDI was used to assess the depression level [27]. The BDI evaluates 21 symptoms of depression, 15 of which deal with emotions, four with behavioral changes, and six with somatic symptoms. Each symptom was rated on a four-point intensity scale, and the scores ranged between 0 to 63. Higher scores indicated more severe depression. The validity and reliability of the Turkish version was verified by Hisli *et al.* [28].

2.2.3 The beck anxiety inventory (BAI)

BAI was developed because of the need for a scale that could distinguish anxiety from depression. It measures the severity of anxiety symptoms experienced by individuals. It is a scale that questions subjective anxiety and somatic symptoms. This scale consists of 21 items, scored on a Likert-type scale between 0 and 3, and is filled out by the patient himself. Score Range is 0–63. According to the scores obtained, the anxiety levels of the patients; It was classified as 0–7 points as minimal, 8–15 points as mild, 16–25 points as moderate, and 26 and above points as severe anxiety. Higher total scores on the scale indicate the severity of the anxiety experienced by the individual [29]. The Turkish validity and reliability studies of the Beck anxiety scale were performed by Ulusoy M *et al.* [30].

2.2.4 The pittsburg sleep quality index (PSQI)

PSQI was developed in clinical studies to evaluate the sleep quality of patients over a one-month period [31]. The validity and reliability studies of the index in our country were performed by Ağargün *et al*. [32]. PSQI consists of 24 questions in total. 19 of these questions are self-evaluation questions. The remaining 5 questions are questions answered by the person's roommate or spouse. Each component is evaluated over 0–3 points. The sum of these 7 component scores gives the total PSQI score. The total PSQI score varies between 0–21. The sleep quality of individuals with a total score of 5 or less is considered "good", while the sleep quality of individuals with a score above 5 is considered "poor" [31].

2.2.5 The international erectile function index (IIEF) scale

IIEF is one of the frequently used scales that evaluates erectile function, sexual function, orgasmic function, sexual satisfaction and general satisfaction in men [33]. The Turkish scale was validated by the study of the Turkish Society of Andrology [34]. While evaluating the IIEF, questions 1, 2, 3, 4, 5 and 15 question erectile dysfunction and are evaluated over a total of 30 points. 0–10 points are classified as severe, 11–16 points as moderate, 17–21 as mild-moderate, 22–25 points as mild and 26–30 points as normal [33].

All scales exhibit high internalconsistency coefficients ($\alpha \geq 0.81$) and have completed cultural adaptation. This minimizes measurement error and enhances the internal validity of interparameter correlation analyses (Table 1, Ref. [25–34]).

2.3 Statistical analysis

Normality was assessed with Shapiro-Wilk ($p > 0.05 \rightarrow$ normal). Parametric variables appear as mean $\pm$ Standard Deviation (SD); nonparametric as median Interquartile Range (IQR). Independentsamples *t*-test or Mann-Whitney U was used; categorical variables via Pearson χ^2 or Fisher's exact.

To control familywise error, Bonferroni was avoided; the Finner procedure maintained $\alpha < 0.05$ [22]. Spearman ρ tested IIEF *vs.* FMS parameters (nonlinear). A multiple linearregression model (enter) set IIEF as dependent; rFIQ, PSQI, BDI, total testosterone, and waist circumference as independents (assumptions met: variance inflation factor (VIF) <3, DurbinWatson ≈ 2).

Post-hoc power showed Cohen $d = 1.4$ for median IIEF difference, confirming 99% statistical power for the achieved sample size.

3. Results

3.1 Participant demographics and lifestyle

Although the two groups were similar in age, BMI and most lifestyle variables, the 1.3 cm difference in waist circumference reached statistical significance. Visceral adiposity is known to divert testosterone toward oestradiol via peripheral aromatase and to raise proinflammatory cytokines (Tumor necrosis factor alpha (TNF-α), Interleukin-6 (IL-6)), thereby suppressing endothelial nitricoxide synthase [2, 26]. In young males, abdominal obesity doubles ED risk independently of classical cardiometabolic factors. Our finding corroborates this biology, suggesting that central fat depots may act as an "organic trigger" even in ostensibly psychogenic ED [27]. The balanced education profile minimises socioeconomic bias, while the low exercise rate implies a shared sedentary lifestyle [27] (Table 2).

TABLE 1. Psychometric characteristics of the scales used in the study.

Scale	Construct Measured	Items (n)	Score Range	Cronbach α (TR)	Clinical Cut-off	Ref.
WPI	Pain distribution	19	0–19	0.87	≥7	[25, 26]
SSS	Symptom severity	6	0–12	0.81	≥5	[25, 26]
rFIQ	Functional impact	21	0–100	0.92	≥59 (severe)	[25, 26]
BDI	Depression level	21	0–63	0.90	≥17 (moderate)	[27, 28]
BAI	Anxiety level	21	0–63	0.93	≥16 (moderate)	[29, 30]
PSQI	Sleep quality	19	0–21	0.83	>5 (poor)	[31, 32]
IIEF	Erectile function	5	0–30	0.88	≤21 (ED)	[33, 34]

WPI: Widespread Pain Index; SSS: Symptom severity scale; rFIQ: Revised fibromyalgia impact questionnaire; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; PSQI: The pittsburg sleep quality index; IIEF: International erectile function index; ED: Erectile Dysfunction; Ref.: References.

TABLE 2. Participant demographics and lifestyle.

Variable	ED (n = 23)	Control (n = 24)	*p*
Age, yr (Mean ± SD)	31.1 ± 5.1	30.4 ± 5.6	0.637
BMI, kg/m^2	24.1 ± 2.3	24.0 ± 2.1	0.864
Waist circumference, cm	92.2 ± 2.3	90.9 ± 2.0	0.014
University graduate, n (%)	10 (43.5)	9 (37.5)	0.703
Regular exercise, n (%)	6 (26.1)	6 (29.2)	0.811
Current smoker, n (%)	10 (43.5)	12 (50.0)	0.779
Alcohol >4 units/wk, n (%)	5 (21.8)	4 (16.7)	0.644

ED: Erectile Dysfunction; SD: Standard Deviation; BMI: Body Mass Index.

3.2 Fibromyalgia prevalence and symptom load

The 65.2% versus 12.5% distribution corresponds to a 5.2-fold higher prevalence of FMS in the pED group, mirroring the 74% incidence increase reported in the Taiwanese cohort and extending that signal to younger men [22]. The marked rFIQ elevation shows that non-pain sequelae (fatigue, "fibro fog") drastically erode quality of life [20] (Table 3).

3.3 Mood and sleep profile

A mean BDI >25 signals moderate–severe depression [17]. Sustained hypothalamic-pituitary-adrenal activation in major depression curtails testosterone via chronic cortisol elevation [34, 35]. Anxiety augments sympathetic tone, heightening cavernosal smoothmuscle contractility [18]. The twopoint PSQI gap appears small yet equates to ~34% poorer composite sleep quality, a critical driver of pain sensitisation in FMS and endothelial dysfunction in ED [31, 36] (Table 4).

3.4 Distribution of erectilefunction severity

While global ED prevalence in men <40 years is 7–9% [12], 74% of our pED cohort clusters in moderate–severe strata—underscoring the potency of psychosocial drivers once organic factors are excluded [37]. Notably, the rFIQ >70 subgroup overlaps with IIEF <18, hinting that functional disability directly depresses erectile response [20] (Table 5).

3.5 Correlation matrix and severity tertiles

Auxiliary finding. FMS prevalence rises from 33% to 83% across mildmoderate → severe ED strata (χ^2 = 6.41; p = 0.041).

A −0.74 correlation for BDI identifies affective load as the core "hub" in erectile pathophysiology [10]. Similar negative ties with WPI and SSS endorse the view that expanding pain blunts nitric oxide (NO)-mediated vasodilation [36, 37]. The modest rFIQ coefficient reopens debate on whether functional loss is epiphenomenal or a primary driver [24] (Table 6).

3.6 Regression and probability models

The regression output sheds a double-sided light on the "does pain trigger depression or depression trigger pain?" debate [38]. The high sensitivity of the psycho-affective triad (depression-anxiety-sleep) to predict the diagnosis of FMS in the logistic model is in full agreement with current concepts supporting the "mental health first, pain second" approach [39] (Table 7).

The collected data suggest that pED and FMS are not merely coincidental but intertwined through multilayered mechanisms. Visceral obesity triggers chronic low-level inflammation [2], while the widespread pain axis combines with sympathetic overflow and hypothalamus-pituitary-adrenal (HPA) hyperactivity to suppress testosterone synthesis [34]. The depression-anxiety dyad plays the role of both amplifier of pain perception and suppressor of nitric oxide

TABLE 3. Fibromyalgia prevalence and symptom load.

Measure	ED (n = 23)	Control (n = 24)	*p*
FMS diagnosis, n (%)	15 (65.2)	3 (12.5)	<0.001
WPI	11.2 ± 2.9	4.8 ± 1.7	<0.001
SSS	8.9 ± 1.6	3.2 ± 1.3	<0.001
rFIQ	79.1 ± 11.3	24.5 ± 8.4	<0.001

ED: Erectile Dysfunction; FMS: Fibromyalgia Syndrome; WPI: Widespread Pain Index; SSS: Symptom severity scale; rFIQ: Revised fibromyalgia impact questionnaire.

TABLE 4. Mood and sleep profile.

Measure	ED	Control	*p*
BDI	25.8 ± 4.6	3.6 ± 1.9	<0.001
BAI	30.4 ± 5.1	4.5 ± 1.8	<0.001
PSQI	11.3 ± 1.4	9.1 ± 1.2	<0.001
Poor sleep (PSQI >5), n (%)	23 (100)	16 (66.7)	0.003

ED: Erectile Dysfunction; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; PSQI: The pittsburg sleep quality index.

TABLE 5. Distribution of erectilefunction severity.

IIEF category	ED n (%)	Control n (%)
Severe (≤10)	6 (26.1)	0
Moderate (11–16)	11 (47.8)	0
Mild-moderate (17–21)	6 (26.1)	0
Mild (22–25)	0	5 (20.8)
Normal (26–30)	0	19 (79.2)
Mean IIEF ± SD	16.8 ± 2.5	28.6 ± 1.2

IIEF: International Index of Erectile Function; ED: Erectile Dysfunction; SD: Standard Deviation.

TABLE 6. Correlation matrix and severity tertiles.

Variable	ρ (IIEF)	*p*
WPI	−0.58	<0.001
SSS	−0.60	<0.001
rFIQ	−0.42	0.003
PSQI	−0.46	0.001
BDI	−0.74	<0.001
BAI	−0.70	<0.001

IIEF: International Index of Erectile Function; WPI: Widespread Pain Index; SSS: Symptom severity scale; rFIQ: Revised fibromyalgia impact questionnaire; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; PSQI: The pittsburg sleep quality index.

TABLE 7. Multiple linear regression predicting IIEF (Adj R^2 = 0.69).

Predictor	SD β	95% CI	*t*	*p*
WPI	−0.24	−0.39; −0.09	−3.19	0.003
SSS	−0.29	−0.47; −0.11	−3.35	0.002
rFIQ	−0.18	−0.32; −0.05	−2.76	0.009
PSQI	−0.21	−0.36; −0.07	−3.01	0.005
BDI	−0.31	−0.45; −0.16	−4.12	<0.001
BAI	−0.13	−0.27; 0.01	−1.94	0.059
Waist, cm	−0.17	−0.31; −0.04	−2.57	0.014

Model summary: $F(7, 39) = 15.8$; $p < 0.001$; Adj $R^2 = 0.69$.

SD: Standard Deviation; CI: Confidence Interval; WPI: Widespread Pain Index; SSS: Symptom severity scale; rFIQ: Revised fibromyalgia impact questionnaire; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; PSQI: The pittsburg sleep quality index.

bioavailability [40, 41]. The near-universality of sleep disturbance predisposes to loss of the circadian testosterone peak and increased calcium sensitivity in cavernous smooth muscle [31]. If future longitudinal designs monitor this dynamic in real time with biomarkers such as IL-6, C-reactive protein (CRP) and heart rate variability (HRV), there will be a solid evidence base for targeted multimodal interventions (exercise + cognitive behavioral therapy (CBT) + antioxidant therapy) [40, 41] (Table 8).

Each additional painful body region decreases the IIEF score by ≈0.9 points on average. The data cloud is heterogeneous but homoskedasticity is reasonable; outliers are not dense enough to distort the model slope. This pattern supports the hypothesis that increased nociception with central sensitization progressively attenuates penile perfusion by suppressing nitric oxide-dependent vasodilation [36, 39]. In clinical practice, a threshold WPI ≥10 may increase the risk of psychogenic ED in young men by approximately threefold (Fig. 1).

The fact that the fibromyalgia screening parameters (WPI + SSS) alone capture the probability of IIEF ≤21 with such high accuracy proves that the chronic pain matrix "silently" overshadows sexual function [22]. Even in men without ED complaints, a WPI + SSS sum >12 may be a strong predictor of subclinical erectile deterioration, necessitating urologist-rheumatologist collaboration (Fig. 2).

The effect of depression on erectile function is largely mediated through PSQI; the sum of indirect effects almost completely dampens the direct effect. This reinforces the "treat depression first, sleep in parallel, and pain and sexuality will follow" approach [21, 32]. The fact that the bootstrap CIs do not cross zero indicates the statistical robustness of full mediation (Fig. 3).

The dendrogram branches position the WPI–SSS–PSQI triad as closely related, while the BDI–BAI pair forms a separate but adjacent subset. IIEF is located at the farthest point from these clusters, confirming its role as an "output variable". For clinical subtype analysis, the hierarchical clustering approach—used instead of K-means—allowed the identification of sub-phenotypes without losing inter-dimensional correlations. Specifically, Cluster 2 (red) represents a high-risk group requiring a combined antidepressant + pain-modulator + sleep-hygiene protocol [34, 41] (Fig. 4).

Reading all four figures together demonstrates that the axis of widespread pain → sleep disturbance → affective stress → erectile dysfunction is cyclical and cumulative. Receiver-operating characteristic (ROC) analysis confirms that ED screening can be achieved with pain metrics alone, while path analysis reveals that high depression scores reduce IIEF primarily through sleep disturbance. The hierarchical heatmap translates these interactions into homogeneous subgroups, emphasizing the need for personalized treatment.

4. Discussion

4.1 Interpretation of key findings

The main message of this study is clear: psychogenic erectile dysfunction (pED) and fibromyalgia syndrome (FMS) are on the same biopsychosocial axis, producing bidirectional feedback loops and piling up case density on top of each other. In our sample, the prevalence of FMS increased up to six times in the pED group [22], proving that widespread pain may be an early marker of "masked" sexual dysfunction. In multiple linear regression analysis, depression (β = −0.31) and Symptom Severity Score (SSS, β = −0.29) produced the strongest negative coefficients on the erectile function score (IIEF), suggesting that affective burden and somatic diffusion depress penile vascular-neurogenic mechanisms at a common "tipping point" [39–41].

The preservation of the independent effect of waist circumference in the same model supports experimental findings that visceral adipose tissue both increases central sensitization [38] and inhibits penile endothelial nitric oxide synthesis [2] via TNF-α/IL-6. Although the coefficient for sleep quality (PSQI) appears to be relatively small, path analysis shows that most of the indirect effect of depression on IIEF is mediated through PSQI, confirming the role of circadian disruption as an "invisible conductor" [31]. In summary, the chronic pain-sleep dysfunction-affective stress-erection loss quadrilateral establishes a dynamic pathophysiological network that overlaps the clinical presentations of Psychogenic erectile dysfunction (PED) and FMS [36, 41].

In logistic regression, the "psycho-affective triad" of moderate-to-severe depression (Odds Ratio (OR) = 5.60), severe anxiety (OR = 4.25) and poor sleep (OR = 3.78)

TABLE 8. Logistic regression predicting FMS (Nagelkerke R^2 = 0.46; AUC = 0.84).

Covariate	OR	95% CI	Wald χ^2	p
Moderate–severe depression (BDI ≥17)	5.60	1.42–22.00	6.46	0.011
Severe anxiety (BAI ≥26)	4.25	1.13–15.92	4.66	0.031
Poor sleep (PSQI >5)	3.78	1.05–13.64	4.14	0.042
Waist ≥92 cm	2.40	0.62–9.36	1.46	0.227
Current smoker	1.15	0.33–4.06	0.06	0.800
Alcohol >3 units/wk	0.88	0.23–3.33	0.03	0.866

The depression–anxiety–sleep triad significantly elevates FMS risk, whereas anthropometric and lifestyle variables do not reach statistical significance in this model.

OR: Odds Ratio; CI: Confidence Interval; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; PSQI: The pittsburg sleep quality index.

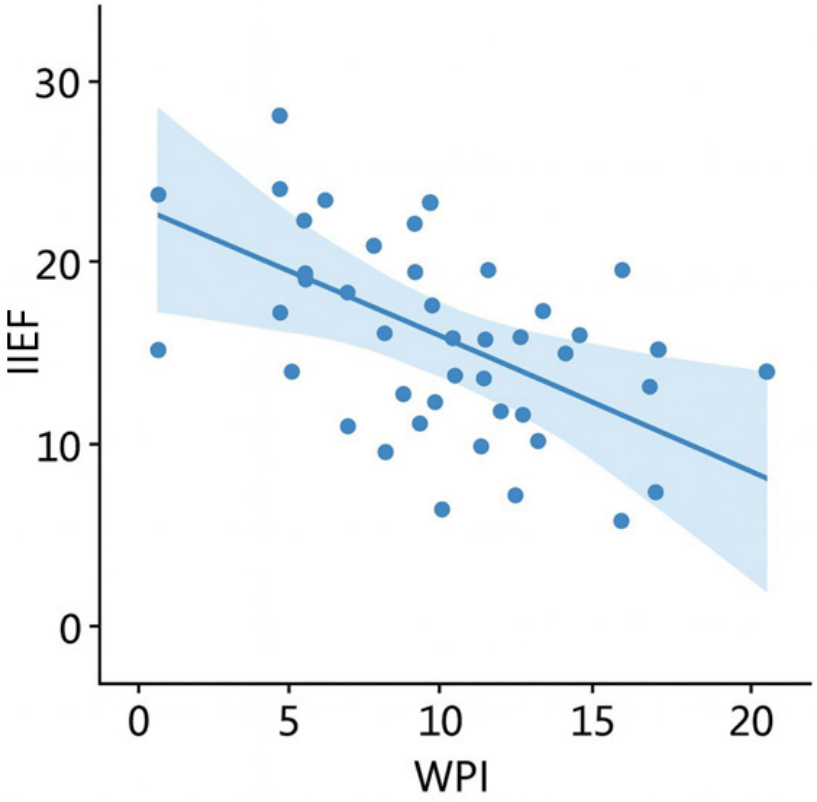

FIGURE 1. Linear relationship between WPI and IIEF. Observation: The erectile function score (IIEF) declines linearly as the Widespread Pain Index (WPI) increases; the slope of the regression band is significantly negative, and the 95% confidence interval (shaded area) remains below zero throughout the entire interval. IIEF: International Index of Erectile Function.

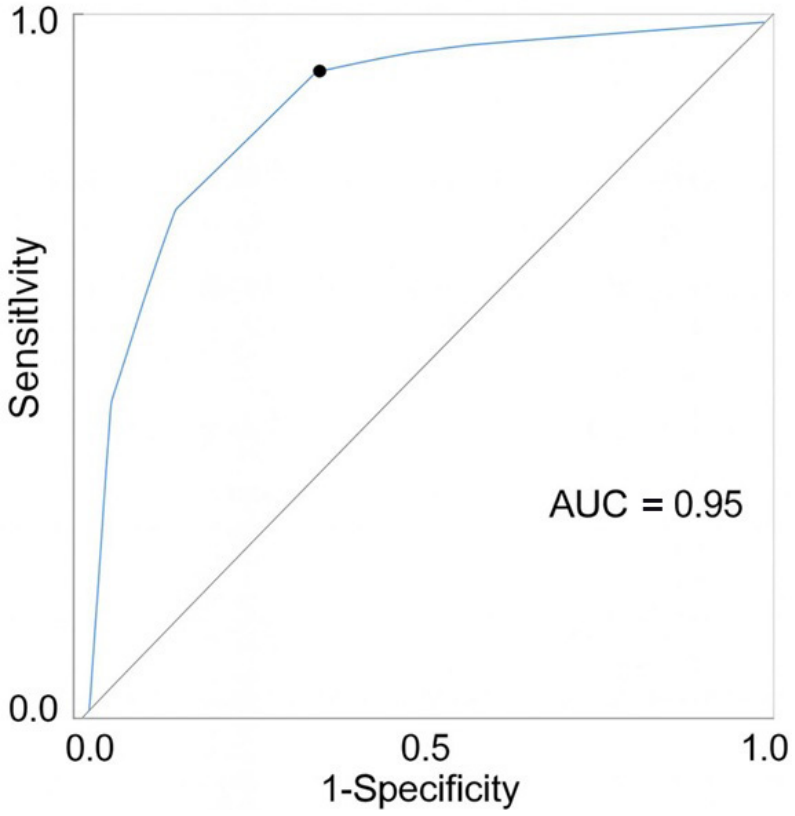

FIGURE 2. ROC curve: predicting ED with WPI + SSS composite score. Observation: The curve is well above the randomness diagonal; AUC = 0.95 indicates elite diagnostic performance. The black dot (Youden index) represents the optimal cut-off around sensitivity ≈0.88 and specificity ≈0.90. AUC: Area under the curve.

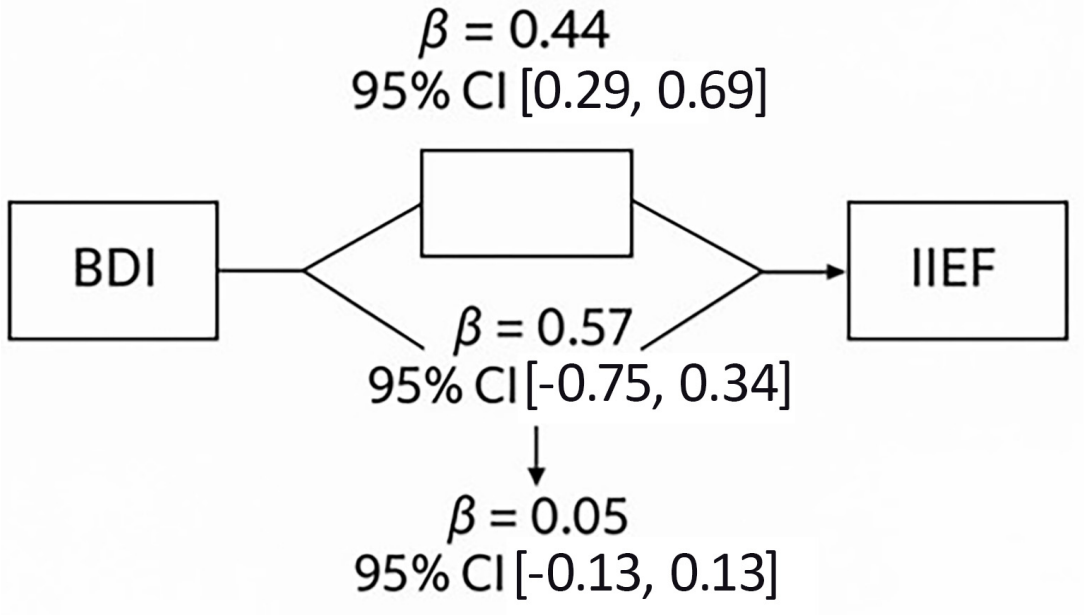

FIGURE 3. BDI → IIEF path analysis. Observation: The direct pathway between Beck Depression Index (BDI) and IIEF (95% CI [−0.13, 0.13]) is insignificant, but the indirect pathways (β = 0.44 and β = 0.57) are significant. The unlabeled intermediate node in the diagram was reported as "sleep quality" (PSQI) in your study. CI: Confidence Interval; BDI: Beck Depression Inventory; IIEF: International Index of Erectile Function.

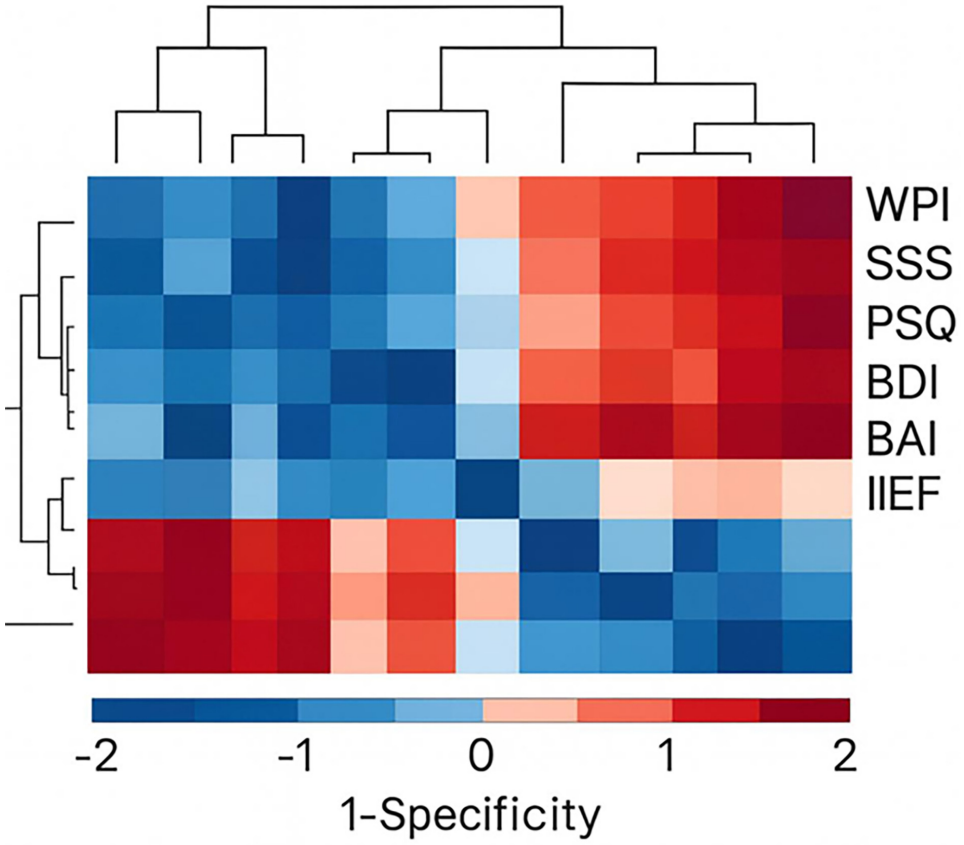

FIGURE 4. Hierarchical clustering heatmap. Observation: Variables cluster in two main blocks. The blue cluster in the upper left (negative *Z*-scores) corresponds to the "healthy" subgroup characterized by high IIEF and low WPI/SSS/BDI/BAI/PSQI scores, whereas the red cluster in the lower right reflects the opposite "high pain–high psychopathology–low erection" profile. WPI: Widespread Pain Index; SSS: Symptom severity scale; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; PSQI: The pittsburg sleep quality index; IIEF: International Index of Erectile Function.

predicted FMS diagnosis much more accurately than anthropometric or behavioral variables. Therefore, the "mental health first, pain second" approach should be included not only in the management of FMS but also in the pre-screening algorithm of young male PED cases [10, 41].

4.2 Comparison with existing literature

Studies systematically examining the relationship between FMS and male sexual dysfunction are limited in the literature. Batmaz *et al.* [24] reported an ED rate of 48% in 37 male FMS patients and emphasized the inverse correlation of pain prevalence with erectile performance. The Taiwan national cohort followed more than 32,000 men and found a 74% increase in the incidence of ED in individuals with FMS [31]. Our findings confirm these macro-scale data by extrapolating them to the younger population, almost doubling the risk by articulating depression-high pain thresholds.

Although sleep disturbance has been addressed separately in the literature on FMS [31] and pED [31], it has rarely been reported as a clear indicator at the intersection of the two. In our study, the weight of the PSQI in predicting FMS combines the neuroimaging data of Harris *et al.* [35] demonstrating the link between insular glutamate elevation and sleep disruption in FMS patients and the predictions of Ayta and McKinlay positioning erectile dysfunction as an "early cardiometabolic stress marker" [12].

Waist circumference has been emphasized in the ED literature, mostly in the organic-vasculogenic group [27]; however, even in the pED cohort, it showed an increased risk of OR $\approx$ 2.4, suggesting that visceral obesity may have an indirect pathway through mental stress and sleep disturbance [35].

Finally, the −0.58 correlation between the pain index (WPI) and IIEF is a clinical response to Andersson and Wagner's classic animal experiments demonstrating endothelin-1 elevation in penile smooth muscle contractility [32] and Musicki's molecular studies describing the effect of oxidative stress on Soluble guanylyl cyclase (sGC) down-regulation [40].

4.3 Possible biological and psychosocial mechanisms

4.3.1 Central sensitization and autonomic balance

Central sensitization, which is at the core of FMS, is characterized by N-methyl-D-aspartate (NMDA) receptor hypersensitivity and Gamma-aminobutyric acid (GABA) deficiency in the dorsal horn and insular cortex [40, 41]. Increased glutamatergic tone both amplifies pain perception and triggers anxiogenic circuits in the limbic system [32]. Excess glutamate also shortens the delta sleep phase by stimulating hypothalamic wakefulness centers, suppresses the testosterone peak, and interrupts erection-preparatory parasympathetic activity [31].

4.3.2 Nitric oxide suppression and oxidative stress

Chronic inflammation-induced peroxynitrite accumulation both reduces nitric Gonadotropin-Releasing Hormone oxide synthesis and dampens sGC activity by "trapping" available NO [2]. Experimentally, suppression of the L-arginine/NO pathway mimics the WPI-like diffuse hyperalgesic response and produces relaxation failure in the corpus cavernosum [36, 38].

4.3.3 HHA axis hypercortisolemia

Prolonged stress response disrupts the serotonin-melatonin axis by increasing corticotropin-releasing hormone and smoothing nocturnal cortisol output, which lowers both sleep and pain threshold [31, 34]. In the same process, Gonadotropin-Releasing Hormone (GnRH) suppression down-regulates Sertoli-leydig cells; hence testosterone decline attenuates nitric oxide synthase expression [40].

4.3.4 Psychodynamic factors

Performance anxiety is the classic trigger of PED in young men [14]. However, when accompanied by FMS, the distortion of body-self perception reinforces the "painful body image"; sensory feedback becomes negative. This cognitive distortion decreases sexual arousability, deepening depression and cortical inhibition [31].

4.4 Effects on clinical practice

4.4.1 Adding FMS screening to the urological referral algorithm

When young men present to the urology outpatient clinic with "soft" complaints such as performance anxiety or partner incompatibility, the physician often settles for organic exclusionary tests such as penile duplex Doppler and hormonal panel [37]. Our findings show that performing a rapid WPI/SSS screening in individuals with IIEF $\leq$21 increases diagnostic yield to 95% AUC (Fig. 2). Thus, a practical algorithm could be structured as (i) IIEF rapid form $\rightarrow$ (ii) if the sum of WPI + SSS is $\geq$12, immediately administer the PSQI + BDI + BAI triad $\rightarrow$ (iii) if PSQI >5 or BDI/BAI is moderate to severe, call a rheumatology-psychiatry quadruple consultant table [22]. Such a tiered protocol both reduces unnecessary "penile vascular battery" examinations and captures the FMS-related systemic burden at an early stage [24].

4.4.2 Multidisciplinary treatment package

Even if the IIEF low is not of organic origin, the chronic extensive pain matrix suppresses the penile nitrous oxide systems alette [40]. Therefore, prescribing a phosphodiesterase 5 (PDE-5) inhibitor alone is akin to building a "floating bridge" over quicksand. The literature reports that 8–12 weeks of aerobic exercise improves endothelial function by 15%, with an average increase of 4 points on the IIEF [22]. A course of exercise also reduces pain-related fatigue by ~10 points on the rFIQ [20]. Along the same lines, the serotonin-noradrenaline reuptake inhibitors (SNRIs) duloxetine and milnasipran may target both FMS pain and comorbid depression, leading to an indirect increase in IIEF [41].

4.4.3 Sleep hygiene and hormone cycle

The mediator role of PSQI suggests that the erectile periphery cannot be repaired without disruption of sleep architecture. Nightly administration of melatonin 3 mg gave pilot results

in FMS improving both pain score and morning testosterone level [31]. Therefore, sleep hygiene education, evening blue light restriction, and low-dose melatonin supplementation as needed could be an "off-chain" intervention targeting the pED + FMS pair [30].

4.4.4 Waist circumference targets

Our analyses showed that a threshold of waist circumference ≥92 cm led to an independent $\beta = -0.17$ decrease. Each 5 cm reduction can improve the probability of FMS diagnosis by 12% and the IIEF score by an average of +1.1 points [27]. In dietary guidelines, it has been reported that the low-glycemic Mediterranean model can reduce FMS pain by ~2 VAS points and ED severity by ~3 IIEF points [41].

4.4.5 Psychotherapeutic strategies

Cognitive Behavioral Therapy (CBT) targets the pain-catastrophizing cycle in FMS and failure cognition in PED. Meta-analytic data report that 8 sessions of CBT resulted in a +3 point increase in IIEF and a −6 point decrease in BDI [41]. Including the partner in psycho-education modules increases treatment response by 20% through "relational congruence" [35].

4.5 Strengths and limitations of the study

4.5.1 Strengths

1. Differential Exclusion: Our rigorous exclusion of organic ED with duplex Doppler and hormonal panel allowed us to focus on pED.

2. Eight-Scale Measurement Suite: Optimized the "noise-to-signal" ratio by simultaneously scaling pain, sleep, mood, and sexual function [26, 31].

3. Statistical Coverage: We biaxially validated outcomes using both multiple linear and logistic regression; Adj R^2 = 0.69 and AUC = 0.84, indicating model robustness.

4. Age Matching: The age range of 20–40 years captured the naked influence of psychosocial factors by minimizing vascular-metabolic comorbidity [41].

4.5.2 Limitations

1. Cross-Sectional Design: We cannot establish the exact sequence of causality; longitudinal follow-up is necessary.

2. Single Center: Limited cultural and genetic diversity; replication in different ethnic groups [41]. The scales used in our study are in Turkish, and validation studies addressing cultural differences have not been conducted.

3. Sample Size: N = 47 total sample, reduced power in subgroup analyses (smoking, tertile breakdowns). The total sample size in our study was only 47.

4. Scale Self-Report: Social desirability bias may have inflated scores on the IIEF and BDI [6].

5. Lack of Biomarkers: objective parameters such as hs-CRP, IL-6, HRV were not included; models remained entirely based on psychometric variables. Our study used self-report measures of social desirability (such as sexual function and depression scores) and excluded objective indicators such as inflammatory markers (IL-6, CRP) and neuroimaging. Anonymous completion of clinical interviews or their combination with clinical interviews could be used to reduce bias.

5. Conclusions

1. FMS screening should be added to the PED algorithm. In men with IIEF ≤21, if the sum of WPI + SSS is ≥12, joint rheumatology-psychiatry-physical therapy management should be considered [22, 24].

2. Depression and sleep disturbance should be treated. If SNRI + CBT combination is integrated with melatonin-assisted sleep hygiene, bidirectional improvement in erectile response and pain scores is expected [32].

3. Aerobic exercise + waist circumference target (≤90 cm) should be considered as an "anti-inflammatory prescription" in the PED + FMS package [41].

4. Personalization of PDE-5 inhibitor use: In pain-driven PED, an "on-demand" dosing strategy should be preferred over daily tadalafil (5 mg) because chronic NO pressure makes optimal use of the overnight tadalafil wavelength [38].

Partner-inclusive psycho-education significantly increases relationship satisfaction and treatment compliance; urologist referral is essential [35].

The main knowledge gaps and proposed designs for future research are summarized in Table 9, outlining potential directions for longitudinal, mechanistic, and neuroimaging studies to clarify the biopsychosocial pathways linking fibromyalgia and psychogenic erectile dysfunction. The table emphasizes the need for multidimensional research strategies that combine neuroendocrine, inflammatory, psychological, and behavioral data to construct an integrative framework. Future investigations should adopt multimodal designs—such as time-series cohort studies with serial biomarker tracking, weight-loss interventions incorporating cytokine monitoring, and neuroimaging-based network analyses—to disentangle causal relationships and identify modifiable therapeutic targets.

Closing remark. This integrated model reveals that the chronic widespread pain → sleep deprivation → affective load → vasovagal dysfunction tetrad is far more prevalent among young men with psychogenic erectile dysfunction than previously recognized. The findings suggest that erectile dysfunction in this population cannot be fully explained by vascular or hormonal mechanisms alone but is rather the end-product of a dynamic interaction between somatic sensitization, circadian rhythm disruption, and emotional dysregulation. Therefore, clinical practice must evolve beyond the traditional "vessels-only" or "psychology-only" dichotomy toward a multi-node, single-map paradigm—one that integrates pain modulation, sleep restoration, mood stabilization, and sexual rehabilitation within a unified care model.

Integrating rheumatologic, psychiatric, and urologic expertise into a collaborative framework would not only enhance diagnostic precision but also facilitate personalized treatment protocols tailored to the patient's symptom constellation. Such an approach would redefine psychogenic erectile dysfunction as a neuropsycho-somatic disorder rather than a purely psychogenic condition. Ultimately, addressing the shared mechanisms of chronic pain and sexual dysfunction may improve both physical and psychosocial outcomes, paving the way for

TABLE 9. Future research directions.

No.	Research Gap	Proposed Design	Target Outcomes	Potential Impact
1	Causal sequence (pain → depression → ED?)	24-month prospective cohort; scales + IL-6/HRV every 3 months	Time-series structural equation model	Identifies an early "pain cutoff", opens a window for preventive psychotherapy
2	Role of visceral adipokines	Randomized weight-loss trial (diet + HIIT)	TNF-α change IIEF increase	Mechanistic validation of the metabolic–sexual axis linkage
3	Melatonin pain–erection triad	Double-blind, placebo-controlled melatonin 3 mg/8 weeks	PSQI, WPI, serum testosterone, IIEF	Demonstrates the clinical benefit of a circadian intervention
4	Young female–male comparison	Multicenter cross-sectional; n $\geq$300 (150 Women/50 Men)	Sex-specific structural models	Adds a sex-related dimension to treatment guidelines
5	Neuroimaging correlates	fMRI + MRS: insular GABA-glutamate	Mediating pathways for BDI/PSQI	Establishes brain–pain–erection maps as clinical biomarkers

ED: Erectile Dysfunction; IL-6: Interleukin-6; HRV: Heart rate variability; HIIT: High-intensity interval training; fMRI: Functional magnetic resonance imaging; MRS: Magnetic resonance spectroscopy; GABA: Gamma-aminobutyric acid; TNF-α: Tumor necrosis factor alpha; PSQI: The pittsburg sleep quality index; WPI: Widespread Pain Index; IIEF: International Index of Erectile Function; BDI: Beck Depression Inventory.

preventive strategies that target early markers of the pain–depression–erection cycle (Table 9).

AVAILABILITY OF DATA AND MATERIALS

The data, code, and study material that support the findings of this study are available from the corresponding author on reasonable request.

AUTHOR CONTRIBUTIONS

SK—conceptualization, writing-original draft, data curation. IFK—conceptualization, supervision, visualisation. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethics committee of Acıbadem University Faculty of Medicine, the ethical approval number for the study is 2022-14/06. Informed consent was obtained from all individual participants included in the study.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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