

ORIGINAL RESEARCH

Association between sleep quality, depression, and sexual dysfunction in men seeking treatment for sexual dysfunction: a multicenter prospective analysis

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Abstract

Background: Sleep quality, depression, and sexual dysfunction are interrelated yet under-investigated areas, particularly in clinical urology practice. Given the high prevalence of these conditions and their impact on men's health, our study aimed to explore the associations between these factors in men seeking treatment for sexual dysfunction. **Methods:** This prospective multicenter study included 121 men aged 18–65 years with sexual dysfunction, recruited from eight urology clinics. Patients were assessed using the Pittsburgh Sleep Quality Index (PSQI), the International Index of Erectile Function (IIEF), the Premature Ejaculation Diagnostic Tool (PEDT), and the Beck Depression Inventory (BDI). Hormonal parameters were also evaluated. Participants were classified according to sleep quality and depression severity. Comparative and logistic regression analyses were performed. **Results:** Poor sleep quality was found in 72.7% of patients. These individuals had lower sexual desire and overall satisfaction scores, and higher PEDT scores ($p < 0.05$). Moderate to severe erectile dysfunction and premature ejaculation were more prevalent in this group. Depression severity was also significantly higher among those with poor sleep ($p = 0.03$). Logistic regression analysis showed independent associations between poor sleep quality, older age, erectile dysfunction severity, lower sexual desire, and the presence of premature ejaculation. **Conclusions:** Poor sleep quality is associated with increased sexual dysfunction and depressive symptoms. Evaluation of sleep and mood should be integrated into the clinical assessment of male patients presenting with sexual complaints.

Keywords

Sleep wake disorders; Erectile dysfunction; Premature ejaculation; Depression; Sexual dysfunction

1. Introduction

Male sexual dysfunction encompasses a range of conditions, including erectile dysfunction, ejaculatory disorders, and disorders of libido. These conditions can significantly impair quality of life and are typically attributed to a variety of vascular, neurological, hormonal, and psychological etiologies. Common organic causes include diabetes mellitus, cardiovascular diseases, hypogonadism, and coexisting urological conditions such as chronic prostatitis and lower urinary tract symptoms [1, 2]. The treatment of male sexual dysfunction involves a multifaceted approach tailored to the underlying etiology and may include phytotherapeutic, behavioral, medical, psychosocial, and surgical interventions. A comprehensive evaluation of all potential contributing factors is essential to accurately identify the cause and implement the most appro-

priate therapeutic strategy [3, 4].

The relationship between sleep and sexual function has become an increasingly prominent area of interest in the medical literature in recent years. Disruptions in sleep patterns and quality can impact numerous physiological and psychological processes and have been associated with common sexual dysfunctions in men [5, 6].

Various mechanisms have been proposed to explain how sleep disorders may negatively affect sexual function. Disruptions in the circadian rhythm and hormonal balance—particularly alterations in testosterone and prolactin levels—can contribute to sexual dysfunction [6–8]. Additionally, irregular sleep patterns and chronic insomnia are frequently accompanied by psychological conditions such as stress, anxiety, and depression, which may further impair sexual function [9, 10]. Moreover, the adverse effects of sleep

disturbances on the cardiovascular system can exacerbate erectile dysfunction through vascular mechanisms [11].

In this study, we aimed to comprehensively investigate the effects of sleep disorders and depression on male sexual function using a multicenter prospective design.

2. Materials and methods

This prospective study was conducted following the approval of the Izmir City Hospital Ethics Committee on 10 July 2024 (Approval No: 2024/81). The study was carried out in urology outpatient clinics across eight different medical centers. A total of 121 male patients aged 18–65 years who presented to the urology outpatient clinics with any form of sexual dysfunction between 01 August 2024 and 01 February 2025 were included in the study.

Exclusion criteria were as follows: individuals younger than 18 or older than 65 years, patients with substance or drug dependency, uncontrolled diabetes mellitus (Glycated Hemoglobin A1c (HbA1c) >6.5), peripheral vascular disease, New York Heart Association class III or higher heart failure, chronic obstructive pulmonary disease or interstitial lung disease, uncontrolled hypertension, severe arrhythmia, a history of myocardial infarction or cerebrovascular event within the last 6 months, narcolepsy, use of anabolic steroids, active malignancy, or a history of orchiectomy. Patients with a known diagnosis of major psychiatric disorders (e.g., generalized anxiety disorder, bipolar disorder, schizophrenia), patients currently receiving psychotropic medication (e.g., antidepressants, anxiolytics, antipsychotics), and those with secondary sexual dysfunction or sleep disturbance due to chronic medical or psychiatric illness were also excluded. Additionally, patients who had previously received treatment for sexual dysfunction were not included, as all evaluations were performed at the initial presentation prior to any therapeutic intervention.

Demographic data, hormonal parameters, including follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, and total testosterone, and sexual functions were evaluated. Sexual functions were assessed using the validated versions of the International Index of Erectile Function (IIEF) and the Premature Ejaculation Diagnostic Tool (PEDT). Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). Participants with a PSQI score <5 were classified as having good sleep quality (Group 1), whereas those with a score ≥ 5 were classified as having poor sleep quality (Group 2). Sexual function parameters were compared between the two groups.

In addition, the Beck Depression Inventory was used to assess participants' depressive symptoms, with scores categorized as follows: 0–9 (minimal), 10–16 (mild), 17–63 (moderate-to-severe depression). Based on these thresholds, patients were grouped into three categories: minimal depression (Group A), mild depression (Group B), and moderate-to-severe depression (Group C). Sexual function parameters were compared among these groups, and *post hoc* analyses were performed to examine the relationship between depression severity and sexual dysfunction.

Statistical analyses were performed using the SPSS (Sta-

tistical Package for the Social Sciences) version 23 software (IBM SPSS Statistics for Windows, (IBM Corp., Armonk, NY, USA)). Parametric descriptive data were expressed as mean \pm standard deviation while the normality of data distribution was assessed using the one-sample Kolmogorov-Smirnov test. For normally distributed continuous variables, comparisons were made using one-way Analysis of Variance (ANOVA) and Student's *t*-test. Tukey's Honestly Significant Difference (HSD) test was applied to identify the group(s) responsible for significant differences. The Chi-square test was used to compare categorical variables. A *p*-value of < 0.05 was considered statistically significant with a 95% confidence interval. Logistic regression analysis was performed to identify independent predictors of sexual dysfunction, with odds ratios (ORs) and 95% confidence intervals (CIs) reported.

3. Results

Among the 121 patients included in the study, sleep disturbances were observed in 88 individuals. An increase in age was found to be significantly associated with the presence of sleep disorders ($p = 0.037$). When hormonal parameters were examined, no significant differences were found between patients with and without sleep disturbances.

Although there was no statistically significant difference in IIEF erectile function scores between the two groups ($p = 0.128$), when patients were categorized according to the severity of erectile dysfunction (none to mild vs. moderate to severe), the prevalence of sleep disorders was significantly higher in the moderate to severe erectile dysfunction group ($p = 0.023$). The IIEF sexual desire score was significantly lower in the group with sleep disturbances ($p = 0.006$), as was the IIEF overall satisfaction score ($p = 0.03$).

The PEDT scores were significantly higher among patients with sleep disturbances ($p = 0.03$). When patients were grouped by PEDT scores into those without premature ejaculation (<9) and those with premature ejaculation (≥ 9), the presence of sleep disorders was significantly higher among those with premature ejaculation ($p = 0.034$).

Logistic regression analysis showed that increasing age was positively correlated with the presence of sleep disturbances ($p = 0.04$, $r = 0.037$, OR: 1.038; 95% CI: 1.002–1.075). When patients were categorized by erectile dysfunction severity based on IIEF erectile function scores, a positive correlation was observed between sleep disturbances and erectile dysfunction severity ($p = 0.025$, $r = 0.999$, OR: 0.368; 95% CI: 0.153–0.885). The IIEF sexual desire score ($p = 0.008$, $r = -0.237$, OR: 0.789; 95% CI: 0.662–0.940) and IIEF overall satisfaction score ($p = 0.033$, $r = -0.209$, OR: 0.811; 95% CI: 0.670–0.983) were both negatively correlated with sleep disturbances. PEDT scores also showed a positive correlation with sleep disturbances ($p = 0.036$, $r = 0.091$, OR: 1.095; 95% CI: 1.006–1.191). Thus, the presence of premature ejaculation was found to be significantly and positively correlated with sleep disturbances ($p = 0.037$, $r = 0.927$, OR: 2.528; 95% CI: 1.06–6.03). The relationship between sleep quality and sexual function is presented in Table 1.

When patients were grouped according to the Beck Depression Inventory (BDI) scores, the distribution among those with

TABLE 1. Comparison of demographic, hormonal, sexual function, and ejaculatory parameters between patients with and without sleep disorders.

| | Group 1 (n = 33) | Group 2 (n = 88) | p Value | p [‡] |
|--|---------------------|---------------------|--------------------|---|
| Age (yr) | 41.50 ± 12.76 | 46.87 ± 11.75 | 0.037* | 0.04 OR: 1.038 95% CI: 1.002–1.075 r = 0.037 |
| Total PSQI Score | 2.96 ± 0.84 | 8.03 ± 2.08 | <0.001* | |
| FSH (IU/L) | 5.31 ± 2.94 | 5.95 ± 3.74 | 0.410* | |
| LH (IU/L) | 4.07 ± 2.07 | 5.32 ± 3.22 | 0.060* | |
| Prolactin (µg/L) | 8.20 ± 3.98 | 10.86 ± 10.47 | 0.200* | |
| Total Testosterone (ng/dL) | 399.59 ± 170.08 | 387.95 ± 163.11 | 0.740* | |
| IIEF Erectile Function Score | 17.54 ± 7.40 | 15.31 ± 7.01 | 0.128* | |
| IIEF Erectile Dysfunction Group ^α | | | | |
| Moderate to Severe | 20 (60.60%) | 71 (80.70%) | 0.023 [#] | 0.025 OR: 0.368 95% CI: 0.153–0.885 r = 0.999 |
| Mild to None | 13 (39.40%) | 17 (17.30%) | | |
| IIEF Orgasmic Function Score | 7.36 ± 3.19 | 6.27 ± 3.12 | 0.092* | |
| IIEF Sexual Desire Score | 7.45 ± 2.41 | 6.00 ± 2.60 | 0.006* | 0.008 OR: 0.789 95% CI: 0.660–0.940 r = -0.237 |
| IIEF Intercourse Satisfaction Score | 7.36 ± 4.43 | 6.68 ± 3.31 | 0.362* | |
| IIEF Overall Satisfaction Score | 6.15 ± 2.15 | 5.15 ± 2.22 | 0.030* | 0.033 OR: 0.811 95% CI: 0.670–0.983 r = -0.209 |
| PEDT Score | 10.39 ± 4.89 | 12.57 ± 4.97 | 0.030* | 0.036 OR: 1.095 95% CI: 1.006–1.191 r = 0.091 |
| Premature Ejaculation ^β | | | | |
| Absent | 13 (39.40%) | 19 (21.60%) | 0.034 [#] | 0.037 OR: 2.528 95% CI: 1.060–6.030 r = 0.927 |
| Present | 20 (60.60%) | 69 (78.40%) | | |

*T-test; [‡]Binary Logistic Regression; [#]Chi-square test.

^αErectile dysfunction (ED) severity based on the International Index of Erectile Function (IIEF-5): 0–10 = Severe ED; 11–16 = Moderate ED; 17–21 = Mild to moderate ED; 22–25 = Mild ED.

^βPremature ejaculation (PE) classification based on the Premature Ejaculation Diagnostic Tool: <9 = No PE; ≥9 = PE present. PSQI: Pittsburgh Sleep Quality Index; PEDT: Premature Ejaculation Diagnostic Tool; OR: odds ratios; CI: confidence intervals; FSH: follicle-stimulating hormone; LH: luteinizing hormone.

sleep disturbances (n = 88) was as follows: 27 (30.7%) had minimal, 31 (35.2%) had mild, and 30 (34.1%) had moderate to severe depression. Among those without sleep disturbances,

17 (51.5%) had minimal, 12 (36.4%) had mild, and only 4 (12.1%) had moderate to severe depression. Thus, more severe depression was significantly more common among patients

with sleep disturbances ($p = 0.03$).

No significant differences were found between depression severity groups in terms of age or hormonal parameters. Although prolactin levels tended to increase with depression severity, this difference was not statistically significant ($p = 0.161$).

All IIEF subdomains—erectile function, sexual desire, sexual satisfaction, orgasmic function, and overall satisfaction—differed significantly among the three depression severity groups. *Post hoc* analysis showed that the IIEF orgasmic function score was significantly lower in Group C (moderate to severe depression) compared to Group A (minimal depression) ($p < 0.001$, $r = -0.311$, OR: 0.733; 95% CI: 0.620–0.866) and Group B (mild depression) ($p = 0.012$, $r = -0.198$, OR: 0.821; 95% CI: 0.703–0.958). No significant difference was observed between Groups A and B ($p = 0.199$).

Regarding the IIEF sexual desire score, a negative correlation was found between increasing depression severity and sexual desire, with the difference driven by comparisons between Groups A and C ($p = 0.008$, $r = -0.237$, OR: 0.789; 95% CI: 0.662–0.941). No significant differences were found between Group B and Groups A or C ($p = 0.182$ and $p = 0.402$, respectively).

The IIEF sexual satisfaction score also decreased with increasing depression severity, and this difference was significant between Groups A and C ($p < 0.001$, $r = -0.261$, OR: 0.771; 95% CI: 0.662–0.897). Group B did not significantly differ from either Group A or C ($p = 0.053$ and $p = 0.152$, respectively).

Similarly, IIEF overall satisfaction scores declined with increasing depression severity, with a significant difference observed between Groups A and C ($p = 0.001$, $r = -0.368$, OR: 0.692; 95% CI: 0.553–0.866). Group B did not significantly differ from Group A or C ($p = 0.102$ and $p = 0.195$, respectively).

A significant inverse correlation was observed between IIEF erectile function scores and depression severity ($p < 0.001$). When erectile dysfunction severity was compared between groups categorized as mild to none and moderate to severe, a significant difference in erectile function scores was found among the depression groups ($p = 0.043$). This difference was mainly between Groups A and C, indicating a negative correlation between increasing depression severity and erectile function ($p < 0.001$, $r = -0.126$, OR: 0.881; 95% CI: 0.820–0.948). No significant differences were observed between Group B and Groups A or C ($p = 0.098$ and $p = 0.117$, respectively).

When the groups were compared in terms of premature ejaculation, significant differences were found in PEDT scores across the three groups ($p = 0.002$). Similarly, when patients were grouped as no, probable, and definite premature ejaculation, significant differences were again observed ($p = 0.001$). PEDT scores were significantly higher in both Groups B ($p = 0.011$, $r = 0.128$, OR: 1.137; 95% CI: 1.030–1.255) and C ($p = 0.002$, $r = 0.157$, OR: 1.169; 95% CI: 1.057–1.294) compared to Group A, suggesting that patients with premature ejaculation also had more severe depression. No significant difference was found between Groups B and C ($p = 0.560$). The relationship between depression severity and sexual function is shown in

Table 2.

And finally in our study, the severity of depression was found to be significantly higher among patients with sleep disturbances. Among patients without sleep disorders, 51.5% had minimal depression, 36.4% had mild depression, and only 12.1% had moderate to severe depression. In contrast, among those with sleep disturbances, 30.7% had minimal depression, 35.2% had mild depression, and 34.1% had moderate to severe depression. This distribution indicates a significant association between poor sleep quality and the presence of more severe forms of depression ($p = 0.03$) (Table 3).

4. Discussion

This study demonstrated that the presence of sleep disturbances in men is significantly associated with sexual dysfunctions, including erectile dysfunction (ED) and premature ejaculation (PE). Notably, advancing age was correlated with worsening sleep quality, which in turn appeared to exacerbate the severity of age-related sexual dysfunctions [12–15]. These findings are consistent with previous literature and support the substantial impact of sleep quality on sexual function.

The effects of sleep disturbances on sexual function have been explained in the literature through hormonal, psychological, and vascular mechanisms [16–18]. Theoretically sleep disturbances, particularly short sleep duration and insomnia, are increasingly recognized as significant risk factors for the development of ED. This relationship can be explained through multiple physiological and molecular mechanisms. Primarily, the secretion of key reproductive hormones, especially testosterone, is closely regulated by sleep. Reduced or fragmented sleep disrupts the physiological nocturnal rise in testosterone levels, leading to diminished libido and impaired erectile function. Moreover, sleep disturbances alter the regulatory secretion of other gonadotropic hormones such as prolactin, FSH, and LH, further contributing to sexual dysfunction [17, 19].

However, similar to some previous studies, our findings did not show significant differences in key hormonal parameters (FSH, LH, prolactin, and testosterone) between patients with and without sleep disturbances [20–22]. This suggests that the link between sleep disturbances and sexual dysfunction may not always be mediated by alterations in reproductive hormone levels. Consequently, further large-scale, long-term prospective studies are required to clarify the underlying hormonal mechanisms.

Sleep deprivation also activates the hypothalamic-pituitary-adrenal axis, resulting in elevated cortisol levels. Sustained cortisol elevation promotes vascular smooth muscle contraction and impairs penile vasodilation, thereby contributing to ED. Additionally, autonomic nervous system imbalance—characterized by heightened sympathetic activity and reduced parasympathetic tone—is commonly observed in sleep-deprived individuals and adversely affects the neurovascular mechanisms essential for penile erection [23–25].

Endothelial dysfunction is another critical pathway linking poor sleep and ED. Sleep loss reduces nitric oxide bioavailability and impairs endothelial-dependent vasodilation, thereby compromising penile blood flow. This is exacerbated by

TABLE 2. Comparison of demographic, hormonal, and sexual function parameters across depression severity groups.

| | Group A (n = 44) | Group B (n = 43) | Group C (n = 34) | p Value |
|--|---------------------|---------------------|---------------------|--------------------|
| Age (yr) | 44.72 ± 12.68 | 46.00 ± 12.49 | 45.87 ± 11.44 | 0.874* |
| FSH (IU/L) | 6.17 ± 3.29 | 5.71 ± 2.90 | 5.37 ± 4.55 | 0.652* |
| LH (IU/L) | 5.36 ± 2.52 | 4.47 ± 2.78 | 5.20 ± 3.80 | 0.377* |
| Prolactin (µg/L) | 8.25 ± 4.20 | 10.48 ± 8.18 | 12.48 ± 10.52 | 0.161* |
| Total Testosterone (ng/dL) | 353.86 ± 113.68 | 391.07 ± 153.79 | 439.35 ± 217.22 | 0.084* |
| IIEF Erectile Function Score | 18.72 ± 6.88 | 15.69 ± 6.65 | 12.58 ± 6.81 | <0.001* |
| IIEF Erectile Dysfunction Group ^α | | | | |
| Moderate-Severe | 28 (63.6%) | 33 (76.7%) | 30 (88.2%) | 0.043 [#] |
| Mild-None | 16 (36.4%) | 10 (23.3%) | 4 (11.8%) | |
| IIEF Orgasmic Function Score | 7.79 ± 2.78 | 6.69 ± 2.93 | 4.82 ± 3.21 | <0.001* |
| IIEF Sexual Desire Score | 7.22 ± 2.64 | 6.25 ± 2.31 | 5.50 ± 2.69 | 0.013* |
| IIEF Intercourse Satisfaction Score | 8.38 ± 3.55 | 6.65 ± 3.22 | 5.17 ± 3.55 | <0.001* |
| IIEF Overall Satisfaction Score | 6.27 ± 2.23 | 5.32 ± 2.07 | 4.47 ± 2.09 | <0.001* |
| PEDT Score | 9.97 ± 4.41 | 12.62 ± 4.69 | 13.76 ± 5.42 | 0.002* |
| Premature Ejaculation ^β | | | | |
| Absent | 17 (38.6%) | 8 (18.6%) | 6 (17.6%) | 0.001 [#] |
| Probable | 11 (25%) | 6 (14%) | 1 (2.9%) | |
| Present | 16 (36.4%) | 29 (67.4%) | 27 (79.4%) | |

*One Way ANOVA; [#]Chi-square test.

^αErectile dysfunction (ED) severity based on the International Index of Erectile Function (IIEF-5): 0–10 = Severe ED; 11–16 = Moderate ED; 17–21 = Mild to moderate ED; 22–25 = Mild ED.

^βPremature ejaculation (PE) classification based on the Premature Ejaculation Diagnostic Tool (PEDT): <9 = No PE; 9–10 = Probable PE; >10 = PE present.

PEDT: Premature Ejaculation Diagnostic Tool; FSH: follicle-stimulating hormone; LH: luteinizing hormone.

TABLE 3. Distribution of depression severity among patients with and without sleep disorders.

| | Patients with Minimal Depression | Patients with Mild Depression | Patients with Moderate to Severe Depression | p Value |
|---|-------------------------------------|----------------------------------|--|---------|
| Patients without Sleep Disorder (n = 33) | 17 (51.5%) | 12 (36.4%) | 4 (12.1%) | 0.03 |
| Patients with Sleep Disorder (n = 88) | 27 (30.7%) | 31 (35.2%) | 30 (34.1%) | |

increased levels of vasoconstrictors such as endothelin-1 and angiotensin II. Furthermore, poor sleep induces a systemic pro-inflammatory state, characterized by elevated cytokines such as interleukin-6, tumor necrosis factor-α, and C-reactive protein, which are known to impair vascular integrity and erectile function [26, 27].

These theoretical and mechanistic insights are supported by our findings. When patients were stratified by ED severity, the frequency of sleep disturbances was found to be higher in the moderate-to-severe ED group. Logistic regression analysis also revealed a strong positive correlation between ED severity and sleep disturbance. This finding is consistent with earlier studies suggesting that sleep disorders may serve as an inde-

pendent risk factor for the development or worsening of ED [28, 29].

Additionally, patients with sleep disturbances were found to have significantly lower scores for sexual desire and overall satisfaction. These results align with previous studies indicating that chronic sleep problems can reduce sexual satisfaction and libido [30, 31]. Similarly, the higher prevalence of sleep disturbances in patients with PE supports earlier findings that suggest a significant relationship between sleep quality and ejaculatory function [32, 33].

Psychological factors such as depression and anxiety play a critical role in the development and persistence of erectile dysfunction. Depressed individuals often experience low

energy, anhedonia, and diminished motivation, which can impair sexual desire and performance. Biologically, depression is linked to dysregulation of the hypothalamic-pituitary-adrenal axis, resulting in excessive catecholamine release and reduced serotonin activity—mechanisms that may impair cavernosal smooth muscle relaxation and contribute to sexual dysfunction. Anxiety, on the other hand, interferes with sexual arousal by diverting attention from erotic stimuli and increasing performance-related stress. Anxiety-related ED can also initiate a vicious cycle of reduced self-confidence, partner dissatisfaction, and relational conflict. Low self-esteem, which frequently coexists with both anxiety and depression, has likewise been shown to negatively influence sexual performance and satisfaction. Together, these interrelated psychosocial mechanisms highlight the complex relationship between mental health and erectile function [34, 35].

According to the Beck Depression Inventory (BDI) scores, increased depression severity was associated with significant declines in erectile function, sexual desire, orgasmic function and overall satisfaction. The close relationship between depression and sexual dysfunction has been widely documented in the literature [33]. Given that depression is often comorbid with sleep disorders and may further impair sexual function, our findings suggest that sleep disturbances may negatively impact not only sexual function but also mental health. It is critical that depression is thoroughly assessed during the clinical evaluation of these patients.

The relationship between prolactin and depression has been a frequently discussed topic in the literature. Elevated prolactin levels have been suggested to be associated with depressive symptoms [36, 37]. Although our study showed a trend toward increased prolactin levels with higher depression severity, the difference did not reach statistical significance. Considering the potential role of prolactin in the central nervous system and mood regulation, large-scale longitudinal studies are warranted to further elucidate the relationship between prolactin levels and depression.

One of the strengths of this study is its multicenter prospective design. However, the relatively small sample size is a limitation, and our findings should be validated by larger prospective studies. Additionally, the use of objective sleep assessment methods such as polysomnography may enhance the reliability of our results more than the subjective sleep quality measurements used in this study. Although we applied strict exclusion criteria—excluding patients with known psychiatric disorders, psychotropic medication use, and major systemic diseases—to reduce potential confounding, undiagnosed psychological conditions, variations in disease awareness, and psychosocial factors such as marital status or family support may still have influenced the outcomes. These variables were not specifically analyzed in the present study, yet prior research indicates that insufficient partner support and low disease-related awareness can contribute to increased psychological distress and diminished sexual health [38, 39]. The integration of these psychosocial dimensions into future research designs could offer a more comprehensive understanding of the complex interactions affecting men with sexual dysfunction.

Furthermore, the study primarily focused on depressive symptoms measured by the Beck Depression Inventory;

however, anxiety and other mood-related disorders, which are also known to affect sleep and sexual function, were not independently assessed. This remains a relevant limitation. Future studies with larger samples, longitudinal follow-up, and broader psychiatric assessments are warranted to clarify these complex interrelationships.

In conclusion, our findings indicate that sleep disturbances in men are significantly associated with ED and PE. Sleep quality and depression should be considered key factors during the evaluation of patients with sexual dysfunction. Based on these findings, incorporating sleep and depression screening tools into routine urological assessments may be an important step toward improving patient management and quality of life.

5. Conclusions

This multicenter prospective study demonstrated that sleep disturbances in men are significantly associated with sexual dysfunctions such as erectile dysfunction and premature ejaculation. Patients with poor sleep quality exhibited greater impairment in sexual function and increased depression severity. Depression itself may serve as an independent cause of sexual dysfunction. Therefore, evaluating sleep quality and depressive symptoms during urological assessments is crucial for a comprehensive patient evaluation and the development of effective treatment strategies.

AVAILABILITY OF DATA AND MATERIALS

The data supporting the findings of this study are available from the corresponding author upon reasonable request. Due to patient confidentiality, the raw data are not publicly accessible.

AUTHOR CONTRIBUTIONS

AE—conceptualization, study design, project administration, writing-original draft. MDT, TB, OA, CS—Data Collection, Patient Recruitment. MSD, AA, AY—Statistical Analysis, Interpretation of Data. IA, ECA, VS, YC, BI—Critical Revision, Manuscript Editing. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Ethics Committee of İzmir City Hospital (Approval Number: 2024/81, Date: 10 July 2024). Written informed consent was obtained from all participants before inclusion in the study. All procedures were conducted in accordance with the Declaration of Helsinki.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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