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The Importance of Sleep and Sleep Disorders in Multiple Sclerosis

Multipl Sklerozda Uykunun ve Uyku Bozukluklarının Önemi

İnan Özdemir, Semai Bek, Gülnihal Kutlu

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Abstract

Multiple sclerosis (MS) and sleep disorders form a vicious cycle when assessing quality of life and disability. Sleep disorders can exacerbate the symptoms and signs of MS, while conversely, symptoms and signs of MS can worsen sleep disorders. Therefore, sleep disorders should be addressed, identified, and treated in the medical practice related to MS. Sleep disorders are summarized under essential headings, and their relationship with MS has been demonstrated based on current literature data.

Keywords: Multiple sclerosis, sleep, sleep disorders

Öz

Multipl skleroz (MS) ve uyku bozuklukları yaşam kalitesi ve engelliliğin değerlendirilmesinde bir kısır döngü oluştururlar. Uyku bozuklukları, MS'ye ait belirti ve bulguların artmasına neden olurken diğer yandan MS'nin kendi semptomları ve kullanılan ilaçlar uyku bozukluklarını arttırmaktadır. Dolayısıyla MS pratiğinde uyku bozuklukları sorgulanmalı, tanınmalı ve tedavi edilmelidir. Bu yazıda temel olarak uyku bozuklukları başlıklar halinde özetlenmiş ve MS ile ilişkisi güncel literatür verisi eşliğinde ortaya konmuştur.

Anahtar Kelimeler: Multipl skleroz, uyku, uyku bozuklukları

Introduction

Sleep, once regarded a passive process of life until the mid-20th century, has undergone a conceptual transformation over the years with the advancement of electrophysiological studies. Healthy sleep is the physiological state that arises from a combination of voluntary decisions and involuntary biological activities. If you pay attention, you can actually see what a sleep disorder is in its definition. If biological activity puts you to sleep although you do not want to, the condition is considered a pathology. Similarly, if you cannot sleep although you want to, this is also classified as a pathology. The dynamic relationship between demand and biological activity must be balanced. Based on this interplay, healthy adult individuals are advised to aim for 7-9 hours of sleep per night. The body sleeps in the darkness of the night, integrated with nature, and awakens in the light of the day.

While the circadian rhythm triggers wakefulness, the homeostatic rhythm induces sleep. As long as this balance continues and remains rhythmically stable, we can consider it a healthy sleep. Although the anatomy of sleep physiology is

not our main topic, many anatomical regions are active in the sleep-wake cycle. While brainstem histaminergic, serotonergic, noradrenergic, cholinergic, and dopaminergic neurons, along with hypothalamic orexinergic neurons, collaborate to enhance wakefulness, the hypothalamic preoptic area is responsible for inducing sleep. This cycle is controlled by the suprachiasmatic nucleus, which regulates the circadian clock.

Although sleep disorders are prevalent among individuals diagnosed with multiple sclerosis (MS), they are unfortunately much less frequently diagnosed than expected. This is because they are not routinely inquired about in clinical practice and are often not reported by the patients themselves. Of course, systemic disease itself has a deleterious impact on the standard of living. Even if patients are unaware, investigating sleep quality and habits is crucial when obtaining a detailed medical history. Although the prevalence of sleep disorders in healthy individuals is found to be 42-65% in different publications, it is reported that this rate increases approximately four times in patients with MS, despite methodological differences in studies.¹ Sleep disorders cause disability on their own and raise

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questions about their contribution to or coexistence with MS. As a result, both medical conditions are diseases that require treatment, and their coexistence puts the patient in a more difficult situation.

The pathophysiology of MS-related sleep disorders is multifactorial. MS, which is based on autoimmune pathology, and sleep, whose immunology is not fully understood, create a dilemma. Interleukin-1 (IL1), IL2, IL6, IL8, IL15, IL18, epidermal growth factor, tumor necrosis factor-alpha, acidic fibroblast growth factor, colony-stimulating factor, and interferons trigger sleep. IL1 receptor antagonist IL1-RA, IL4, IL10, IL13, insulin-like growth factor, and soluble IL1 inhibit sleep.² In MS, overproduction of proinflammatory cytokines (IL-1, IL-6, tumor necrosis factor-alpha (TNF- α)) contributes to both neuronal damage and sleep architecture disruptions. These cytokines particularly affect non-rapid eye movement (NREM) sleep, leading to decreased sleep duration and quality.² Although IL-1 and TNF- α have been shown to increase NREM sleep, these effects may disrupt homeostatic sleep regulation in chronic inflammation.² Another factor involved in the pathophysiology is disruptions in melatonin secretion and circadian rhythms. Disturbances in melatonin secretion are common in individuals with MS. The melatonin rhythm is disrupted by involvement of the suprachiasmatic nucleus and pineal gland.^{1,2} One study identified genetic polymorphisms in melatonin metabolism, particularly in the progressive MS subtype, and the resulting decrease in melatonin levels disrupts the sleep-wake cycle.³ MS lesions occurring in areas involved in sleep and respiratory regulation, such as the brainstem, hypothalamus, and cervical cord, play a role in the pathophysiology of disorders, such as obstructive sleep apnea (OSAS) and restless legs syndrome (RLS).^{2,4} Medications used to treat MS, such as beta-interferons, antidepressants, and muscle relaxants, can impair sleep quality. These medications are associated with insomnia, daytime sleepiness, or parasomnias.⁴ The onset of sleep disorders approximately five years before the clinical diagnosis of MS, and their significant exacerbation by the time of diagnosis, suggest that MS and sleep disorders may be linked in a cause-and-effect relationship rather than simply co-occurring.⁵

The International Classification of Sleep Disorders published by the American Academy of Sleep Medicine is main source for setting standards in sleep medicine. Within this classification framework, sleep disorders will be categorized into main headings, such as insomnia, sleep-related breathing disorders, centrally caused hypersomnolence, circadian rhythm sleep-wakefulness disorders, parasomnia, and sleep-related movement disorders. The evaluation will focus on their relationship with MS.⁶

Insomnia: Under the title of insomnia, there are chronic insomnia, short-term insomnia, isolated symptoms, other insomnia disorders, and normal variants (long-time in bed and short sleepers). Researchers have conducted extensive research on the coexistence of MS and chronic insomnia in patients with MS. Insomnia is defined as persistent difficulty in initiating, maintaining, intensifying, and maintaining the quality of sleep, resulting in impairment in daily activities, despite

having adequate opportunities and conditions for sleep. In general terms, insomnia refers to the inability to sleep despite having the opportunity to obtain sufficient and high-quality sleep, leading to daytime dysfunction. Although its prevalence is 10% in the healthy population, it is observed six times more frequently in patients with MS.⁷

Depression, nocturia, pain, and spasticity are the most significant causes of insomnia in MS.⁸ While depression is a more prevalent cause in women, pain, resulting from the effects on sensory pathways, is the most common cause in men. Nocturia caused by brainstem and particularly pontine lesions more prominently leads to insomnia.⁹

The first-line treatment for insomnia is cognitive behavioral therapy. It provides more lasting results than medication, has no side effects, and does not increase the medication burden in patients with MS. It includes sleep hygiene, stimulus control, and relaxation techniques. It also improves fatigue symptoms in MS.¹⁻⁴ The basic principle in treating it is to ensure excellent sleep hygiene. Under the concept of sleep hygiene, the patient should adhere to a consistent bedtime and wake-up time. If the patient takes a nap during the day, it should not exceed 1 hour and should be completed by 1:00 PM at the latest. The patient should avoid consuming food and drinks after dinner and refrain from napping in bed, including activities, such as watching television or using the phone. It is advisable to avoid using sleep-inducing devices with light, to only go to bed for sleeping purposes, and not to engage in activities, such as reading books. To obtain restorative slumber, it is essential to reside in an environment characterized by minimal illumination and sound, accompanied by the judicious selection of bedding and pillows. 5-Hydroxytryptamine 2 ligands, such as risperidone, olanzapine, mirtazapine, and mianserin can be used in medical treatment. It is important to recognize that these pharmacological agents may trigger or exacerbate symptoms related to RLS. Melatonin can be administered in doses up to 12 mg per day. There are study results reporting that a series of treatments, in the form of daily moderate-weight resistance exercises and transcranial direct electrical stimulation, facilitates the process of achieving sleep.¹⁰⁻¹²

Sleep-related Breathing Disorders: Obstructive sleep apnea syndrome, central sleep apnea syndrome (CSAS), sleep-related hypoventilation disorder [$\text{PaCO}_2 > 55$ mmHg and > 10 min. (minimum)], sleep-related hypoxemia disorder ($\text{SpO}_2 < 88\%$ and < 5 min.), as well as isolated symptoms and normal variants, such as snoring and catathrenia. Studies on sleep disorders and MS have primarily focused on apnea.

The presence of apneas characterized by upper airway narrowing or closure despite continuing respiratory effort is defined as OSAS, and the presence of apneas that occur after a decrease or cessation of airflow secondary to lost or decreased respiratory effort is defined as CSAS (Figure 1 and 2). The majority of sleep laboratory patients are diagnosed with OSAS. However, when sleep studies are conducted in patients with MS, the rate of OSAS diagnosis can be as high as 62%. This rate increases particularly in the phase of (REM) sleep, which is distinguished by rapid movements and a state of muscular

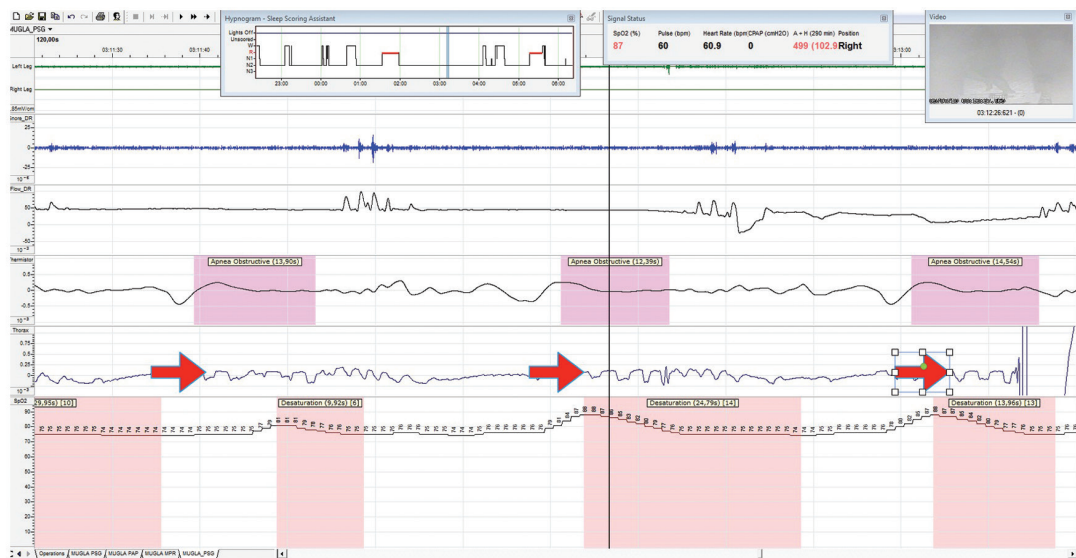


Figure 1. During the polysomnography recording, obstructive sleep apnea is observed in the epoch where respiratory effort (indicated by the red arrow) persists during apneas (highlighted in pink), leading to desaturations (shown in salmon color) detected in pulse oximetry

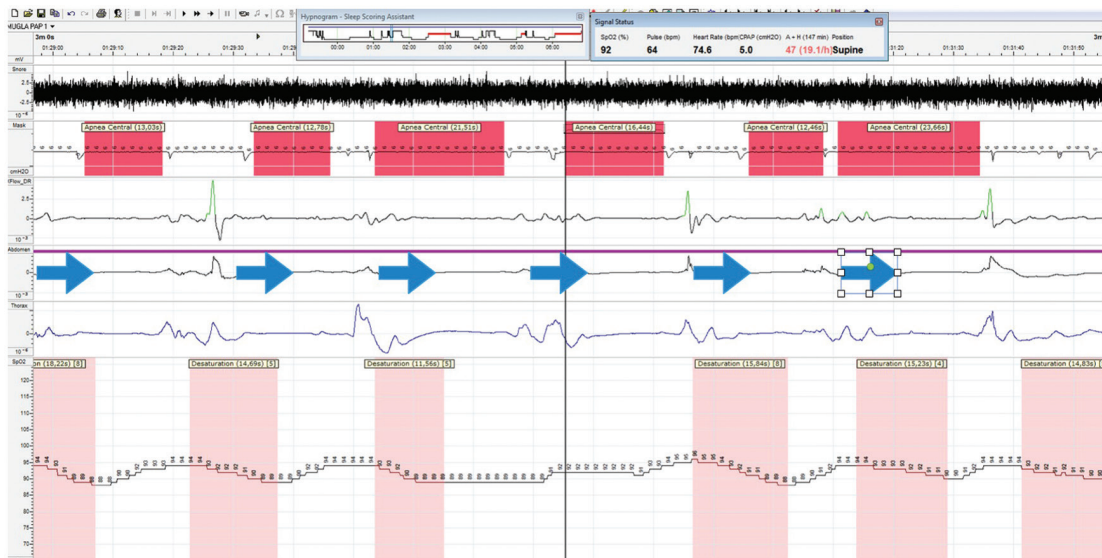


Figure 2. During the epoch captured in the polysomnography record, central sleep apnea is observed. This is characterized by a lack of respiratory effort (indicated by the blue arrow) during apneas (highlighted by the red arrow), leading to desaturations (shown in salmon color) detected in pulse oximetry

atonia. As expected, the frequency of OSAS tends to increase in individuals with brainstem lesions. However, studies indicate that the rise in OSAS frequency is associated with low Expanded Disability Status Scale (EDSS) scores rather than with brainstem lesions.¹³⁻¹⁵ CSAS, which occurs in 1% of the healthy population, is 8 times more prevalent in patients with MS. Although CSAS is more common in pontine and mesencephalic lesions, as expected, this increase in frequency was not found in medullary lesions.¹⁶⁻¹⁸

The coexistence of apnea and MS has been determined to be directly related to fatigue, daytime sleepiness, neurocognitive dysfunction, and falls.¹⁹ Therefore, sleep disturbance should be considered in patients experiencing falls that are unrelated to the EDSS score.

Even when subjective scales are used to diagnose apnea, the literature indicates that this approach is not very objective for patients with MS. The treatment of apnea primarily involves the application of positive airway pressure (PAP). In the study conducted in the polysomnography (PSG) unit, the appropriate

pressure level (continuous or bi-level) of PAP pressure that will decrease apneas and hypopneas and prevent the patient's oxygen desaturation is identified. Subsequently, the patient is instructed to use the PAP device at this optimal pressure. It has been observed that fatigue levels decrease after PAP application in patients with MS with moderate apnea.²⁰ In another case series, a decrease in neurocognitive dysfunction was observed after PAP treatment in patients with MS with apnea.²¹ Apart from PAP, treatment options include reducing body weight, cessation of smoking and alcohol consumption, using orthoses to relax the airway, and surgical intervention for cases unresponsive to PAP.¹⁹

Central Disorders of Hypersomnolence: It is characterized by the existence of daytime sleepiness without nighttime sleep disorders or circadian rhythm sleep disorders. In general, the examination encompasses narcolepsy types I and II, idiopathic hypersomnia, Klein-Levin syndrome (characterized by disinhibition and eating disorder between long sleep attacks), other causes of hypersomnolence, insufficient sleep syndrome, isolated symptoms, and normal variants (such as long sleepers who sleep for more than 10 hours). Subjective scales that evaluate daytime sleepiness and the multiple sleep latency test performed in the PSG laboratory are helpful in diagnosis.

It has been reported that central hypersomnolence is observed in 53% of patients with MS experiencing daytime sleepiness and fatigue.²² Narcolepsy is six times more prevalent in patients with MS.²³ Owing to the restricted quantity of cases, literature information is primarily based on case reports.^{24,25} The treatment approach does not vary from that of patients without MS.

Circadian Rhythm Sleep-wake Disorders: It is basically examined under the headings of delayed sleep-wake phase disorder, advanced phase sleep syndrome, irregular sleep-wake rhythm disorder, non-24-hour sleep-wake disorder, other causes of hypersomnolence, shift work disorder, and jet lag. Generally, delayed sleep-wake phase disorder is more common in adolescence, while it is more widespread in the geriatric age group. Shift work disorder is most commonly observed in health and safety workers. Demyelination of the afferents and efferents of the suprachiasmatic nucleus may be attributed to the effects on the anatomical structures responsible for regulating the circadian rhythm and can be observed in 30% of patients with MS.¹⁹ Compliance with sleep hygiene is a fundamental element of the therapeutic approach. Bright light therapy is a widely used treatment option, particularly in Scandinavian countries. Melatonin remains a viable therapeutic option; however, its bioavailability is significantly reduced to approximately 20% due to the effects of hepatic first-pass metabolism.²⁶

Parasomnia: Parasomnia refers to undesirable physical events or behaviors occurring during sleep onset, sleep, or awakening. Parasomnias are classified into two main categories based on sleep phase: REM sleep or NREM sleep. Relatively common NREM parasomnias include confusional arousal, somnambulism, pavor nocturnus, and sleep-related eating disorder. REM Parasomnias include REM sleep behavior disorder, recurrent

isolated sleep paralysis, and nightmare disorder. Among others, these include exploding head syndrome, sleep-related hallucinations, enuresis, and sleep talking.

Almost all of the sleep disorders categorized under this heading are not more prevalent in patients with MS compared to those without MS, and the treatment approach remains unchanged. However, if new complaints occur even though there is no sleep disorder in the premorbid condition, a new lesion should be searched for through cerebral imaging. Only REM sleep behavior disorder is observed six times more frequently in patients with MS. The fact that the lesion is anatomically close to the pedunculopontine nucleus suggests that glutamatergic neurons located in the pontine sublaterodorsal nucleus are responsible for its pathology.¹⁷

Sleep-related Movement Disorders: This is the primary sleep disorder most commonly reported by patients. RLS, periodic limb movement disorder (PLMD), isolated symptoms, and normal variants (excessive fragmentary myoclonus, hypnagogic foot tremor, hypnic jerks) fall under this category.

RLS is clearly distinguished from PLMD in that it occurs while awake rather than asleep (Figure 3). It is twice as common in patients with MS compared to the healthy population. In some studies, frequencies of up to 65% have been reported.¹⁷ Although iron deficiency constitutes the predominant etiology of RLS, in secondary RLS patients do not support this association. Hypothetically, the condition is common in patients whose descending dopaminergic pathways are affected.²⁷⁻²⁹ Advanced age, high EDSS scores, and cervical cord lesions are more common in patients, but their presence is not associated with the severity of RLS. Patients with MS who have RLS exhibit higher fatigue scores. It occurs concurrently with the diagnosis of MS in half of the patients. However, in the other half, it begins before the disease. It is considered that this may be related to spinal involvement, which does not cause clinical findings before diagnosis. Pathological changes in the spinal cord that current imaging methods cannot detect may trigger RLS.³⁰

Low-dose evening treatment with dopamine agonists (e.g., pramipexole, ropinirole) is recommended for patients with MS diagnosed with RLS. If iron deficiency is present, replacement is recommended. Antiemetics, antipsychotics, antidepressants, and antihistamines, among other symptomatic agents used by the patient, can exacerbate RLS symptoms. Discontinuing these medications may be enough to alleviate the symptoms.

Periodic leg movements during sleep are more common in patients with MS (Figure 4). It is posited to originate from decreased supraspinal inhibitory control. However, when accompanied by RLS, it creates an indication for treatment.¹⁷ Excessive fragmentary myoclonus may occur due to increased excitability of spinal segmental motor generators.¹⁷

The coexistence of sleep disorders and MS is now widely accepted based on numerous studies. Sleep disorders are observed in 24% to 62% of individuals diagnosed with MS. Sleep disruption exerts a negative influence on the comprehensive quality of life, both individually and cumulatively, by increasing

fatigue, depression, and disability.³¹ A vicious cycle occurs as fatigue, depression, and disability also affect sleep, leading to insomnia and daytime sleepiness.³² It is inevitable that cognitive deterioration will also become part of this vicious circle. Do attacks, disease-modifying treatments, or symptomatic treatments in MS have an effect on sleep, or do sleep disorders trigger attacks in MS? Interferon beta, methylprednisolone, selective serotonin reuptake inhibitors, modafinil, and methylphenidate have sleep-decreasing effects, while baclofen, tizanidine, gabapentin, oxybutynin, and carbamazepine have sleep-promoting effects.^{33,34} It has been reported that sleep disturbance can increase attacks by 1.75 times.³⁵

Conclusion

Sleep disorders and MS, whether experienced individually or concurrently, negatively impact the patients' quality of life. In the long journey of MS, which develops on an autoimmune basis and managed through disease-modifying and symptomatic treatments rather than curative ones, it is crucial in clinical practice to inquire about our patients' sleep disorders, identify any issues, and implement appropriate interventions to minimize the patient's disability. Of course, correct questioning is only possible by recognizing sleep disorders. In this study, sleep disorders are evaluated under different headings, and their relationship with MS is discussed based on current literature data.

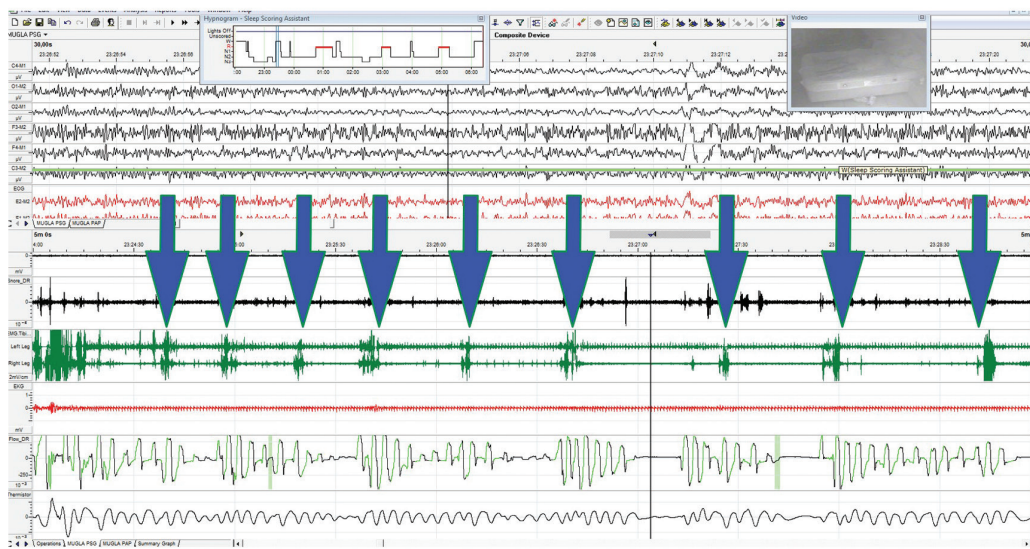


Figure 3. In the polysomnography recording, the epoch showing leg movements characteristic of restless legs syndrome (indicated by the dark blue arrow) recorded from the anterior tibial muscle electrodes (shown in green) while the patient is awake is observed

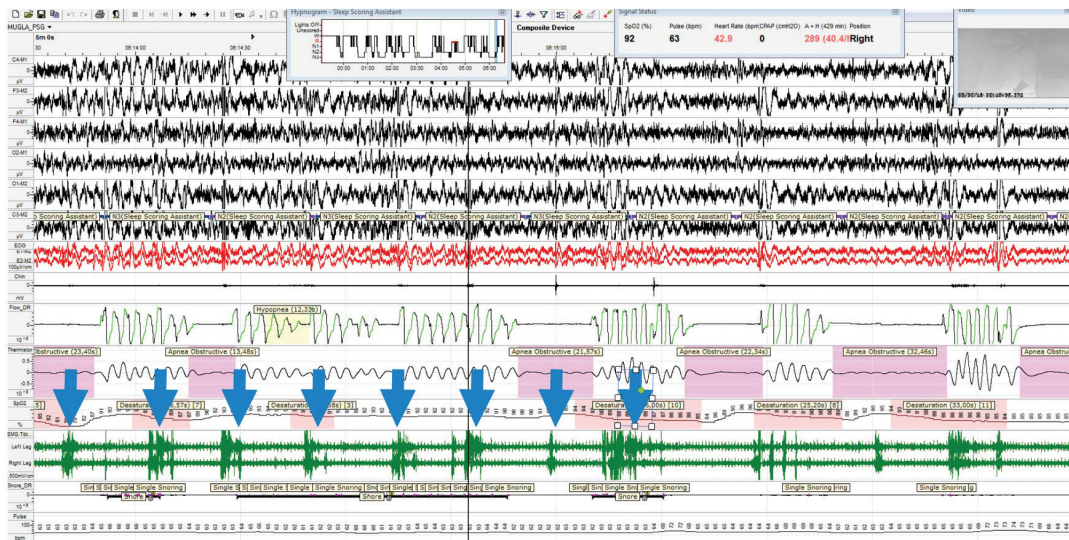


Figure 4. In the polysomnography recording, the epoch shows the leg movements observed during the periodic leg movements of sleep (blue arrow) recorded from the anterior tibial muscle electrodes (green color) while the patient was asleep

Footnotes

Authorship Contributions

Concept: İ.Ö., S.B., G.K., Design: İ.Ö., S.B., G.K., Data Collection or Processing: İ.Ö., S.B., G.K., Analysis or Interpretation: İ.Ö., S.B., G.K., Literature Search: İ.Ö., S.B., G.K., Writing: İ.Ö., S.B., G.K.

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Evaluation of Treatment Effects on Patients with Sleep-related Breathing Disorders and Epilepsy Comorbidity

Uyku ile İlişkili Solunum Bozuklukları ve Epilepsi Komorbiditesi Olan Hastalarda Tedavi Etkilerinin Değerlendirilmesi

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Abstract

Objective: Epilepsy and sleep-related breathing disorders (SBD) are among the most prevalent conditions in neurology. This study aimed to compare sleep parameters in patients with primary snoring-epilepsy (PrS-E) and severe obstructive sleep apnea syndrome-epilepsy (OSAS-E). Additionally, the study sought to assess the mutual effects of treatments administered in each clinical area on the other.

Materials and Methods: Patients who were subjected to follow-up in the sleep laboratory and epilepsy outpatient clinic between 2006 and 2022 were analysed retrospectively, following approval from ethics committee. The demographic data, medical history, characteristics of epileptic seizures, electroencephalograms (EEGs) and antiseizure drugs (ASD) before and after SBD treatment were compared. The effect on sleep structure and respiratory issues of lowering the dose of ASD after SBD treatment was also assessed.

Results: The study included 28 patients with PrS-E and 28 patients with severe OSAS-E. Focal onset impaired awareness focal to bilateral tonic-clonic seizures were found to be more prevalent in both groups. Following treatment for SBD, there was a decline in seizure severity and frequency, a resolution of EEG pathologies, or a decrease ASDs in both groups. The reduction in ASDs and treatment for SBD resulted in an improvement in sleep structure, a reduction in body mass index, snoring, and Epworth Sleepiness Scale scores.

Conclusion: Treating SBDs can lead to a reduction in the dosage of ASD, improved sleep structure, and decreased snoring. This study demonstrated mutual benefits in the management of both epilepsy and SBDs. SBD symptoms should be investigated in epilepsy patients, and the presence of epileptic seizures should be considered in patients with SBD.

Keywords: Obstructive sleep apnea syndrome, primary snoring, epilepsy, antiepileptic drug, body mass index, Epworth Sleepiness Scale

Öz

Amaç: Nörolojide en çok görülen hastalıklar arasında epilepsi ve uyku ile ilişkili solunum bozuklukları (USB) yer almaktadır. Çalışmamızda primer horlama-epilepsi (PrH-E) ile ağır obstruktif uyku apne sendromu-epilepsi (OUAS-E) birlikteliği olan hastalarda uyku parametrelerinin karşılaştırmasının yanı sıra, her iki klinikte uygulanan tedavilerin birbirlerine olan etkisinin değerlendirilmesi amaçlandı.

Gereç ve Yöntem: 2006-2022 yılları arasında kurumumuz uyku laboratuvarı ve epilepsi polikliniğinde takip edilen hastalar etik kurul onayı sonrası retrospektif olarak incelendi. USB tedavisi öncesi ve sonrası demografik veriler, tıbbi öykü, epileptik nöbetlerin özellikleri, elektroensefalogramlar (EEG) ve antiepileptik ilaç (AEI) tedavileri karşılaştırıldı. Ek olarak, USB tedavisi sonrası AEI'nin doz ve sayısının azaltılmasının uyku yapısı ve solunum yolu üzerindeki etkileri değerlendirildi.

Bulgular: Çalışmaya 28 PrH-E hastası ve 28 ağır OUAS-E hastası dahil edildi. Farkındalığın bozulduğu fokal başlangıçlı bilateral tonik-klonik nöbetler her iki grupta daha sık bulundu. USB tedavisini takiben, her iki grupta da nöbet şiddeti ve sıklığında bir azalma, EEG patolojilerinde bir azalma veya AEI'lerin sayısında ve dozlarında azalma vardı. AEI'lerdeki azalma ve USB tedavisi, uyku yapısında iyileşme, vücut kitle indeksinde düşüş, horlama ve Epworth Uykululuk Ölçeği skorlarında bir azalma ile sonuçlandı.

Sonuç: Uykuda solunum bozukluklarının tedavisiyle AEI'nin sayısı ve dozu azalmakta, uyku yapısı düzelmekte ve horlama yakınması azalmaktadır. Çalışmamızda tedavi ile hem epilepsi hem de USB kliniklerinde karşılıklı düzelmeler saptanmıştır. Epilepsili hastalarda USB ile ilişkili şikayetler ve USB'li hastalarda ise olası epileptik nöbet varlığı sorgulanmalıdır.

Anahtar Kelimeler: Obstruktif uyku apne sendromu, primer horlama, epilepsi, antiepileptik ilaç, vücut kitle indeksi, Epworth Uykululuk Ölçeği

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Introduction

Epilepsy and sleep-related breathing disorders (SBD) are among the most common clinical conditions encountered in neurology.¹ SBD encompasses a range of disorders, from primary snoring (PrS) to severe obstructive sleep apnea syndrome (OSAS).² These conditions can occur independently or coexist, and early recognition of SBD is crucial, as it exacerbates the frequency and severity of epileptic seizures. In cases where both SBD and epileptic seizures are present, treatment management becomes more complex, leading to more frequent complications.^{3,4}

OSAS is characterized by repetitive reductions or interruptions of airflow in the upper respiratory tract during sleep. Its hallmark symptoms include witnessed apneas, particularly snoring, and excessive daytime sleepiness.⁵ Polysomnography (PSG) is the gold standard for diagnosing OSAS. The severity of OSAS is determined by the Apnea-Hypopnea Index (AHI), which measures the total number of apneas and hypopneas per hour of sleep. AHI values of 5-14 indicate mild OSAS, 15-29 indicate moderate OSAS, and AHI >30 indicates severe OSAS. PrS refers to respiratory noise during the inspiratory (and sometimes expiratory) phases of breathing during sleep, without associated sleep apnea.⁶ Treatment for PrS includes conservative approaches, intraoral implants, and surgery,⁷ while OSAS treatment involves positive airway pressure (PAP) therapy and/or upper airway surgery.⁸⁻¹⁰

Patients with epilepsy have a higher prevalence of OSAS compared to the general population. OSAS can lower the seizure threshold through mechanisms such as sleep fragmentation, oxygen desaturation, and chronic sleep deprivation.⁹⁻¹¹ Older age, and having sleep disorders are all associated with a higher risk of epilepsy.¹¹ Furthermore, it has been established that certain conditions, including the occurrence of refractory seizures, the utilisation of antiseizure medications, and the administration of antiseizure drugs (ASDs) in the form of polytherapy, render these patients more susceptible to OSAS. Although data on the efficacy of PAP therapy for controlling seizures are limited, retrospective studies suggest it can significantly reduce seizure frequency, excessive daytime sleepiness, and cognitive complaints.¹²

The relationship between sleep and epilepsy is complex, with each condition influencing the other in various ways. In epilepsy patients with SBD, seizures may become more resistant and harder to manage. OSAS increases seizure frequency and severity, as well as daytime sleepiness in these patients.¹³ It also reduces oxygen levels in the brain, disrupts natural sleep patterns, and triggers spike discharges and paroxysmal activities on electroencephalography (EEG), further aggravating epileptic seizures. However, effective OSAS treatment can lead to better seizure control and a reduction in epileptiform activity on EEG.¹⁴ In our study, we aimed to compare the demographic data, disease duration, clinical findings, PSG data, seizure types, seizure frequency and EEG examination results between patients with primary snoring-epilepsy (PrS-E) and those with severe obstructive sleep apnea syndrome-epilepsy (OSAS-E).

Materials and Methods

This study was conducted as a retrospective study following the approval of Manisa Celal Bayar University Faculty of Medicine Clinical Research Ethics Committee (approval number: 307, date: 25.07.2022). The study population comprised 56 patients who were under follow-up in the Department of Neurology-Sleep Outpatient Clinic-Sleep Laboratory and Epilepsy Outpatient Clinic of Private Buca Hospital between January 2006 and June 2022. The study population was further subdivided into two groups: 28 patients with PrS-E and 28 patients with severe OSAS-E. It should be noted that the follow-up and treatment of these patients are ongoing.

Inclusion Criteria

- The patient has been diagnosed with epilepsy [2017 International League Against Epilepsy (ILAE)] and PrS (American Academy of Sleep Medicine Guidelines).
- The diagnosis of epilepsy (2017 ILAE) is made, and the patient also exhibits severe OSAS (American Academy of Sleep Medicine Guidelines).
- Normal brain imaging results, including magnetic resonance imaging and/or other imaging methods such as cranial brain tomography.
- Age over 18 years.
- Voluntary consent form obtained.

Exclusion criteria encompassed epileptic seizures attributable to alternative etiologies, substance abuse, psychiatric diagnoses necessitating pharmacotherapy, mental retardation, other neurodegenerative diseases, syndromic seizures, irregular use of antiseizure medications and vagal nerve stimulators, and epilepsy surgery.

Data Acquisition Tools

The parameters compared between the PrS-E and severe OSAS-E groups included demographic data [age at presentation, gender, body mass index (BMI)], medical history, birth history, duration of epilepsy, duration of snoring, relationship of seizures with sleep, comorbidities (if present). Additional factors included epilepsy features, EEG findings and treatments for epilepsy. Pathological EEG findings were classified as paroxysmal activity disorders with non-specific or epileptiform potential, generalised epileptiform discharges, focal epileptiform discharges, paroxysmal lateralising epileptiform discharges and bilateral paroxysmal lateralising epileptiform discharges were considered pathological. Furthermore, a comparison of the features of epilepsy (clinical and electroencephalographic) after treatment for PrS or severe OSAS was performed. Metrics evaluated in patients diagnosed with PrS-E and severe OSAS-E included the AHI, sleep latency duration, N1, N2, N3, rapid eye movement (REM) latencies, percentages of N1, N2, N3, REM durations, fragmentations, respiratory arousal index, arousal index associated with leg movements (LM), spontaneous arousal index, and sleep efficiency [ratio of total sleep time (TST) to time in bed multiplied by 100 to yield a percentage].

In addition to the aforementioned data, the effects of the SBD treatment on epilepsy and secondary sleep structure were also investigated. The Epworth Sleepiness Scale (ESS) was performed on all patients both before and after SBD treatment in order to determine excessive sleepiness. The ASD used by all patients were recorded both before and after SBD treatment.

Epworth Sleepiness Scale

To assess excessive sleepiness, questionnaire forms are used that vary in compatibility with objective sleepiness measurement methods. The most commonly used of these is the ESS. The ESS includes questions such as, "Do you feel drowsy, sleepy, or experience somnolence?" for each item, scoring is based on the following options: 0=never, 1=mildly, 2=moderately, and 3=severely. The total score ranges from 0 to 24, with sleepiness assessed by summing the scores from the patient's responses. A score of 10 or above is classified as hypersomnia. The Turkish validity and reliability of the ESS have been confirmed in our country.¹⁵

Statistical Analysis

The data obtained from the study were entered into a database created using IBM SPSS 25 (SPSS Inc., Chicago, IL, USA), and statistical analyses were conducted using the same software. For continuous variables, the mean, standard deviation, median, minimum, and maximum values were calculated. The normality of the variables was assessed by using the Shapiro-Wilk test. Independent groups were compared using the Mann-Whitney U test, and pre- and post-treatment comparisons were analyzed with the Wilcoxon test. The chi-square test was employed to assess whether the relationship between two nominal variables was statistically significant. In all statistical tests, a "p" value of less than 0.05 was considered to indicate a statistically significant difference between groups.

Results

Of the 56 patients included in our study, 28 were in the PrS-E group and 28 were in the severe OSAS-E group. The mean age of all patients was 46.54±15.68 years (range: 19-82). Of these, 17 patients (30.4%) were female and 39 (69.6%) were male (p=0.380).

Regarding birth histories, 50 patients (89.3%) were born via normal spontaneous vaginal delivery, while 6 patients (10.7%) were born by cesarean section. Childhood epilepsy was reported in 8 patients (14.3%), and childhood snoring in 37 patients (66.1%).

Physical examination revealed that, based on the Mallampati score (class 1, 2, 3), 30 patients (53%) were classified as Mallampati class 1, 12 patients (22%) as Mallampati class 2 and 14 patients (25%) Mallampati class 3. When considering jaw anatomy, 42 patients (75%) had normal anatomy, 12 patients (21%) had mandibular prognathism, and 2 patients (3%) had micrognathia. In terms of neck anatomy, 13 patients (23%) had a short neck, while 43 patients (77%) had a normal neck.

BMI assessments showed that 16 patients (28.5%) had a normal BMI (<25), 25 patients (44.6%) had a BMI between 25-35, and 15 patients (26.7%) were morbidly obese (BMI >35).

When all patients were evaluated according to seizure types, 30 (53%) patients exhibited focal onset impaired awareness focal to bilateral tonic-clonic seizures, 14 (25%) patients presented with generalised onset motor tonic-clonic seizures. Six (10%) patients had generalised onset motor myoclonic seizures, three (6%) patients had focal onset aware motor automatism seizures and 3 (6%) patients had focal onset aware motor clonic seizures.

In the PrS-E group, 14 patients (50%) exhibited focal onset impaired awareness seizures progressing to bilateral tonic-clonic seizures, 6 patients (21%) presented generalised onset motor tonic-clonic seizures, 4 patients (14%) had generalised onset motor myoclonic seizures, 2 patients (7.5%) experienced focal onset aware motor automatism seizures and 2 patients (7.5%) had focal onset aware motor clonic seizures.

In the severe OSAS-E group, 16 patients (58%) exhibited focal onset impaired awareness seizures progressing to bilateral tonic-clonic seizures, 8 patients (28%) demonstrated generalised onset motor tonic-clonic seizures, 2 patients (7.5%) had generalised onset motor myoclonic seizures, 1 patient (3.25%) experienced focal onset aware motor automatism seizures, and 1 patient (3.25%) had focal onset aware motor clonic seizures.

The analysis revealed no significant disparities in seizure frequency between the two groups. The demographic data, childhood symptoms, physical examination findings and epilepsy-related characteristics of all patients and patient groups are outlined in Table 1.

Before treatment of sleep-related disorders, the ESS was 9.73±6.77 (1.00-24.00), whereas after treatment, the ESS was 4.66±2.89 (1.00-12.00) (p<0.001). The data obtained in the PSG examination of all patients and patient groups and ESS values were summarized in Table 2.

Before treatment for SBD, 30 patients (53.6%) were on monotherapy for epilepsy, while 26 patients (46.4%) were on polytherapy. The mean seizure frequency was 5.02±4.63 (range: 1-12) per year. Regarding EEG activity, 20 patients (35.7%) showed paroxysmal activity disorder, 10 patients (17.9%) had paroxysmal activity disorder with epileptiform potential, 5 patients (8.9%) had generalized epileptiform discharges, and 4 patients (7.1%) had focal epileptiform activity. EEG results were normal in 17 patients (31%).

After SBD treatment, 40 patients (71.4%) were on monotherapy, while 16 patients (28.6%) were on polytherapy. The mean seizure frequency decreased to 3.21±2.92 per year (range: 0-12). When post-treatment EEG results were evaluated as normal or pathological, 35 patients (62.5%) had normal EEGs, while 21 patients (37.5%) showed pathological EEG findings during their treatment for sleep-related disorders.

The results of the pre- and post-treatment evaluations of patients with PrS-E are displayed in Table 3, while those with severe OSAS-E are shown in Table 4.

In terms of seizure frequency prior to PrS treatment, the average frequency was 3.25±3.84 per year (range: 1.00-12.00). Four patients (14%) experienced seizures once a month, 6 patients (21%) had seizures once every 3 months, 8 patients (29%)

	All patients (n=56) mean ± SD (minimum-maximum)	PrS-E (n=28) mean ± SD (minimum-maximum)	Severe OSAS-E (n=28) mean ± SD (minimum-maximum)	p-value PH + E vs. severe OSAS-E
Age	46.54±15.68 (19-82)	37.68±12.66 (19.00-66.00)	55.39±13.32 (22.00-82.00)	<0.001 [¶]
Gender F/M	17/39	7/21	10/18	0.380 ^{¶2}
Age at admission	38.45±15.39 (18-72)	27.75±10.17 (18.00-56.00)	49.14±11.89 (18.00-72.00)	<0.001 [¶]
Age of onset of epilepsy	30.73±13.91 (3-65)	25.25±10.02 (8.00-46.00)	36.21±15.21 (3.00-65.00)	0.004 [¶]
Epilepsy duration (years)	15.80±8.03 (2-40)	12.53±5.16 (2.00-22.00)	19.18±9.01 (8.00-40.00)	0.005 [¶]
Duration of adult snoring (years)	20.25±15.28 (1-62)	17.2±7.7 (1.00-46.00)	31.46±13.26 (5.00-62.00)	<0.001 [¶]
Type of birth (N/CS)	50/6	23/5	27/1	0.083 ^{¶2}
Childhood snoring (yes/no)	37/19	22/6	15/13	0.048 ^{¶2}
Childhood epilepsy (yes/no)	8/48	5/23	3/25	0.445 ^{¶2}
Mallampati score (n)	Class I: 30 Class II: 12 Class III: 14	Class I: 17 Class II: 9 Class III: 2	Class I: 13 Class II: 3 Class III: 12	0.004 ^{¶2}
Jaw anatomy (n)	Normal: 42 Mandibular prognathism: 12 Micrognathia: 2	Normal: 19 Mandibular prognathism: 8 Micrognathia: 1	Normal: 23 Mandibular prognathism: 4 Micrognathia: 1	0.424 ^{¶2}
Neck anatomy (n)	Normal: 43 Short neck: 13	Normal: 25 Short neck: 3	Normal: 18 Short neck: 10	0.026 ^{¶2}
BMI	28.87±7.28 (17.30-46.10)	24.03±4.19 (17.30-33.60)	33.70±6.47 (23.10-46.10)	<0.001 [¶]
Epilepsy seizure type (n)	FIAFBTC: 30 GMTC: 14 GMMC: 6 FAMAC: 3 FAMCC: 3	FIAFBTC: 14 GMTC: 6 GMMC: 4 FAMAC: 2 FAMCC: 2	FIAFBTC: 16 GMTC: 8 GMMC: 2 FAMAC: 1 FAMCC: 1	0.566 ^{¶2}
Seizure frequency (number per year)	5.02±4.63 (1-12)	3.25±3.83 (1-12)	6.79±4.73 (1-12)	0.530 [¶]

PrS-E: Patients with primary snoring and epilepsy, Severe OSAS-E: Patients with severe obstructive sleep apnea syndrome and epilepsy, SD: Standard deviation, F: Female, M: Male, N: Normal delivery, CS: Caesarean section, BMI: Body mass index, FIAFBTC: Focal onset impaired awareness focal to bilateral tonic-clonic seizures, GMTC: Generalized onset motor tonic-clonic seizures, GMMC: Generalized onset motor myoclonic seizures, FAMAC: Focal onset aware motor automatisms seizures, FAMCC: Focal onset aware motor clonic seizures, [¶]: Mann-Whitney U testi, ^{¶2}: Chi-square test

had seizures once a year, and 10 patients (36%) experienced seizures less than once a year. The results of the study demonstrated a significant decrease in seizure frequency and excessive daytime sleepiness following SBD treatment ($p=0.004$ and $p<0.001$, respectively). The rate of pathological EEG findings also decreased after treatment for PrS.

In the PrS-E group, before SBD treatment, 16 patients (57%) were on monotherapy, and 12 patients (43%) were on polytherapy. After treatment, 23 patients (89%) were on monotherapy, while 3 patients (11%) remained on polytherapy, showing a statistically significant change in treatment approach ($p=0.002$) (Table 3).

In severe OSAS-E group, following treatment for severe OSAS, it was observed that seizure frequency decreased in 13 patients (46%), increased in 1 patient (4%), and remained unchanged in 14 patients (50%). Overall, there was a significant reduction in both seizure frequency and daily excessive sleepiness complaints

following treatment ($p<0.001$). Additionally, a comparison of pre- and post-treatment EEG examinations showed a significant decrease in pathological findings ($p<0.001$).

In severe OSAS-E group regarding medication use, the number and/or doses of medications remained unchanged in 17 patients (61%), while 11 patients (39%) experienced a reduction in the number and/or doses of medications. In terms of monotherapy *versus* polytherapy, 12 patients (43%) were on monotherapy and 16 patients (57%) were on polytherapy before treatment. After treatment, 14 patients (50%) were on monotherapy, and 14 patients (50%) were on polytherapy. However, this change in therapy was not statistically significant ($p=0.895$) (Table 4).

An evaluation of the impact of modifications to antiseizure treatments on snoring, revealed that the weight reduction associated with these changes led to a decrease in snoring among 4 patients (14%) diagnosed with PrS and treated for sleep-disordered breathing. Conversely, weight gain in

Table 2. Polysomnographic data of all patients and both groups

	All patients (n=56) mean ± SD (minimum-maximum)	PrS-E (n=28) mean ± SD (minimum-maximum)	Severe OSAS-E (n=28) mean ± SD (minimum-maximum)	p-value PH+E vs. severe OSAS-E
Apnea-hypopnea index	23.67±24.96 (0.10-89)	1.44±1.48 (0.10-4.90)	45.89±15.57 (30.10-89.00)	<0.001 [¶]
N1_latency (min.)	16.56±12.04 (0.30-49.50)	13.56±13.05 (0.30-49.50)	19.57±10.31 (10.10-47.30)	0.003 [¶]
N2_latency (min.)	27.97±28.52 (2.80-180.30)	25.81±36.68 (2.80-180.30)	30.13±17.39 (13.40-96.50)	0.002 [¶]
N3_latency (min.)	52.79±49.37 (9.30-281.30)	59.86±66.27 (9.30-281.30)	45.71±21.68 (17.30-107.50)	0.476 [¶]
REM latency (min.)	156.95±93.38 (41.50-473)	140.15±75.69 (41.50-379.00)	173.75±106.99 (46.30-473.00)	0.210 [¶]
N1 duration (%)	7.38±5.92 (0.30-23)	5.78±4.81 (0.3-19.10)	8.98±6.55 (0.50-23.00)	0.053 [¶]
N2 duration (%)	46.74±11.22 (22-71.20)	44.19±9.75 (22.00-61.30)	49.29±12.17 (25.80-71.20)	0.096 [¶]
N3 duration (%)	28.65±12.67 (0.80-60.90)	30.72±9.37 (10.30-54.30)	26.57±15.17 (0.80-60.90)	0.125 [¶]
REM duration (%)	16.02±7.02 (0.60-40)	18.10±8.23 (0.6-40.00)	13.94±4.85 (4.80-26.80)	0.022 [¶]
Respiratory arousal index	24.29±70.66 (0-417)	1.25±2.81 (0.00-12.00)	47.32±95.19 (0.00-417.00)	<0.001 [¶]
Leg movement arousal index	16.11±19.08 (0-77)	24.96±21.25 (0.00-77.00)	7.25±11.30 (0.00-42.00)	<0.001 [¶]
Sleep efficiency (%) (total sleep time/total recording time)	83.63±8.60 (70-99.40)	89.63±7.64 (75.30-99.40)	77.63±4.19 (70.00-84.60)	<0.001 [¶]
Min. SatO ₂	84.23±10.14 (54-97.70)	91.35±5.61 (73.00-97.70)	77.11±8.54 (54.00-87.00)	<0.001 [¶]
Pre-treatment ESS	9.73±6.77 (1-24)	3.75±2.25 (1.00-10.00)	15.71±3.76 (10.00-24.00)	<0.001 [¶]
Post-treatment ESS	4.66±2.89 (1.00-12.00)	2.50±1.26 (1.00-6.00)	6.82±2.39 (3.00-12.00)	0.036 [¶]

PrS-E: Patients with primary snoring and Epilepsy, Severe OSAS-E: Patients with severe obstructive sleep apnea syndrome and epilepsy, SD: Standard deviation, ESS: Epworth Sleepiness Scale, min.: Minute, ¶: Mann Whitney U, REM: Rapid eye movement

Table 3. Pre- and post-treatment evaluation of PrS-E patients

	Pre-treatment Mean ± SD (min.-max.)	Post-treatment Mean ± SD (min.-max.)	p
Seizure frequency (per year)	3.25±3.84 (1.00-12.00)	2.11±2.13 (1-8)	0.004 [¶]
EEG findings (normal/pathological)	14/14	19/9	0.344 ^{χ²}
ASD treatment (monotherapy/polytherapy)	16/12	23/3	0.002 ^{χ²}
ESS	3.75±2.25 (1.00-10.00)	2.50±1.26 (1.00-6.00)	<0.001 [¶]

PrS-E: Patients with primary snoring and epilepsy, EEG: Electroencephalography, ASD: Antiseizure Drug, ESS: Epworth Sleepiness Scale, SD: Standard deviation, min.: Minimum, max.: Maximum, ^{χ²}: Chi-square test, [¶]: Wilcoxon test

Table 4. Pre- and post-treatment evaluation of severe OSAS-E patients

	Pre-treatment Mean ± SD (min.-max.)	Post-treatment Mean ± SD (min.-max.)	p
Seizure frequency (per year)	6.79±4.73 (1-12)	4.32±3.21 (1-8)	<0.001 [¶]
EEG findings (normal/pathological)	3/25	16/12	<0.001 ^{χ²}
ASD treatment (monotherapy/polytherapy)	12/16	14/14	0.895 ^{χ²}
ESS	15.71±3.76 (10.00-24.00)	6.82±2.39 (3.00-12.00)	<0.001 [¶]

EEG: Electroencephalography, ASD: Antiseizure drug, ESS: Epworth Sleepiness Scale, SD: Standard deviation, Min.: Minimum, Max: Maximum, ^{χ²}: Chi-square test, [¶]: Mann-Whitney U test

4 patients (14%) resulted in increased snoring, which was attributed to weight gain associated with valproate treatment for other conditions. Snoring patterns unchanged in 20 patients (72%) (Supplementary Table 1). Additionally, 15 patients (53%) reported a positive effect on sleep, such as falling asleep faster and maintaining sleep continuity, while 3 patients (11%)

experienced negative effects, including difficulty falling asleep and staying asleep. Sleep patterns remained unaffected in 10 patients (36%).

An analysis was conducted to evaluate the effects of changes in antiseizure treatments on snoring among patients diagnosed and treated for severe OSAS. The results showed that weight

reduction due to ASDs led to a decrease in snoring in 8 patients (29%), while weight gain caused an increase in snoring in 4 patients (14%). Snoring patterns remained unchanged in 16 patients (57%) (Supplementary Table 2).

The impact of reducing ASDs on sleep was also analysed. Positive effects were observed in 17 patients (61%), characterized by reduced sleep latency and improved sleep continuity. Conversely, 4 patients (14%) experienced negative effects, including difficulty falling asleep and maintaining sleep, while 7 patients (25%) showed no change in sleep.

The impact of the reducing antiseizure medications on the occurrence of snoring was analyzed in both groups. The results showed that the intervention was effective in 12 patients (43%), and ineffective in 16 patients (57%) in the severe OSAS-E group. Conversely, it was effective in 8 patients (29%) and ineffective in 20 patients (71%) in the PrS-E group. Although the intervention appeared to be more effective in the severe OSAS group, the difference between the groups was not statistically significant ($p=0.403$).

Discussion

Epilepsy and SBD are among the most common neurological conditions encountered. Both sleep and epilepsy are dynamic processes within the central nervous system that influence one another. These conditions can occur independently or coexist. This study focused on patients with PrS-E and severe OSAS-E.

To date, studies on the association between PrS-E have only been conducted in children, making this the first study to evaluate this relationship in adults. Furthermore, no prior studies have compared the PrS-E and severe OSAS-E groups. This study also evaluated the clinical impact of treatments for both epilepsy and SBD.

In this study, demographic data, disease duration, clinical findings, polysomnographic data, seizure types, seizure frequency, and EEG results were assessed for both groups.

In both the PrS-E and severe OSAS-E groups, SBD treatment was observed to have a positive effect on epilepsy, while antiseizure treatment improved sleep structure and reduced complaints such as had a positive effect on epilepsy, and antiseizure treatment was effective in improving sleep structure and reducing complaints such as snoring through different mechanisms. While improvements in sleep structure and SBD-related symptoms, in addition to EEG findings in the epilepsy clinic, were observed in both groups following treatment, the severity of these improvements was found to be more pronounced in the severe OSAS-E group.

Numerous studies have been conducted to explore the association and frequency of epilepsy with SBD yielding varied results. In a study patients with epilepsy, the risk of OSAS in epilepsy patients was shown to be higher than in the general population, with an overall prevalence of 9%.¹⁶ In studies conducted within our country, the prevalence of OSAS among patients diagnosed with epilepsy has been reported to range from 10% to 55%.^{17,18} A meta-analysis of 26 studies estimated the prevalence of OSAS in epilepsy patients at approximately 33.4%.¹⁹ OSAS has been shown to be more common in drug-

resistant epilepsy. OSAS is reported in 10% of adult epilepsy patients, 20% of children with epilepsy, and 30% of patients with drug-resistant epilepsy.²⁰ In this retrospective study, 28 patients with PrS-E and 28 patients with severe OSAS-E were identified. The relatively small sample size, particularly in the severe OSAS group, was attributed to the retrospective design of the study, which may have been influenced by data loss.

Sleep interruption and deprivation in OSAS have been shown to increase the incidence of epilepsy.²⁰ Conversely, the hyperpolarisation-synchronisation of thalamocortical neurons caused by hypoxia during non-REM sleep in OSAS patients plays a significant role in the activation of epileptic neurons. Frequent nighttime awakenings in OSAS also promote synchronization, thereby lowering the seizure threshold.²¹ However, it is important to note that epilepsy, including refractory epilepsy, can occur not only in OSAS patients but also in those with PrS-E. Pathophysiologically, both PrS and severe OSAS disrupt sleep microstructure by reducing cerebral oxygen levels, leading to spike discharges and paroxysmal activity on EEG. Sleep spindles, particularly during superficial sleep stages, are thought to contribute to seizure development and propagation. This leads to occurrence of seizures both during and outside the ictal phase, while also disrupting sleep structure.²² Generalized tonic-clonic seizures are more frequent during non-REM sleep and less common during REM sleep.²¹ Generalized epileptiform discharges and interictal epileptiform activity are more commonly observed in non-REM sleep than in REM sleep.²³ Additionally, focal onset seizures, particularly those originating from the temporal lobes, are more frequently observed in patients with severe OSAS. Nocturnal seizures are commonly reported in individuals with OSAS, with focal seizures often associated with specific cortical regions, most notably the temporal lobes. The association between sleep fragmentation, hypoxia, and autonomic dysregulation in severe OSAS may increase the risk of focal seizures due to the heightened stress on the brain.²⁴

In this study, the most prevalent seizure type observed in patients with SDB and epilepsy was focal onset impaired awareness seizures progressing to bilateral tonic-clonic seizures, followed by generalised onset motor tonic-clonic seizures. This pattern was observed to be analogous in both PrS-E and severe OSAS-E groups.

PrS is a commonly observed in children and affects approximately 30-50% of the adult population, with a higher prevalence among middle-aged men.⁶ Severe OSAS is most common in individuals aged 40-65, with its prevalence declining after age of 65.^{24,25} While severe OSAS is more common in older adults, PrS can occur across all age groups. In our study, the mean age was 27 (range: 18-56) in the PrS-E group and 49 (range: 18-72) in the severe OSAS-E group. The prevalence of PrS was found to be three times more common in males, while OSAS was found to be 1.8 times more prevalent in males.

A substantial body of research has been conducted employing PSG in patients with sleep-disordered breathing.²⁶⁻²⁸ PSG examinations of patients with OSAS have reported that REM sleep duration is shortened, while N1 and N2 stages are

prolonged.²⁹ In our study, we observed that in the severe OSAS group, sleep efficiency and minimum oxygen saturation were lower, N1 and N2 latencies were prolonged, LM increased, N1 and N2 durations were prolonged, N3 and REM durations were shortened, and arousals were more frequent. Consequently, the ESS values were significantly higher in the severe OSAS group compared to the PrS group. Interestingly, sleep efficiency was also decreased in the PrS group, which was thought to be related to the co-occurrence of epilepsy, as epilepsy itself can disrupt sleep architecture. Additionally in the severe OSAS group, EEG examinations frequently revealed pathological findings with epileptiform discharges. A subsequent analysis of epileptic seizure characteristics revealed that the seizures were predominantly focal onset impaired awareness seizures progressing to bilateral tonic-clonic seizures. The treatment of epilepsy associated with SBD was another key area of discussion. Notably, antiseizure treatment in both study groups-particularly in the severe OSAS group-was predominantly administered as polytherapy, often involving three or more drugs. The data collectively suggested that seizures in these patients demonstrated resistance to treatment. The presence of SBD was identified as a potential contributing factor to this resistance. In the second phase of our study, after SBD treatments were administered to both groups, we re-evaluated and compared seizure frequency, EEG findings, antiseizure treatments. Shortened REM sleep, which normally suppresses epileptic activity, can increase seizure susceptibility. This highlights the importance of PAP therapy, which not only corrects hypoxia but also prolongs REM sleep, thereby reducing the likelihood of seizures.²⁹ PAP treatment has been shown to help control seizures more effectively and reduce epileptiform activity on EEG.¹⁴ Retrospective studies further indicate that it decreases seizure frequency and alleviates prolonged daytime sleepiness.¹³ Additionally, treating sleep disorders has been associated with a reduction in spike activity rates during the first sleep cycle, particularly in slow-wave sleep and across the entire cycle.^{22,30-32} Improvements in EEG findings, combined with reduced seizure frequency and severity following SBD treatment, have also been linked to decreases in both the number and dosage of ASDs.^{33,34} In our study, following the implementation of PAP therapy, which was utilised by all severe OSAS-E group patients, showed a significant reduction in seizure frequency. However, there was no significant change in ASD regimens (polytherapy vs. monotherapy). It is hypothesized that the more frequent and severe epileptic seizures observed in the severe OSAS group may be attributed to the smaller reduction in the number of drugs and the use of three or more drugs in polytherapy treatment. Literature has also emphasized the reduction in seizure frequency and severity after surgical treatment for adenoid hypertrophy, a primary cause of snoring in children.^{35,36} In contrast, this study examined the effects of appropriate treatments on adults with PrS, focusing on seizure frequency before and after treatment. The findings revealed a significant reduction in seizure frequency, highlighting a potential therapeutic benefit for individuals with this condition. However, no significant changes were observed in EEG findings,

likely because the EEG results were normal or nearly normal before treatment. It was also noteworthy that the transition from polytherapy to monotherapy was more prevalent in the PrS-E group.

Excessive daytime sleepiness and frequent nighttime awakenings are among the most common complaints in epilepsy patients.³² Acute neurochemical changes in the brain caused by seizures can disrupt sleep and wakefulness. Seizures and epileptic discharges during sleep interfere with both the initiation and maintenance of sleep. Generalized and partial seizures occurring during sleep may reduce TST and REM sleep, contributing to sleep disturbances.^{32,37}

Studies have also shown that, even during seizure-free periods, epilepsy patients experience sleep issues such as reduced sleep efficiency, altered sleep stages, and increased wakefulness, leading to morning fatigue and daytime sleepiness.^{38,39} In this study, the mean ESS score in the PrS-E group was 3.75 before SBD treatment, and decreased to 2.50 after treatment. In the severe OSAS-E group, the mean ESS score was 15.71 before treatment and decreased to 6.82 after treatment.

In addition to the effects of SBD on epilepsy, ASDs can also alter the natural architecture and organization of sleep, affecting sleep stages and wakefulness.⁴⁰ The use of antiseizure medications has been showed to exacerbate SBD, primarily due to the relaxation of upper airway muscles and the elevation of the arousal threshold through various mechanisms. Spontaneous arousals and frequent awakenings caused by ASDs significantly disrupt the sleep patterns of epilepsy patients.^{39,41} Medications such as phenobarbital, valproate, gabapentin, and pregabalin have been shown to negatively impact OSAS.⁴² Patients with drug-resistant epilepsy on polytherapy may have a higher risk of obesity compared to those on monotherapy.⁴³ ASDs associated with weight gain include valproic acid, pregabalin, gabapentin, and vigabatrin.^{44,45} While the effect of carbamazepine on weight remains unclear, lamotrigine generally does not affect weight. In contrast drugs such as felbamate, topiramate, and zonisamide are linked to weight loss.^{46,47} Weight gain associated with ASDs may also exacerbate OSAS.⁴⁸

ASDs affect not only weight and metabolism but also sleep patterns. Medications such as phenobarbital, phenytoin, carbamazepine, valproate, gabapentin, topiramate, vigabatrin, levetiracetam, pregabalin, oxcarbazepine, rufinamide, and clobazam are reported to increase sleepiness. Conversely ethosuximide, felbamate, lamotrigine, and zonisamide may cause insomnia. Additionally phenobarbital, phenytoin, carbamazepine, and pregabalin have been shown to reduce sleep latency.⁴⁹

The findings of this study demonstrated that the majority of patients diagnosed with PrS-E who received SBD treatment did not require changes to their epilepsy treatment. An increase in the use of monotherapy was observed, while snoring decreased in some cases, and excessive daytime sleepiness diminished in others, corresponding with a reduction in ASD use. Additionally more than half of these patients experienced faster sleep onset and improved sleep continuity, leading to an overall enhancement in sleep structure.

Approximately one-third of patients receiving PAP therapy for severe OSAS-E demonstrated a significant reduction in snoring, which was attributed to a reduction in ASDs (though polytherapy was often continued) and weight loss. Additionally, notable improvements were observed in sleep initiation and continuity for most patients with severe OSAS-E, along with a more pronounced reduction in excessive daytime sleepiness.

Study Limitations

In summary, the treatment of both epileptic seizures and SBD resulted in a reduction in the number and dosage of ASDs, improved sleep structure, and decreased daytime sleepiness and snoring, particularly in the severe OSAS group. However, the study's limitations include its small sample size and retrospective design. This study is the first to report on these features in the PrS-E group. However the study design, which included PSG testing before and after treatment, limited ability to compare PSG data comprehensively. This methodological limitation is an important aspect that should be addressed in future research.

Conclusion

In conclusion, understanding the relationship between epilepsy and sleep disorders, as well as identifying and treating coexisting sleep-related issues, can reduce the frequency and severity of epileptic seizures, decrease the number and dosage of ASDs, and improve EEG findings. Effective seizure management and optimization of medication regimens can enhance sleep quality, including the ability to fall asleep and maintain asleep, while significantly reducing snoring and daytime sleepiness.

Ethics

Ethics Committee Approval: This study was conducted as a retrospective study following the approval of Manisa Celal Bayar University Faculty of Medicine Clinical Research Ethics Committee (approval number: 307, date: 25.07.2022).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ü.Ö., A.K.A., Concept: A.K.A., H.Y., Design: A.K.A., Data Collection or Processing: Ü.Ö., Analysis or Interpretation: A.K.A., M.B., H.Y., Literature Search: Ü.Ö., M.B., Writing: Ü.Ö., A.K.A., M.B., H.Y.

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Supplementary Table 1. Treatments used by PrS-E patients before and after treating SBD	
ASDs used before SBD treatment in PrS-E group	ASDs used after SBD treatment in PrS-E group
VPA 2x1000 mg, TPM 2x100 mg	TPM 2x100 mg
CBZ 2x400 mg	CBZ 2x200 mg
VPA 2x1000 mg	VPA 2x1000 mg
VPA 2x1000 mg	VPA 2x1000 mg
CBZ 2x400 mg, LEV 2x1000 mg	LEV 2x500 mg
CBZ 2x400 mg	CBZ 2x200 mg
VPA 2x1000 mg	VPA 2x1000 mg
VPA 2x1000 mg, LTG 2x100 mg	VPA 2x500 mg, LTG 2x100 mg
LEV 2x1000 mg	LEV 2x1000 mg
VPA 2x1000 mg	VPA 2x1000 mg
LEV 2x1000 mg, OXC 2x600 mg, PHN 3x100 mg, CBZ 2x400 mg	LEV 2x1000 mg, OXC 2x600 mg
CBZ 2x400 mg, VPA 2x1000 mg	CBZ 2x400 mg
CBZ 2x400 mg	CBZ 2x200 mg
CBZ 2x400 mg, LEV 2x1000 mg, PRG 2x300 mg	LEV 2x500 mg
LTG 2x100 mg	LTG 2x100 mg
PHN 3x100 mg, OXC 2x600 mg	PHN 3x100 mg, OXZ 2x600 mg
VPA 2x1000 mg	VPA 2x1000 mg
LEV 2x100 mg, CBZ 2x400 mg	LEV 2x500 mg
LTG 2x100 mg, CBZ 2x400 mg	LTG 2x100 mg
CBZ 2x400 mg	CBZ 2x400 mg
VPA 2x1000 mg	VPA 2x1000 mg
LEV 2x1000 mg	LEV 2x1000 mg
LEV 2x1000 mg, PHN: 3x100 mg, VPA 2x500 mg	LEV 2x1000 mg
VPA 2x1000 mg	VPA 2x1000 mg
LTG 2x100 mg	LTG 2x100 mg
CBZ 2x400 mg, LEV 2x1000 mg	LEV 2x500 mg
VPA 2x1000 mg, LEV 2x1000 mg	LEV 2x500 mg
LEV 2x1000 mg	LEV 2x1000 mg

PrS-E: Patients with primary snoring and epilepsy, SBD: Sleep-related breathing disorders, ASD: Antiseizure drug, VPA: Valproate, TPM: Topiramate, CBZ: Carbamazepine, LEV: Leviratracetam, LTG: Lamotrigine, OXC: Oxcarbazepine, PHN: Phenytoin, PRG: Pregabalin

Supplementary Table 2. Treatments used by severe OSAS-E patients before and after treating SBD	
ASDs used before SBD treatment in OSAS-E group	ASDs used after SBD treatment in OSAS-E group
VPA 2x1000 mg	VPA 2x500 mg
LEV 2x1000, VPA 2x500 mg, LCZ 2x100 mg, PHN 3x100 mg	LEV 2x500 mg, VPA 2x500 mg, LCZ 2x100 mg
LEV 2x1000 mg, VPA 2x500 mg, PHN 3x100 mg	LEV 2x1000 mg, VPA 2x500 mg
LEV 2x1000 mg, CBZ 2x400 mg	LEV 2x1000 mg, CBZ 2x400 mg
TPM 2x100 mg	TPM 2x100 mg
VPA 2x1000 mg	VPA 2x1000 mg
LEV 2x1000 mg	LEV 2x1000 mg
LEV 2x1500 mg, LTG 2x100 mg, CBZ 2x400 mg, VPA 2x500 mg	LEV 2x1000 mg, LTG 2x100 mg
LEV 2x1000 mg, CBZ 2x400 mg	LEV 2x1000 mg
CBZ 2x400 mg, LEV 2x1000 mg, PHN 3x100 mg	CBZ 2x400 mg, LEV 2x1000 mg, PHN 3x100 mg
TPM 2x100 mg, LEV 2x1000 mg, VPA 2x1000 mg	TPM 2x100 mg, LEV 2x1000 mg
LEV 2x1500 mg, VPA 2x1000 mg	LEV 2x1500 mg, VPA 2x1000 mg
VPA 2x1000 mg, LEV 2x1500 mg	VPA 2x1000 mg, LEV 2x1500 mg
LEV 2x1500 mg, CBZ 2x400 mg, VPA 2x500 mg	LEV 2x1500 mg, CBZ 2x400 mg
VPA 2x1000 mg	VPA 2x1000 mg
TPM 2x100 mg, VPA 2x1000 mg, LEV 2x1000 mg	TPM 2x100 mg, VPA 2x500 mg, LEV 2x1000 mg
PHN 3x100 mg, CBZ 2x400 mg	PHN 3x100 mg, CBZ 2x400 mg
VPA 2x1000 mg	VPA 2x1000 mg
PHN 3x100 mg	PHN 3x100 mg
TPM 2x100 mg	TPM 2x100 mg
PHN 3x100 mg, LEV 2x1000 mg	PHN 3x100 mg, LEV 2x1000 mg
LEV 2x1000 mg, CBZ 2x400 mg	LEV 2x1000 mg, CBZ 2x400 mg
LEV 2x1000 mg	LEV 2x1000 mg
CBZ 2x400 mg	CBZ 2x400 mg
LEV 2x1000 mg, LTG 2x100 mg	LEV 2x1000 mg, LTG 2x100 mg
LEV 2x1000 mg, LTG 2x100 mg, VPA 2x500 mg	LEV 2x500 mg
VPA 2x1000 mg	VPA 2x500 mg
VPA 2x1000 mg	VPA 2x500 mg

OSAS-E: Obstructive sleep apnea syndrome-epilepsy, SBD: Sleep-related breathing disorders, ASD: Antiseizure drug, VPA: Valproate, LEV: Levitiracetam, LCZ: Lacosamide, PHN: Phenytoin, CBZ: Carbamazepine, TPM: Topiramate, LTG: Lamotrigine



Relationship of Sleep Quality with Health-related Quality of Life in Type 2 Diabetic Patients of Twin Cities of Pakistan

Pakistan'ın İkiz Şehirlerindeki Tip 2 Diyabetik Hastalarda Uyku Kalitesinin Sağlıkla İlgili Yaşam Kalitesi ile İlişkisi

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Abstract

Objective: To determine the relationship of sleep quality with health-related quality of life (HRQOL). To find frequency of restless leg syndrome (RLS), and to compare sleep quality and HRQOL with or without RLS in diabetic patients.

Materials and Methods: This analytical cross-sectional study was conducted in twin cities of Pakistan from February 2023 to June 2023. A total of 377 participants of both genders aged 30 to 60 years having type 2 diabetes mellitus diagnosed in the last 5 years were selected using a non-probability convenience sampling technique whereas participants with any cardiac disease and uncontrolled hypertension were excluded. To determine sleep quality, HRQOL, and RLS, the European Quality of Life-5 Dimension-5 Levels, Pittsburgh Sleep Quality Index, and Cambridge-Hopkins RLS short-form 2 diagnostic questionnaires were used, respectively.

Results: Sleep quality showed significant relation with HRQOL ($p<0.01$, $r=-0.422$) in type 2 diabetic patients. 10.6% of the participants reported a definite RLS. Significant difference ($p=0.001$) was found in sleep quality and HRQOL with RLS as compared to those having no RLS.

Conclusion: The study concludes that there is a relationship of sleep quality with HRQOL in patients with type 2 diabetes. Further, patients with type 2 diabetes having RLS have poor sleep quality and HRQOL in comparison of the participants without RLS.

Keywords: Pakistan, quality of life, restless leg syndrome, sleep quality, sleep initiation and maintenance, type 2 diabetes mellitus

Öz

Amaç: Uyku kalitesinin sağlıkla ilişkili yaşam kalitesi (HRQOL) ile ilişkisini belirlemek. Diyabetik hastalarda huzursuz bacak sendromunun (HBS) sıklığını bulmak ve HBS olan ve olmayan uyku kalitesi ve sağlıkla ilişkili yaşam kalitesini karşılaştırmak.

Gereç ve Yöntem: Bu analitik kesitsel çalışma, Şubat 2023 ile Haziran 2023 arasında Pakistan'ın ikiz şehirlerinde gerçekleştirildi. Son 5 yılda tip 2 diyabet tanısı alan, 30 ila 60 yaşları arasındaki her iki cinsiyetten toplam 377 katılımcı, bir anket kullanılarak seçildi. Olasılığa dayalı olmayan kolayda örnekleme tekniği ile herhangi bir kalp hastalığı ve kontrolsüz hipertansiyonu olan katılımcılar hariç tutulmuştur. Uyku kalitesini, HRQOL ve HBS, Avrupa Yaşam Kalitesi-5 Boyut-5 Düzeylerini Pittsburgh Uyku Kalitesi İndeksini ve Cambridge-Hopkins'i belirlemek için. HBS kısa form 2 tanılmal anketleri sırasıyla kullanıldı.

Bulgular: Tip 2 diyabet hastalarında HRQOL arasında anlamlı ilişki olduğu görüldü ($p<0,01$, $r=-0,422$). Katılımcıların %10,6'sı kesin bir HBS bildirdi. HBS olanlarda, HBS olmayanlara göre uyku kalitesi ve sağlıkla ilişkili yaşam kalitesi açısından anlamlı fark ($p=0,001$) bulundu.

Sonuç: Çalışma, tip 2 diyabetli hastalarda uyku kalitesi ile sağlıkla ilişkili yaşam kalitesi arasında bir ilişki olduğu sonucuna varmıştır. Ayrıca HBS olan tip 2 diyabetli hastaların uyku kalitesi ve sağlıkla ilgili yaşam kaliteleri, HBS olmayan katılımcılarla karşılaştırıldığında daha düşüktür.

Anahtar Kelimeler: Pakistan, yaşam kalitesi, huzursuz bacak sendromu, uyku kalitesi, uykunun başlatılması ve sürdürülmesi, tip 2 diyabet

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Introduction

Diabetes mellitus (DM), a chronic metabolic disorder marked by high blood sugar levels due to inadequate insulin production or resistance, affects millions globally, and is among one of the most common non-communicable diseases.^{1,2} Among its types, type 2 DM (T2DM) is the most common, making up 90-95% of all cases and increasingly affecting younger populations.³ Factors like age, family history, obesity, physical inactivity, hypertension, gestational diabetes, smoking, and unhealthy diets are well-known contributors with recent research also pointing to sleep disturbances, chemical exposure and high iron levels as emerging risks.^{4,5}

The complications of T2DM are extensive, affecting multiple organ systems and including cardiovascular disease, nerve damage, kidney damage, eye damage, infections, and foot problems.⁶ The International Diabetes Federation estimates that 536.6 million adults worldwide have diabetes, with this number expected to rise to 643 million by 2030 and 783 million by 2045.⁶ In Pakistan, the prevalence is particularly high, with 33 million individuals- 26.7% of the population-affected, driven by sedentary lifestyles, unhealthy diets, genetic factors, and urbanization.⁷

Sleep quality, essential for physical and mental health, is significantly impacted in individuals with T2DM. Poor sleep is linked to a host of adverse outcomes, including depression, anxiety, obesity, and cardiovascular disease.⁸ Restless legs syndrome (RLS), a major sleep disorder, affected 11% of the global population in 2017 and it has drastically increased to 17% in last 3 years.^{9,10} Whereas, prevalence was notably higher (23.6%) in 2017 and now it has raised to 28% in Pakistan highlighting the need for focused attention on sleep disorders in this group.^{10,11}

Health-related quality of life (HRQOL) is a critical measure that looks at how diseases affect an individual's physical, social, and mental well-being. T2DM greatly impacts HRQOL, showing up as physical symptoms, emotional distress, social isolation, and financial strain.¹² Previous studies have consistently shown that T2DM patients have lower HRQOL scores, particularly in areas related to physical performance, functional impairments, and overall well-being.¹³ Additionally, poor sleep quality makes these effects worse, leading to higher levels of depression and anxiety, insulin resistance, and inflammation.^{14,15}

Pakistan has a high prevalence of T2DM, with substantial literature on T2DM and HRQOL. However, there is limited research on the relationship between sleep quality and HRQOL in individuals with T2DM. This study focuses on participants with diabetes for up to 5 years, minimizing the impact of diabetes-related complications on sleep and HRQOL. While various sleep disorder affect sleep, RLS is a significant concern. Though some studies have examined RLS prevalence in different disease populations, there is limited research on its prevalence in the T2DM population. Moreover, the literature lacks studies comparing sleep quality and HRQOL between diabetic patients with and without RLS, highlighting a significant gap in existing research globally.

This study aimed to fill the gap in existing research by exploring the relationship between sleep quality and HRQOL in T2DM patients in Pakistan, with a special focus on those with and without RLS. Secondly, the study findings can enhance the existing knowledge on the impact of sleep quality on HRQOL, particularly in context of T2DM in Pakistan.

Materials and Methods

This analytical cross-sectional study was conducted from February to July 2023, from hospitals of Rawalpindi and Islamabad (Pakistan Institute of Medical Sciences, Smile Dental Hospitals Care Clinic, Abid Hospital Islamabad and Friends Hospital). Sample size was 377 for our primary objective, which was calculated using the Rao software with a 95% confidence interval, 5% margin of error, response rate of 50%, and a population of 20,000. In our study, 40 participants were diagnosed with RLS. So to achieve our secondary objective, 40 participants without RLS were selected by using random sampling method (every 6th interval) to equalize the groups for the comparison of sleep quality and HRQOL in participants with and without RLS. Participants of both gender with age range of 30 to 60 years and had been diagnosed with T2DM within the last 1 to 5 years were included in the study. Whereas, participants who had any heart disease, chronic kidney disease, liver disease, active thyroid disorder, uncontrolled hypertension, diagnosed depression, obstructive sleep apnea, recent trauma, hospital admission within the past 3 months, were also excluded from the study.

A self-structured questionnaire was used to gather basic demographic data of each participant that included age, gender, marital status, occupation, body mass index (BMI), past medical history, comorbidity and the duration of diabetes.

The European Quality of Life-5 Dimension-5 Levels (EQ-5D-5L) questionnaire was used and has shown strong validity and reliability with a Cronbach's alpha coefficient of 0.87 in assessing HRQOL across a range of demographics and different health conditions.¹⁶ It is a tool that enabled a quantitative expression of a person's values and preferences in terms of their general state of health. The EQ-5D-5L uses a five-digit code to score an individual's chosen severity level for each of the five dimensions. It provided a descriptive profile of health status and converted into an index score ranging from- 0.594 to 1. The index score represented the individual's health state, with 1 indicating full health and 0 representing a state worse than death.¹⁷ Another tool used was Pittsburgh Sleep Quality Index (PSQI) to determine sleep quality created by Buysse. It is a standardized self-administered questionnaire for evaluating overall sleep quality over one month. PSQI is a very reliable tool with Cronbach's alpha coefficient value of 0.72 and demonstrated to have good validity.¹⁸ PSQI scores of 7 components range from 0 to 3. The global score, obtained by summing component scores, ranges from 0 to 21, with higher scores indicating poorer sleep quality and lower global scores indicating better sleep quality.¹⁸

Cambridge Hopkins RLS short-form 2 diagnostic (CH-RLSQ13) is a diagnostic questionnaire which has been proved to be

a valid and reliable tool for diagnosing RLS. The CH-RLSQ13 divides subjects into four categories, definite RLS, probable, uncertain and no RLS. According to a study, this tool has a sensitivity and specificity of 87.2% and 94.4%, respectively.¹⁹ After screening 500 participants, we took data from 377 participants falling in our inclusion criteria, prior to data collection participants signed an informed consent. The Institutional Review Board and Ethical Committee IRB & EC (approval number: #0356-22, date: 24.12.2022) of Shifa International Hospital, Islamabad Pakistan have authorized this study.

Statistical Analysis

The statistical package of social sciences version 23 was used to analyze the data of 377 participants. For descriptive analysis, the mean and standard deviation (SD) of age, BMI, and duration of diabetes, whereas frequency of gender, BMI categories, marital status, occupation, and RLS were calculated and shown in the form of tables, pie, and bar charts. The relationship of sleep quality with HRQOL was determined using Pearson correlation. The Independent-T test was used to determine the HRQOL and sleep in T2DM with and without RLS

Results

The study sample consisted of total 377 participants, out of which males were 224 (59%) and females were 153 (40%). The mean and SD of age of the participants was 48.77±7.175 (years). The majority of participants were in the healthy category of BMI, 219 (58.1%) followed by overweight 146 (38.7%). The mean and SD of BMI and duration of diabetes were 24.67±2.59 (Kg/m²) and 3.73±1.07 (years) respectively. In the total sample, laborers were 32 (9%), businessman 66 (17%), housewives 124 (32%) whereas majority of the participants had other occupations 139 (36.9%). A total of 40 participants, accounting for 10.6% of the sample, reported a definite RLS, details of which is given in (Figure 1). Total males and females in Group A (RLS) and Group B (no RLS) were 16 (40%), 24 (60%) and 26 (65%), 14 (35%) respectively. Demographic details of Group A and Group B is given in (Table 1).

Sleep quality showed a negative moderate and significant

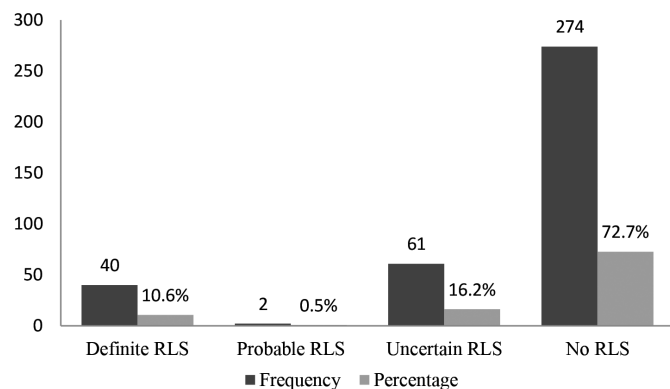


Figure 1. Frequency (%) of RLS of the participants

RLS: Restless leg syndrome

relation ($r=-0.422$, $p<0.01$) with HRQOL in T2DM patients (Table 2). A sample of 80 participants with 40 in each group RLS (Group A) and no RLS (Group B) was assessed further for group comparison of sleep quality and HRQL of diabetes patients with and without RLS. There was a significant difference ($p=0.001$) in sleep quality and HRQOL of T2DM patients with RLS as compared to those having no RLS (Table 3).

Discussion

The present study showed an inverse and significant relationship of sleep quality with HRQOL in T2DM patients.

Our findings were supported by the study conducted in China by Peian Lou et al.²⁰ in 2015 and North India by MD Azharuddin²¹, both studies concluded that poor sleep is common in T2DM population which was linked to reduced HRQL.²⁰ Furthermore, poor sleep quality has adverse effect on various aspects of HRQOL, specifically the functional abilities in daily activities.^{21,22} In addition, the results are also consistent with the cross-sectional study by Chasens and Luyster²² luyster conducted in 2016 focusing on sleep disturbances and their effect on QOL in diabetic patients. RLS was one of the major sleep-disturbance causing factors discussed in this study. They reported that sleep problems such as insomnia, RLS and poor sleep quality, are all associated with lower QOL in those with diabetes.²²

People with diabetes experience poor sleep quality in response to lifelong and comprehensive self-management. Poor sleep leads to tiredness, decrease energy, poor diet and glycemic control, weakened immune system and insufficient diabetes

Table 1. Demographic details of the participants

Variables	Group A (RLS)	Group B (no RLS)
	Mean ± SD	
Age (years)	53.82±5.21	48.62±6.65
BMI (Kg/m ²)	24.11±2.98	24.92±2.84
Duration of diabetes (years)	2.98±1.35	3.7±1.07

BMI: Body mass index, RLS: Restless leg syndrome, SD: Standard deviation

Table 2. Correlation of sleep quality with HRQOL in diabetic patients

Variables	Coefficient of correlation (R value)	P
Sleep quality HRQOL	-0.422	<0.01**

**Indicates significant correlation (p-value <0.01).
HRQOL: Quality with health-related quality of life

Table 3. Comparison of sleep quality and HRQOL with or without RLS in diabetic patients

Variables	Group A (RLS)	Group B (No RLS)	p
	Mean ± SD		
Sleep quality	9.35±3.23	6.9±2.83	0.001***
HRQOL	0.5±0.16	0.67±0.26	0.001***

***Indicates significant difference (p-value <0.001).
RLS: Restless leg syndrome, HRQOL: Quality with health-related quality of life

self-care. All these factors taken together produce a vicious cycle that might be the reason of decrease quality of life with poor sleep quality in diabetes patient.²³

The current study reported that RLS had a prevalence of 10.6% among participants diagnosed with T2DM which is supported by Shin-Ichi Harashima cross sectional study on Japanese diabetic population which reported 8% prevalence of RLS.²⁴

RLS may affect anyone, it has been found to be more common in those who have certain underlying health issues, such as diabetes. The link between diabetes and RLS is not entirely understood, however various studies have found a greater frequency of RLS among diabetics than in the general population.²⁵ The exact mechanisms relating the two disorders are unknown, however some studies suggest that diabetes-related nerve injury, poor circulation, or changes in neurotransmitter levels may contribute to the development or worsening of RLS symptoms.²⁵

A Turkish cross-sectional study in 2019 reported higher prevalence (28.3%) of RLS in patients with T2DM in comparison to our findings. The possible explanation of contradictory findings between the studies might be attributed to various factors such as geographical location, genetic predisposition and environmental variables. Further sample inclusion criteria of wide age group (18-80 years) in the previous study may have contributed to the variations. Additionally, the longer duration of diabetes in the Turkish study may have impacted the progression and severity of RLS symptoms, potentially leading to a higher prevalence.²⁵

Furthermore, contradictory to our study results, higher prevalence of 55.8% was reported in a study conducted in Pakistan in 2015. The smaller sample size (n=120) and different questionnaire for RLS diagnosis International RLS Study Group criteria, in the previous study might be the reason for conflicting results.²⁶

Our study further concluded that T2DM patients with RLS experienced significantly poorer sleep quality and HRQOL as compared to participants without RLS. Our results are in line with cross sectional study conducted on 210 Irani²¹ an diabetic participant.²⁷ Furthermore, the current study results are also consistent with a study conducted in India in 2020 by Thara Pinheiro et al.¹¹ that reported that diabetic patients who also had RLS had considerably poorer sleep quality and HRQOL in comparison to those who did not have RLS.¹¹

The sleep quality of participants in our study was subjectively assessed, in future studies polysomnography could be used for more accurate results. Further Physical level of T2DM patients were not assessed in this study. Seminars should be conducted on increasing awareness about the significance of better sleep quality in individuals with T2DM.

Conclusion

The current study concludes a negative and moderate relationship of sleep quality with HRQOL in T2DM patients. 10.6% prevalence of RLS was found in patients with diabetes. Further, diabetic patients with RLS experienced poor sleep

quality and decrease HRQOL as compared to participants without RLS.

Ethics

Ethics Committee Approval: The Institutional Review Board and Ethical Committee IRB & EC (approval number: #0356-22, date: 24.12.2022) of Shifa International Hospital, Islamabad Pakistan have authorized this study.

Informed Consent: An informed consent form was signed by each participant prior to the enrollment in the study. Each participant received a comprehensive explanation of the entire procedure and participation in the study was entirely voluntary.

Footnotes

Authorship Contributions

Concept: H.F., Design: N.B., Data Collection or Processing: N.B., J.S., Z.M., Analysis or Interpretation: H.F., N.B., T.A., Literature Search: J.S., Z.M., Writing: H.F., T.A., I.I.

Conflict of Interest: No conflict of interest was declared by the authors.

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Evaluation of the Relationship Between Sleep Quality and Occupational Accidents in Nurses

Hemşirelerde Uyku Kalitesinin ve İş Kazalarıyla İlişkisinin Değerlendirilmesi

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Abstract

Objective: The aim of this study was to evaluate sleep quality in nurses and to investigate the relationship between poor sleep quality and occupational accidents.

Materials and Methods: This cross-sectional study was conducted with nurses (n=164) who had been working in different clinics of a university hospital for at least one year and who had not previously been diagnosed with sleep disorders and who agreed to participate in the research. In the data collection form, sociodemographic characteristics, medical history, information about sleep and working conditions, and hospital employee health and safety unit records were questioned. Pittsburgh Sleep Quality Index was used to evaluate sleep quality.

Results: It was found that 77.4% of nurses had poor sleep quality. It was determined that 66.9% of nurses with poor sleep quality and 45.9% of nurses with good sleep quality worked day and night in shifts. It was observed that 38% of the nurses who participated in the study had an occupational accident in the last year, and it was found that all occupational accidents occurred in the group with impaired sleep quality and a significant difference was found between the two groups in terms of the frequency of occupational accidents.

Conclusion: It was found that nurses had poor sleep quality. Individuals with impaired sleep quality have a high frequency of working alternating day and night shifts and continuous night shifts.

Keywords: Shift work, sleep quality, occupational accidents, nurses

Öz

Amaç: Bu çalışmada hemşirelerde uyku kalitesinin değerlendirilmesi ve kötü uyku kalitesinin iş kazalarıyla ilişkisinin araştırılması amaçlanmıştır.

Gereç ve Yöntem: Kesitsel tipte olan bu araştırma bir üniversite hastanesinin farklı kliniklerinde en az bir yıldır çalışan ve araştırmaya katılmayı kabul eden, önceden uyku bozukluğu tanısı almamış hemşireler (n=164) ile yürütülmüştür. Veri toplama formunda sosyodemografik özellikler, tıbbi geçmişleri, uyku ve çalışma koşullarına ilişkin bilgileri, hastane çalışan sağlığı ve güvenliği birimi kayıtları sorgulanmıştır. Uyku kalitesi değerlendirilebilmesi için Pittsburgh Uyku Kalitesi İndeksi kullanılmıştır.

Bulgular: Hemşirelerin uyku kalitesinin %77,4'ünün kötü olduğu saptanmıştır. Uyku kalitesi kötü olan hemşirelerin %66,9'unun, iyi olanların ise %45,9'unun gece gündüz vardiya düzeninde çalıştığı saptanmıştır. Çalışmaya katılan hemşirelerin %38'inin son bir yıl içerisinde iş kazası geçirdiği gözlenmiş olup, tüm iş kazalarının uyku kalitesi bozulmuş olan grupta gerçekleştiği bulunmuş ve iki grup arasında iş kazası sıklığı açısından anlamlı farklılık saptanmıştır.

Sonuç: Hemşirelerin uyku kalitesinin kötü olduğu saptanmıştır. Bozulmuş uyku kalitesi olan bireylerde gece-gündüz dönüşümlü ve sürekli gece vardiyasında çalışma sıklığı yüksektir.

Anahtar Kelimeler: Vardiyalı çalışma, uyku kalitesi, iş kazası, hemşireler

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Introduction

Shift work is designed to maintain continuous operations by dividing labor into consecutive shifts. It is becoming increasingly prevalent in modern society, with shift system workers comprising approximately 21% of the workforce in the European Union (EU).^{1,2} In Türkiye, the prevalence of shift work was reported as 8% in a 2003 report on the working conditions of EU candidate countries.³ More recent labor force statistics from the Turkish Statistical Institute indicate that as of 2020, 13.6% of employees worked in a shift system, with 4.7% working primarily night shifts.⁴

A 24-hour work schedule typically consists of at least three shifts. However, in healthcare settings, shift schedules are often structured into two shifts within a 24-hour period.⁵ These irregular work hours disrupt the circadian rhythm, which regulates the 24-hour sleep-wake cycle by promoting sleep at night and wakefulness during the day.⁶ The disruption is particularly pronounced during night shifts and early morning transitions, as these work hours coincide with the body's natural need for sleep.

Research has shown that shift work is a significant risk factor for sleep disorders among nurses, who frequently work long hours and night shifts.⁷ For instance, a study by Takahashi et al.⁸ on 775 nurses working in shifts and providing home care services found that shift workers had a higher prevalence of sleep disorders compared to the control group. These nurses experienced more frequent insomnia symptoms, had greater difficulty falling and staying asleep, and reported lower overall sleep quality.

Insufficient or poor-quality sleep caused by shift work is associated with cognitive impairments, including memory deficits, difficulty concentrating, and reduced decision-making ability. These impairments can negatively affect psychomotor performance, increasing the likelihood of occupational accidents and errors. Experimental research has demonstrated that impaired sleep quality significantly reduces cognitive function, thereby elevating the risk of occupational accidents.⁹ Indeed, studies indicate that the risk of occupational accidents is disproportionately higher during night shifts compared to evening shifts. Longer shift duration has also been associated with a greater likelihood of accidents.^{10,11}

Based on the hypothesis that shift workers experience lower sleep quality, which in turn increases the risk of occupational accidents, this study aimed to assess sleep quality in nurses working in a shift system and investigate the relationship between poor sleep quality and occupational accidents.

Materials and Methods

Study Population

This cross-sectional study was conducted among nurses working in various clinics of a university hospital. A non-probability sampling approach was employed, where participants were selected based on specific eligibility criteria. Nurses who had been employed at the institution for a minimum of one year, provided informed consent, and had no prior diagnosis of sleep

disorders were eligible for inclusion (n=168). Exclusion criteria included absence from work for the past year, pregnancy, having a child under the age of 2 years, being on leave during the study, refusal to participate, and missing data.

Participants were initially screened for any previous diagnoses of sleep-related disorders. Their daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS), and additional sleep-related parameters were evaluated using the Pittsburgh Sleep Quality Index (PSQI).^{12,13} Based on these assessments, no participants were found to have sleep disorders such as sleep apnea syndrome, habitual snoring, or sleep movement disorders. A total of 164 participants with complete survey data were included in the final statistical analysis.

The participants were informed about data security and assured that the collected information would not be shared with third parties. Informed verbal consent was first obtained, followed by written consent. The completion of the survey forms took approximately 15 minutes.

Variables

This study examined two dependent variables: sleep quality and the frequency of occupational accidents within the past year. The International Labour Organization defines an occupational accident as an event that results in a fatal or non-fatal injury during work-related activities or while performing tasks associated with work. This also includes accidents that occur while commuting to or from work.¹ For this study, information on occupational accidents was collected from the nurses, focusing on incidents that occurred within the past month in the hospital setting (e.g., injuries caused by sharp objects, needle sticks, falls, slips, bruises, poisoning, burns, and chemical exposure to mucous membranes).

The independent variables considered for their potential impact on the dependent variables included demographic characteristics, medication use, the presence of depression, clinical department, shift type, total years of work experience, hours worked per day, and the number of nights worked in the past month. Sleep quality (assessed using the PSQI) and the frequency of occupational accidents over the past year were also included as independent variables in the analysis (Table 1).

Data Collection Tools

Sociodemographic characteristics, medical history, and information related to sleep and work conditions were assessed using a 23-item questionnaire developed based on records from the hospital's occupational safety and health unit and the relevant literature. Sleep quality was assessed using the PSQI, and daytime sleepiness was evaluated using the ESS.

Pittsburgh Sleep Quality Index

The PSQI is a self-report measure designed to assess sleep quality over the past month. It includes parameters such as subjective sleep quality, sleep latency, sleep duration, sleep disturbances, use of sleep medication, and daytime dysfunction. The scale consists of 19 self-report questions and 5 additional questions for bed partners, but only the self-report questions

Sleep quality		Total (n=164)	Good (n=37)	Poor (n=127)	X ²	p
		n	n (%)*	n (%)*		
Sex	Female	144	34 (23.6)	110 (76.3)	0.745	0.292
	Male	20	3 (15.0)	17 (85.0)		
Marital status	Single	64	16 (25.0)	48 (75.0)	0.357	0.340
	Married	100	21 (21.0)	79 (79.0)		
Parental status	No	96	21 (21.8)	75 (78.2)	0.062	0.474
	Yes	68	16 (23.5)	52 (76.5)		
Education level	Undergraduate	138	30 (21.7)	108 (78.3)	0.337	0.362
	Postgraduate	26	7 (26.9)	19 (73.1)		
Department	Internal medicine	70	16 (22.8)	54 (77.2)	0.539	0.970
	Surgical unit	18	5 (27.7)	13 (72.3)		
	Intensive care	31	7 (22.5)	24 (77.5)		
	Emergency service	17	3 (17.6)	14 (82.4)		
	Outpatient	28	6 (21.4)	22 (78.6)		
Shift type	Continuous day	43	16 (37.2)	27 (62.8)	7.334	0.026
	Continuous night	19	4 (21.0)	15 (79.0)		
	Rotating day/night	102	17 (16.6)	85 (83.4)		
Work duration (hours/day)	8	205	26 (12.6)	79 (87.4)	0.809	0.242
	16	59	11 (18.6)	48 (81.4)		
Comorbidities		110	26 (23.6)	84 (76.4)	0.221	0.398
Medication use		61	11 (18.0)	50 (82.0)	1.140	0.192
Depression		35	6 (17.1)	29 (82.9)	0.748	0.387
Difficulty staying awake during shifts		131	28 (21.3)	103 (78.7)	0.525	0.305
Occupational accident		63	0 (0.0)	63 (100.0)	29.803	<0.001
		Mean ± SD	Mean ± SD	Mean ± SD		
Age (years)		34.2±8.2	34.4±9.1	34.2±7.9		0.916
Body mass index (kg/m ²)		24.2±4.0	24.0±4.4	24.3±3.8		0.738
Total work experience (years)		12.5±9.2	12.7±9.8	12.4±9.0		0.849
Night shifts in last month (days)		5.5±4.7	4.86±5.0	5.63±4.6		0.385
Epworth Score		1.5±1.1	1.2±0.7	1.5±1.2		0.048
*Row percentages are given. SD: Standard deviation						

contribute to the total score. A total score of 5 or higher was interpreted as poor sleep quality.¹²

Epworth Sleepiness Scale

The ESS is used to assess daytime sleepiness levels. Respondents are asked to rate their tendency to doze off or fall asleep during various daily activities. The ESS consists of 8 questions, each rated on a scale from 0 to 3, for a total score ranging from 0 to 24. A score of 11 or higher indicates clinically significant daytime sleepiness.¹³

Ethical Approval

Ethical approval for the study was obtained from the Ege University Ethics Committee (approval number: 269, date: 07.10.2016).

Statistical Analysis

Data were analyzed using SPSS Statistics version 22.0 for Windows (IBM Corp., Armonk, NY). Descriptive statistics, including frequencies, percentages, means, and standard deviations, were used to summarize the data. Chi-square tests

were used to examine differences between categorical variables related to sleep quality and the occurrence of accidents over the past year. Differences in continuous variables based on sleep quality and accident occurrence were analyzed using independent samples t-tests. Logistic regression analysis was employed to identify factors influencing sleep quality and the occurrence of accidents.

Results

Evaluation of Sleep Quality

This study included 164 nurses working in a shift system [144 females and 20 males, mean age 34.2 ± 8.1 years, mean body mass index (BMI) 24.2 ± 3.9 kg/m²]. According to the PSQI, 77.4% of the nurses reported poor sleep quality. When comparing nurses with good versus poor sleep quality, no statistically significant differences were observed in terms of age, gender, BMI, comorbidities, medication use, marital status, parental status, or education level ($p > 0.05$) (Table 1). Among the nurses, 42.7% worked in internal medicine clinics, followed by intensive care units (18.9%), outpatient clinics (17.1%), surgical clinics (10.9%), and emergency departments (10.4%). No significant differences in sleep quality were found across these clinical settings. A total of 62.2% of the nurses worked a rotating day/night shift schedule, 26.2% followed a continuous daytime schedule, and 11.6% worked continuous night shift. Of the nurses with poor sleep quality, 66.9% worked a rotating day/night shift schedule, compared to 45.9% of those with good sleep quality ($p = 0.026$). No significant differences in sleep quality were found between the groups based on hours worked per day, number of night shifts worked in the past month, years of work experience, or difficulty staying awake during shifts (Table 1).

In regression analysis, working the day shift continuously was associated with 64% lower odds of poor sleep quality than working a rotating shift schedule [odds ratio (OR): 0.36, 95% confidence interval (CI): 0.15-0.82, $p = 0.015$] (Table 2).

Evaluation of Occupational Accidents

We determined that 38% of the participating nurses had experienced an occupational accident in the past year. Notably, 75% of these incidents involved needlestick injuries, a significant and concerning issue within the healthcare profession. Other types of work accidents included cuts from ampoules or surgical instruments (43%), falls or slips (42%), exposure to blood and

bodily fluids (25%), exposure to chemical fluids (21%), and poisoning (15%).

There was a strong correlation between impaired sleep quality and occupational accidents, with all accidents occurring among nurses who reported poor sleep quality. We also observed that the frequency of medication use was higher among nurses who had not experienced an occupational accident (52.4%) compared to those with a history of occupational accident (47.6%, $p = 0.047$) (Table 3).

The prevalence of depression was also significantly higher among nurses who had experienced an occupational accident compared to those who had not (75.8% vs. 34.2%, $p < 0.001$). No other statistical differences were observed between the two groups in terms of demographic variables, years of work experience, clinical department, hours worked per day, or number of night shifts worked in the past month ($p > 0.05$).

Among the factors that may influence occupational accidents, the results of multivariate regression analysis indicated that a higher PSQI score (indicating worse sleep quality) was associated with 33% higher odds of occupational accidents (OR: 1.33, 95% CI: 1.20-1.49, $p < 0.001$). Conversely, the absence of depression was associated with 78% lower odds of an occupational accident (OR: 0.22, 95% CI: 0.08-0.56, $p = 0.002$). Medication use was not a significant predictor of occupational accidents (Table 4).

Discussion

This study assessed the sleep quality of nurses using the PSQI and investigated its relationship with occupational accidents. We found that 77% of nurses had poor sleep quality, and the most significant factor influencing this was working a rotating day/night shift schedule. Notably, half of the nurses with poor sleep quality had experienced occupational accidents, whereas none of those with good sleep quality had a history of accidents. Furthermore, impaired sleep quality and the presence of depression were associated with an increased risk of occupational accidents.

The Relationship Between Shift Work and Sleep Quality

Disrupted sleep quality among shift-working nurses has been widely reported in observational studies. Di Muzio et al.¹⁴ found that 54.6% of shift workers had a PSQI score above 5, whereas Zhang et al.¹⁵ reported a 72% prevalence of poor sleep quality among nurses. In a study conducted in Türkiye, Çelik et al.¹⁶

	B	Standard error	Odds ratio	95% Confidence interval		p
				Lower	Upper	
Intercept	1.22	0.39	3.41			0.002
Shift type						
Continuous day	-1.01	0.41	0.36	0.15	0.82	0.015
Continuous night	-0.16	0.63	0.84	0.26	3.30	0.79
Rotating day/night ^(ref)	0	-	1	-	-	-
Epworth Score	0.25	0.20	1.28	0.88	1.96	0.209

^{ref}: References value

		Total (n=164)	Without occupational accidents (n=101)	With occupational accidents (n=63)	χ ²	p
		n	n (%)*	n (%)*		
Sex	Female	144	91 (63.1)	53 (36.9)	1.292	0.186
	Male	20	10 (50.0)	10 (50.0)		
Marital status	Single	64	43 (67.1)	21 (32.9)	1.392	0.155
	Married	100	58 (58.0)	42 (42.0)		
Parental status	No	96	54 (56.2)	42 (43.8)	2.786	0.065
	Yes	68	47 (69.1)	21 (30.9)		
Education level	Undergraduate	138	86 (62.3)	52 (37.7)	0.198	0.407
	Postgraduate	26	15 (57.6)	11 (42.4)		
Department	Internal medicine	70	41 (58.5)	29 (41.5)	6.964	0.138
	Surgical unit	18	10 (55.5)	8 (44.5)		
	Intensive care	31	25 (80.6)	6 (19.4)		
	Emergency service	17	11 (64.7)	6 (35.3)		
	Outpatient	28	14 (50.0)	14 (50.0)		
Shift type	Continuous day	43	30 (69.7)	13 (30.3)	1.655	0.437
	Continuous night	19	11 (57.8)	8 (42.2)		
	Rotating day-night	102	60 (58.8)	42 (41.2)		
Work duration (hours/day)	8	205	64 (31.2)	41 (68.8)	0.049	0.480
	16	59	37 (62.7)	22 (37.3)		
Comorbidities		110	65 (59.1)	45 (40.9)	0.879	0.222
Medication use		61	32 (52.4)	29 (47.6)	3.419	0.047
Depression		35	12 (34.2)	23 (75.8)	14.017	<0.001
Difficulty staying awake during shifts		131	79 (60.3)	52 (39.7)	0.451	0.322
		Mean ± SD	Mean ± SD	Mean ± SD		
Age (years)		34.2±8.2	34.1±8.4	34.4±7.9		0.848
Body mass index (kg/m ²)		24.2±4.0	24.1±4.0	24.4±3.9		0.624
Total work experience (years)		12.5±9.2	12.0±9.2	13.2±9.1		0.431
Night shifts in last month (days)		5.5±4.7	5.4±4.8	5.5±4.5		0.887
PSQI total score		8.6±4.2	7.0±3.9	11.0±3.5		<0.001
ESS total score		1.5±1.1	1.3±0.8	1.7±1.4		0.057

*Row percentages are given.
PSQI: Pittsburg Sleep Quality Index, ESS: Epworth Sleepiness Scale, SD: Standard deviation

found that 72% of intensive care nurses had a PSQI score of ≥5. Similarly, Haznedaroğlu et al.¹⁷ determined that 59.2% of pulmonologists had poor sleep quality during the Coronavirus Disease 2019 pandemic. Our study also demonstrated a high prevalence of poor sleep quality among nurses in various branches (77%). Moreover, total PSQI scores in our study (8.57±4.22) were higher than in the general population (5.00±3.37) but comparable to those reported for nurses [median 8.30 (0-20)].^{18,19}

Circadian rhythm disruption caused by shift work has been identified as a primary factor contributing to poor sleep

quality.^{6,8} Previous studies have shown that rotating and night shift schedules lead to sleep disturbances due to misalignment of the endogenous circadian rhythm with the work schedule.²⁰ This misalignment becomes more pronounced in rotating shift schedules, where both sleep and work times change frequently.²⁰

Consistent with these findings, our study showed that nurses working rotating day/night shifts had the highest prevalence of poor sleep quality, followed by those on continuous night shifts.²⁰ In contrast, those working continuous day shifts had significantly better sleep quality. This suggests that irregular

Table 4. Logistic regression analysis for factors associated with occupational accidents

	B	Standard error	Odds ratio	%95 Confidence interval		p
				Lower	Upper	
Intercept	-1.71	0.83	0.18	0.03	0.89	0.04
Total work experience (years)	0.01	0.03	1.01	0.95	1.08	0.58
Night shifts in last month (days)	0.01	0.04	1.04	0.92	1.11	0.69
Medication use						
No	-0.45	0.45	0.63	0.26	1.53	0.31
Yes ^{ref}	0	-	1	-	-	-
Depression						
No	-1.50	0.49	0.22	0.08	0.56	0.002
Yes ^{ref}	0	-	1	-	-	-
Work duration (hours/day)						
8	-0.35	0.45	0.69	0.28	1.71	0.43
16 ^{ref}	0	-	1	1	-	-
PSQI total score	0.28	0.05	1.33	1.20	1.49	<0.001
ESS total score	0.037	0.031	1.037	-0.02	0.09	0.23

PSQI: Pittsburg Sleep Quality Index, ESS: Epworth Sleepiness Scale, ^{ref}: Reference value

or nocturnal work schedules contribute significantly to sleep disturbances. Additionally, the group with poor sleep quality had a higher frequency of continuous night shift work, likely due to inadequate sleep during rest periods, whether on days off or following night shifts. The discrepancy in sleep patterns between workdays and rest days may further exacerbate circadian misalignment, ultimately leading to deteriorating sleep quality.

Although age is known to affect sleep duration and quality, with older individuals typically experiencing shorter and poorer sleep,^{21,22} no significant relationship between age and sleep quality was found in our study. This could be because our study population consisted primarily of younger and middle-aged individuals (ages ranging from 18 to 58 years).

The Relationship Between Shift Work and Occupational Accidents

The occupational accident rate among nurses in our study was 38.4%, higher than the rates reported in similar studies. Sonmez et al.²³ reported an occupational accident rate of 12.4% among shift-working nurses in a university hospital in Türkiye. The discrepancy between these rates could be attributed to the wider range of accidents considered in our study. Westwell et al.²⁴ reported a needlestick injury rate of 12% among shift-working nurses, and this variation may also be due to differences in occupational health and safety measures across countries and different definitions of occupational accidents used in the studies. In our study, half of shift-working nurses with poor sleep quality had experienced occupational accidents, while none of those with good sleep quality had a history of accidents. This suggests that impaired sleep quality significantly increases the risk of occupational accidents, likely due to decreased concentration and cognitive function, which are consistent with findings

in the literature. Recent studies indicate that the type of shift schedule influences accident risk indirectly through sleep quality. Specifically, rotating shift schedules and continuous night shifts are associated with a higher risk of accidents.²⁵ In our study, no significant differences were observed between the groups with and without occupational accidents in terms of their work schedules, which may be due to the insufficient sample size for statistically significant results.

The Relationship Between Depression and Occupational Accidents

The prevalence of depression in shift-working nurses in our study was 21.3%, and depression was identified as a significant factor that increased the risk of occupational accidents. Previous studies support a bidirectional relationship between shift work and mental health disorders like anxiety and depression.²⁵⁻²⁸ Frequent shifts and the changes in shift cycles have been shown to increase the risk of developing depression. Li et al.²⁹ found that shift-related factors such as rotating shifts increased the prevalence of depression and anxiety disorders among shift-working nurses. Additionally, another study indicated that anxiety and depression in individuals at risk of insomnia can contribute to the deterioration of sleep quality.³⁰ Depression impairs decision-making abilities, which can increase the risk of occupational accidents. Weaver et al.²⁶ reported a 21.6% prevalence of depression among healthcare workers and demonstrated that mood disorders were associated with a 63% higher risk of adverse events such as motor vehicle accidents, exposure to infected biological material, and medical errors. Their study also indicated that the combination of sleep and mood disorders tripled this risk. High-quality studies focusing on mood disorders in shift workers could provide further evidence of the causal relationship between depression, sleep disorders, and occupational accidents.

Study Limitations

Due to the cross-sectional design of our study, we could not establish a causal relationship between shift work, impaired sleep quality, and occupational accidents. The study sample was predominantly composed of female nurses, meaning gender-related differences in the effects of shift work on sleep disorders could not be assessed. Additionally, our research was conducted among nurses at a university hospital, which may limit the generalizability of the results to other healthcare settings or populations at different care levels. Another limitation is that we did not use the STOP-Bang questionnaire, which assesses the risk of obstructive sleep apnea (OSA) based on factors such as age, neck circumference, snoring, hypertension, and daytime sleepiness. Including this questionnaire might have provided a more accurate identification of individuals at risk for OSA. Furthermore, the presence of depression was based on self-report, which has inherent limitations. Participants responded according to their own perceptions and memory, which could lead to inaccuracies in diagnosing depression, especially for those who have not sought professional help. This could result in underreporting of depression in the study.

Conclusion

This study examined the relationship between sleep quality and occupational accidents among shift-working nurses. The findings revealed that 77% of the nurses had poor sleep quality, with a higher frequency of rotating day/night shifts among those who reported impaired sleep quality. Conversely, working the day shift continuously was associated with 64% lower odds of poor sleep quality. Furthermore, both poor sleep quality and the presence of depression were identified as significant factors associated with increased risk of occupational accidents. These results underline the need for organizational and behavioral interventions to improve sleep quality among shift-working nurses and for the implementation of strategies to prevent occupational accidents. Future studies with prospective designs should be conducted to better understand the relationship between shift patterns, circadian rhythm disturbances, and sleep quality in the nursing profession.

Ethics

Ethics Committee Approval: Ethical approval for the study was obtained from the Ege University Ethics Committee (approval number: 269, date: 07.10.2016).

Informed Consent: Informed verbal consent was first obtained, followed by written consent.

Footnotes

Authorship Contributions

Concept: N.Ç., Z.N.T., H.K., M.S.T., Ö.K.B., Design: N.Ç., Z.N.T., M.S.T., Ö.K.B., Data Collection or Processing: N.Ç., H.K., Analysis or Interpretation: N.Ç., Z.N.T., M.S.T., Ö.K.B., Literature Search: N.Ç., Z.N.T., H.K., Writing: N.Ç., Z.N.T., M.S.T., Ö.K.B.

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The Relationship Between Parents' Sleep Quality and Chronotype and Children's Sleep Habits

Ebeveynlerin Uyku Kalitesi ve Kronotipi ile Çocukların Uyku Alışkanlıkları Arasındaki İlişki

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Abstract

Objective: Sleep is crucial for children's development, affecting their physical, behavioral, emotional, and cognitive performance. Parents' sleep problems can affect their children's sleep patterns and vice versa. The current study aims to assess parental sleep quality and chronotype and their relationship to their children's sleep habits.

Materials and Methods: This study included 122 healthy children between the ages of 4 and 12. Parents' sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI), chronotype was assessed with the Morningness-Eveningness Questionnaire, and children's sleep habits were measured using the Children's Sleep Habits Questionnaire (CSHQ).

Results: Based on the CSHQ (cut-off score >41), 88.5% of the children were found to have sleep disturbances. According to the PSQI, 33% of the mothers and 41.2% of the fathers were identified as having poor sleep quality. Most mothers and fathers were classified as intermediate chronotypes, followed by morning chronotypes, with evening chronotypes being the least common. Parental PSQI scores were positively correlated with children's overall CSHQ scores ($r=0.192$, $p=0.048$), as well as specific CSHQ subscales: bedtime resistance ($r=0.251$, $p=0.010$), sleep onset delay ($r=0.306$, $p=0.001$), sleep duration ($r=0.213$, $p=0.028$), sleep anxiety ($r=0.195$, $p=0.045$), and night waking ($r=0.210$, $p=0.031$).

Conclusion: These findings suggest that children are at a high risk of having sleep disturbances and that there is a link between parental sleep quality and children's sleep habits. Further research is required to enhance the understanding of the relationship between the sleep health of parents and their children.

Keywords: Children, chronotype, sleep quality, parent, sleep habit

Öz

Amaç: Uyku, çocukların gelişimi açısından kritik öneme sahiptir ve fiziksel, davranışsal, duygusal ve bilişsel performanslarını etkileyebilir. Ebeveynlerdeki uyku sorunları çocuklarının uykusunu etkileyebilir ve bunun tersi de geçerlidir. Bu çalışmanın amacı, ebeveynlerin uyku kalitesi ve kronotiplerini değerlendirmek ve bunların çocuklarının uyku alışkanlıklarıyla olan ilişkilerini incelemektir.

Gereç ve Yöntem: Çalışmaya, 4-12 yaş aralığında 122 sağlıklı çocuk dahil edilmiştir. Ebeveynlerin uyku kalitesi Pittsburgh Uyku Kalitesi İndeksi (PSQI) ile, kronotipleri ise Sabahçılık-Akşamcılık Anketi ile değerlendirilmiştir. Çocukların uyku durumu Çocuk Uyku Alışkanlıkları Anketi (CSHQ) kullanılarak incelenmiştir.

Bulgular: CSHQ'ya göre (kesme puanı >41), çocukların %88,5'inde uyku bozukluğu olduğu tespit edilmiştir. PSQI'ye göre, annelerin %33'ü ve babaların %41,2'si kötü uyku kalitesine sahip olarak tanımlanmıştır. Kronotip açısından bakıldığında, ebeveynlerin çoğunun orta kronotipte yer aldığı, bunu sabahçıl kronotipin izlediği ve akşamcıl kronotipin ise en az yaygın grup olduğu gözlenmiştir. Ebeveynlerin PSQI puanları, çocukların genel CSHQ puanları ($r=0,192$, $p=0,048$) ve spesifik CSHQ alt ölçekleri ile pozitif korelasyon göstermiştir: Yatma zamanı direnci ($r=0,251$, $p=0,010$), uyku başlangıcı gecikmesi ($r=0,306$, $p=0,001$), uyku süresi ($r=0,213$, $p=0,028$), uyku kaygısı ($r=0,195$, $p=0,045$) ve gece uyanması ($r=0,210$, $p=0,031$).

Sonuç: Bu bulgular, çocukların uyku bozuklukları açısından yüksek risk altında olduğunu ve ebeveynlerin uyku kalitesinin çocukların uyku alışkanlıklarıyla ilişkili olduğunu göstermektedir. Ebeveyn ve çocuk uyku sağlığı arasındaki ilişkinin daha iyi anlaşılması için daha fazla araştırma yapılması gerekmektedir.

Anahtar Kelimeler: Çocuk, kronotip, uyku kalitesi, ebeveyn, uyku alışkanlığı

Introduction

Sleep is vital for human health and well-being,¹⁻⁴ influencing physical and mental health, performance, growth, energy conservation, brain function, neural maturation, learning, and

memory.⁵ Its role is especially critical during childhood, affecting development, behavior, and cognitive function. Despite its importance, having insufficient sleep has become a widespread health issue in modern societies.^{4,6} Recent meta-analyses have shown that the global prevalence of sleep disturbances has

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reached alarming levels in children and adolescents at 54% and 34% respectively.^{7,8}

Chronotype refers to individual differences in sleep-wake patterns and biological rhythms across the lifespan. It influences psychological, cognitive, and physiological processes, including mood, endocrine function, cognition, and body temperature.⁹ Chronotypes are typically classified into three groups: morning, evening, and intermediate types.¹⁰ A stable circadian rhythm is essential for maintaining human well-being.^{11,12}

Parents and children influence each other's sleep through shared environmental factors such as culture, living conditions, and socio-economic status, as well as any genetic predispositions affecting sleep patterns. Consequently, sleep problems in parents can affect their children's sleep, and vice versa.¹³ Moreover, unhealthy sleep habits established in childhood may contribute to the development of persistent poor sleep patterns later in life.

Insufficient sleep and daytime sleepiness in children can significantly impact their overall health, academic performance, and social relationships.¹⁴ Therefore, promoting healthy sleep habits from an early age is essential.¹⁵ Pediatric healthcare providers should include sleep health assessments in routine well-child visits, emphasizing that a healthy sleep routine is as important as nutrition, physical activity, and dental hygiene.⁵ Studies show that children's sleep is linked to their parents' sleep patterns.^{5,16-18} To our knowledge, no research has been conducted in Türkiye that compares parents' sleep and chronotype with children's sleep habits. The present study aims to address this gap by investigating the relationship between children's sleep habits and their parents' sleep quality and chronotype.

Materials and Methods

Participants

This study was conducted with a non-probability sampling method among the parents of healthy children between the ages of 4 and 12 who visited the General Pediatrics Outpatient Clinic of Koç University Hospital between November 2023 and June 2024. Ethical approval for this research was secured from the Institutional Review Board of Koç University (approval number: 2023.375.IRB3.168, date: 02.11.2023). The research was performed in adherence to the principles of the Declaration of Helsinki. Participants were required to be healthy children between the ages of 4 and 12, with no prior history of chronic illness, sleep disorders, psychiatric conditions, or regular medication use. Parents were asked to complete three questionnaires that covered socio-demographic information, sleep quality, chronotype, and children's sleep habits. The questionnaires were distributed via an online link (Qualtrics). Before starting the survey, participants were provided with a brief summary of the research, and electronic informed consent was obtained.

Inventories

The socio-demographic form was used to collect data on children's age, weight, height, outdoor activity time, and screen

time, in addition to parents' age and education levels and use of electronic devices. Parents were asked to fill out the following inventories: the Pittsburgh Sleep Quality Index (PSQI) and the Morningness-Eveningness Questionnaire (MEQ). Mothers were asked to complete the Children's Sleep Habit Questionnaire (CSHQ).

The Child Sleep Habit Questionnaire

The CSHQ, designed by Owens et al.,²⁰ consists of 33 items designed to examine children's sleep quality and patterns.¹⁹ Fiş et al.²⁰ conducted a study on validity and reliability for Turkish children. The questionnaire targets children aged 4 to 12 years and is organized into eight subscales, including categories such as bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night wakings, parasomnias, sleep-disordered breathing, and daytime sleepiness. The CSHQ is completed retrospectively by parents to examine the children's sleep habits during the previous week. Each item is assessed using a 3-point scale. A score above 41 indicates the likelihood of a pediatric sleep disorder, with higher scores indicating more significant sleep disturbances.¹⁹ The Cronbach's alpha coefficient for this study was 0.728.

Pittsburgh Sleep Quality Index

The PSQI, originally designed by Buysse et al.²¹ in 1989, was translated and modified for Turkish use by Agargun et al.²² The PSQI is a self-administered scale composed of 19 items designed to examine sleep quality and disturbances experienced during the previous month. It comprises 24 items, with 19 being self-report items and 5 requiring responses from a partner or roommate. The 18 scored items on the scale are grouped into seven components. Each item is assigned a rating between 0 and 3. The cumulative score, obtained by summing the values of all seven components, falls between 0 and 21, with a score greater than 5 signifying 'poor sleep quality'.²² The Cronbach's alpha coefficient of the study was 0.701.

The Morningness-Eveningness Questionnaire

The MEQ was designed by Horne and Ostberg.²³ A Turkish reliability study was performed by Pündük et al.²⁴ in 2005. The self-report scale consists of 19 questions that explore an individual's lifestyle, sleep/wake patterns, and overall performance. The total score from the questionnaire is used to determine an individual's chronotype. Scores ranging from 16 to 41 signify an "evening" chronotype, scores from 42 to 58 represent an "intermediate" chronotype, and scores from 59 to 86 indicate a "morning" chronotype. The Cronbach's alpha coefficient of the study was 0.802.

Statistical Analysis

Analyses were conducted using IBM SPSS Statistics Version 28.0. The normality of the variables was assessed through histogram plots and the Shapiro-Wilk test. To assess relationships between independent categorical variables, the Pearson chi-square or chi-square test was used. The Mann-Whitney U test was utilized to compare two groups of continuous independent variables that did not adhere to a normal distribution. Spearman's correlation was used to analyze associations between non-normally distributed continuous variables.

Results

Socio-demographic Characteristics

The participants were 122 children aged 4 to 12 years from İstanbul, Türkiye. The study included all the children's mothers (100%, n=122) and the majority of the fathers (62.3%, n=76). The median age of the children was 6.0 years, and the gender distribution was nearly equal, with 63 females (51.6%) and 59 males (48.4%). The socio-demographic data of the participants are presented in Table 1.

Parents' Sleep and Chronotype Characteristics

The mean bedtime was 23:37 for mothers and 23:35 for fathers (p=0.080). The mean wake time was 7:18 for mothers and 7:18 for fathers (p=0.664), with mean total sleep time of 7.0 hours for mothers and 6.8 hours for fathers (p=0.177). PSQI scores were 4.0 for mothers and 5.0 for fathers (p=0.486). MEQ scores were also similar, with mothers scoring 53.0 and fathers scoring 52.5 (p=0.626). Regarding sleep quality, 67% of mothers reported good sleep compared to 58.8% of fathers (p=0.275). The sleep measurements of the parents are presented in Table 2.

Children Sleep Characteristics

The mean bedtime was 21:31, with a mean wake time of 7:37. The mean total sleep time was 9.3 hours. The median score on the CSHQ was 49.0, and 88.5% of the children (n=108) had a cutoff score greater than 41, indicating the presence of a sleep

disturbance. Sleep characteristics of the children are presented in Table 3.

Associations Between Children and Parents' Sleep Characteristics

Correlation Analysis

The correlation analysis between parental sleep and chronotype variables and children's sleep habits is presented in Table 4. A weak positive correlation was found between parental PSQI scores and several children's sleep-related variables, including: CSHQ scores (r=0.192, p=0.048), bedtime resistance (r=0.251, p=0.010), sleep onset delay (r=0.306, p=0.001), sleep duration (r=0.213, p=0.028), sleep anxiety (r=0.195, p=0.045), and night waking (r=0.210, p=0.031). Furthermore, a weak negative correlation was observed between PSQI scores and child daytime sleepiness (r=-0.202, p=0.038) (Table 4).

There was also a moderate negative correlation between parental MEQ scores and parental PSQI scores (r=-0.359, p<0.001) and a weak negative correlation between parental MEQ scores and child sleep anxiety (r=-0.210, p=0.020) (Table 4).

No significant correlations were found between CSHQ scores and child age (r=-0.170, p=0.062), child screen time (r=-0.109, p=0.255), or child outdoor activity (r=-0.124, p=0.198).

Children age, years, median (IQR)	6.0 (3.0)
Gender, n (%)	
Female	63 (51.6)
Male	59 (48.4)
BMI, (kg/m ²), median (IQR)	16.0 (3.4)
Maternal age, years, median (IQR)	37.0 (8.0)
Paternal age, years, median (IQR)	40.0 (8.0)
Maternal education level, years, median (IQR)	16.0 (3.0)
Paternal education level, years, median (IQR)	16.0 (1.0)
Marriage duration, median (IQR)	10.0 (5.0)
Marriage status, n (%)	
Marriage	114 (93.4)
Divorced	8 (6.6)
Number of children, n (%)	
1 child	67 (54.9)
2 children	47 (38.5)
≥3 children	8 (6.6)
Which child, n (%)	
1 st child	106 (86.9)
≥2 nd child	16 (13.1)
Maternal screen time, hours, median (IQR)	3.0 (5.0)
Paternal screen time, hours, median (IQR)	4.0 (4.0)
Children's screen time, hours, median (IQR)	2.0 (1.0)
Children's outside activity time, hours, median (IQR)	1.0 (1.0)
IQR: Interquartile range, BMI: Body mass index	

	Maternal (n=122)	Paternal (n=76)	p
Bedtime, mean (SD)	23:37 (01:01)	23:35 (02:41)	0.080*
Wake time, mean (SD)	7:18 (01:12)	7:18 (00:51)	0.664*
Total sleep time, mean (SD)	7.0 (1.3)	6.8 (1.2)	0.177*
Sleep latency, mean (SD)	14.1 (12.4)	16.3 (13.4)	0.310*
Sleep efficiency, mean (SD)	89.6 (13.1)	91.2 (9.3)	0.684*
PSQI score, median (min.-max.)	4.0 (1-14)	5.0 (0-13)	0.486*
MEQ score, median (min.-max.)	53.0 (31.0-75.0)	52.5 (33.0-75.0)	0.626*
Good sleep quality, n (%)	71 (67)	40 (58.8)	0.275**
Bad sleep quality, n (%)	35 (33)	28 (41.2)	
Morning chronotype, n (%)	31 (25.4)	22 (28.9)	0.110**
Intermediate chronotype, n (%)	82 (67.2)	42 (55.3)	
Evening chronotype, n (%)	9 (7.4)	12 (15.8)	
*Mann Whitney U test, **Chi-square tests.			
SD: Standard deviation, PSQI: Pittsburgh Sleep Quality Index, MEQ: Morningness-Eveningness Questionnaire, min.: Minimum, max.: Maximum			

Discussion

The aim of the present study is to evaluate the sleep quality and chronotype of parents and to examine their relationship with the sleep habits of their children. A total of 122 children between the ages of 4 and 12 participated in the study. Using the CSHQ questionnaire (with a cut-off score >41), 88.5% of the children were found to have sleep disturbances. According to

the PSQI questionnaire, 33% of mothers and 41.2% of fathers were identified as having poor sleep quality. Poor parental sleep quality was correlated with children's sleep disturbance and several sleep problems, including bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, and night waking. The National Sleep Foundation (NSF) recommends that children aged 3 to 5 should get 10 to 13 hours of sleep, those aged 6 to 13 should receive 9 to 11 hours, and teens aged 14 to 17 should obtain 8 to 10 hours.²⁵ In our study, the mean total sleep duration for children was 9.3 hours. In a study recently conducted by Ünsal et al.²⁶ in our country, the total daily sleep time of children was found to be 9.33±1.09 hours. The NSF recommends 7-9 hours of sleep for adults; in our study, the mean total sleep time was 7.0 hours for mothers and 6.8 hours for fathers. These findings showed that the children and parents in our study were at the lower limit of optimal sleep duration. Additionally, based on the CSHQ questionnaire, which is valid and reliable for assessing children's sleep habits in our country, 88.5% of the children were found to have sleep disorders. Recent meta-analyses have shown that the global prevalence of sleep disorders in children and adolescents has reached the alarming levels of 54% and 34%.^{7,8} Similarly, a recent study conducted in our country found that 62.9% of children experienced sleep disturbances.²⁶ These findings suggest that sleep disturbance is a widespread public health problem that requires urgent attention. Given the critical role of regular and adequate sleep in children's health and well-being, pediatricians should be aware of this issue and provide guidance to families. Moreover, school- and community-based interventions to raise awareness and promote lifestyle changes are essential for improving sleep health in children and their families.

Bedtime, hour, mean (SD)	21:31 (02:01)
Wake time, hour, mean (SD)	7:37 (00:39)
Total sleep time, hour, mean (SD)	9.3 (1.1)
WASO, minute, mean (SD)	6.5 (8.0)
CSHQ score, median (min.-max.)	49.0 (34.0-67.0)
Sleep disturbance, n (%)	108 (88.5)
Bedtime resistance, score, median (IQR)	11.0 (5.0)
Sleep onset delay, score, median (IQR)	1.0 (1.0)
Sleep duration, score, median (IQR)	3.0 (1.0)
Sleep anxiety, score, median (IQR)	8.0 (3.0)
Night wakings, score, median (IQR)	4.0 (3.0)
Parasomnias, score, median (IQR)	8.0 (3.0)
Sleep disordered breathing, score, median (IQR)	3.0 (1.0)
Daytime sleepiness, score, median (IQR)	14.0 (5.0)

A score greater than 41 on the CSHQ indicates a sleep disturbance, with higher CSHQ scores reflecting more sleep problems.
SD: Standard deviation, IQR: Interquartile range, CSHQ: Children's Sleep Habits Questionnaire, WASO: Wake after sleep onset

	1	2	3	4	5	6	7	8	9	10	11
1. CSHQ score	1										
2. Bedtime resistance	0.714**	1									
3. Sleep onset delay	0.366**	0.323**	1								
4. Sleep duration	0.484**	0.251**	0.499**	1							
5. Sleep anxiety	0.681**	0.783**	0.171	0.197*	1						
6. Night wakings	0.526**	0.488**	0.164	0.168	0.489**	1					
7. Parasomnias	0.453**	0.231*	0.100	0.082	0.279**	0.317**	1				
8. Sleep disordered breathing	0.251**	0.012	0.046	0.196*	0.088	0.060	0.230*	1			
9. Daytime sleepiness	0.347**	-0.103	-0.140	0.055	-0.075	-0.199*	-0.129	-0.055	1		
10. PSQI score	0.192*	0.251**	0.306**	0.213*	0.195*	0.210*	0.136	0.064	-0.202*	1	
11. MEQ score	-0.092	-0.108	-0.080	0.022	-0.210*	-0.066	-0.093	-0.146	0.042	-0.359**	1

Spearman Rho correlation coefficients,
*, **: Significant correlations at p<0.05, p<0.01 level, respectively. Higher PSQI scores indicate poorer sleep quality, while higher MEQ scores are related with morningness. Higher CSHQ scores reflect more sleep problems.
CSHQ: Children's Sleep Habits Questionnaire, PSQI: Pittsburgh Sleep Quality Index, MEQ: Morningness-Eveningness Questionnaire

Family structure and parental behavior play a crucial role in shaping children's lives. Parents' sleep disturbances may impact their children's sleep and vice versa. In this study, poor sleep quality was correlated with children's sleep disturbance and several sleep problems, including bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, and night waking. A meta-analysis performed by Varma et al.²⁸ showed associations between parents and their children regarding sleep duration, sleep quality, and sleep efficiency. Sleep problems in children were associated with poorer sleep quality and more insomnia in parents.²⁷ Chehri et al.⁵ reported a significant association between children's sleep patterns and the sleep quality and hygiene of their parents. In the study by Varma et al.¹⁸ children's sleep issues were found to be linked to parents' sleep disturbances.¹⁸ Urfer-Maurer et al.¹³ reported that maternal sleep problems were associated with objective sleep parameters in children, whereas paternal sleep problems showed no significant effect. Given the potential benefits of healthy sleep habits for children's future health, it is critical to consider parental sleep habits and quality. Future research is encouraged to determine the influence of family dynamics on sleep problems in both adults and children.

It is essential to highlight that in this study, children's sleep was assessed based on the mother's reports. Rönnlund et al.¹⁷ reported that parents with sleep problems tended to have more sleep problems in their children; however, parental sleep disturbances were not related with objective measures of child sleep.¹⁶ In the research conducted by Urfer-Maurer et al.¹⁴ maternal insomnia was linked to children's bedtime resistance and sleep anxiety, whereas paternal insomnia was associated with children's sleep duration and daytime sleepiness. However, the objective sleep parameters of the children did not explain these associations.¹³ Therefore, it was thought that future studies should examine objective sleep parameters along with parental reports.

Our findings revealed that more than half of both mothers and fathers were classified as intermediate chronotypes, followed by morning chronotypes, with evening chronotypes being the least common. This suggests that a significant proportion of both mothers and fathers may struggle to adapt to the intense demands of daily parenting. Additionally, the study found that as parental MEQ scores increased, reflecting a greater preference for morningness, children's sleep anxiety decreased and parental sleep quality improved. Consistent with expectations, higher MEQ scores, indicating a morning chronotype, were related with better sleep quality. Morales-Muñoz et al.²⁸ discovered that maternal chronotype was related with sleep problems in early childhood and may be regarded as a potential risk factor for the occurrence of early sleep problems. They also found that maternal eveningness was linked to shorter sleep duration in young children. However, paternal chronotype was not related with sleep parameters. It is important to recognize that parents serve as role models in shaping their children's sleep habits. Healthcare professionals should consider parents' chronotypes and lifestyles when providing guidance on optimizing sleep and addressing sleep problems in both parents and children.

Study Limitations

Several limitations of the study should be acknowledged. First, parental participation and questionnaire responses were voluntary, and both parental and child sleep status were assessed using self-report questionnaires rather than objective measurements. In addition, the assessment of children's sleep relied solely on mothers' perceptions, which may introduce bias. Finally, the cross-sectional design restricts the capacity to draw causal inferences among sleep-related variables. In spite of the limitations, the findings of this study are valuable because they explore the relationship between parents' chronotype, sleep quality, and children's sleep habits, particularly in the relatively understudied age group of 4-12 years.

Conclusion

This study found that 88.5% of children experienced sleep disturbances and highlighted a relationship between children's sleep habits and parents' sleep quality. These results underscore the importance of addressing parental sleep health as a potential strategy for improving children's sleep health. Future research should further explore these relationships to develop comprehensive strategies that support the sleep health of both parents and children.

Ethics

Ethics Committee Approval: Ethical approval for this research was secured from the Institutional Review Board of Koç University (approval number: 2023.375.IRB3.168, date: 02.11.2023).

Informed Consent: It was obtained.

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Footnotes

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Somatization and Sleep Pathology from a Transdiagnostic Dimensional Perspective: Insights into Detachment Spectrum from a Large Community

Somatizasyon ve Uyku Patolojisi: Transdiagnostik Boyutsal Bir Perspektiften Ayrışma Spektrumu Üzerine Büyük Bir Toplumsal Bakış

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Abstract

Objective: Somatization and sleep problems are psychiatric conditions with high comorbidity that appear to be more influenced by emotional dysfunctions than by other transdiagnostic constructs. The present study aimed to predict dimensional measures of somatization, poor sleep quality, insomnia severity, nightmare severity, and nightmare frequency using the transdiagnostic construct of detachment manifestations.

Materials and Methods: An online survey was conducted to collect cross-sectional data from 1,106 Iranian adults (64% female; mean age =32±9.6 years; age range =17 to 73 years) between August and December 2023. Participants completed the measurement inventory of detachment manifestations, the revised form of the symptom checklist-90, and several validated questionnaires assessing sleep disturbances. Data were analyzed using Pearson correlations and multiple linear regression analyses.

Results: Only the self-focused pattern of detachment manifestations significantly predicted somatization ($R^2=0.23$, $p<0.001$), poor sleep quality ($R^2=0.15$, $p<0.001$), insomnia severity ($R^2=0.20$, $p<0.001$), nightmare frequency ($R^2=0.10$, $p<0.001$), and nightmare severity ($R^2=0.14$, $p<0.001$). Specifically, both subtypes of the self-focused pattern-dissociative ($\beta=0.16$ to 0.45, all $p<0.002$) and self-body manifestations ($\beta=0.08$ to 0.18, all $p<0.05$) were meaningful predictors of nearly all criterion variables.

Conclusion: The self-focused pattern of detachment manifestations -especially the dissociative subtype-is a key construct in predicting somatization and sleep problems. A better understanding of the phenomenology and underlying mechanisms underlying somatization and sleep disturbances within transdiagnostic frameworks requires shifting the focus from emotional dysfunction to the detachment spectrum.

Keywords: Detachment, abnormal personality, psychopathology, sleep disturbance, somatization

Öz

Amaç: Somatizasyon ve uyku problemleri, yüksek komorbiditeye sahip, duygusal işlev bozukluklarından daha fazla etkilenen psikiyatrik durumlar olarak görünmektedir. Bu çalışmanın amacı, somatizasyon, kötü uyku kalitesi, insomnia şiddeti, kabus şiddeti ve kabus sıklığı gibi boyutsal ölçümleri, ayrışma belirtilerinin transdiagnostik kavramı ile tahmin etmektir.

Gereç ve Yöntem: 2023 yılı Ağustos ve Aralık ayları arasında 1,106 İranlı yetişkinden (%64 kadın, 32±9,6 yaş, 17 ile 73 arasında) kesitsel verilerin toplandığı çevrimiçi bir anket kullanıldı. Katılımcılar, ayrışma belirtileri ölçüm envanteri, gözden geçirilmiş semptom kontrol listesi-90 ve uyku bozukluklarını ölçmek için birkaç geçerli anket tamamladılar. Veriler, Pearson korelasyonları ve çoklu doğrusal regresyon analizi ile analiz edilmiştir.

Bulgular: Sadece kendine odaklı ayrışma belirtileri modeli, somatizasyonu ($R^2=0,23$; $p<0,001$), kötü uyku kalitesini ($R^2=0,15$; $p<0,001$), insomnia şiddetini ($R^2=0,20$; $p<0,001$), kabus sıklığını ($R^2=0,10$; $p<0,001$) ve kabus şiddetini ($R^2=0,14$; $p<0,001$) önemli ölçüde tahmin etmiştir. Özellikle, kendine odaklı modelin her iki alt tipi, dissosiyatif ($\beta=0,16$ ile 0,45, tüm $p<0,002$) ve kendine-düşkünlük belirtileri ($\beta=0,08$ ile 0,18, tüm $p<0,05$) neredeyse tüm kriter değişkenlerinin anlamlı tahminicileri olmuştur.

Sonuç: Kendine odaklı ayrışma belirtileri modeli -özellikle dissosiyatif alt tipi- somatizasyon ve uyku problemlerini tahmin etmede anahtar bir kavramdır. Somatizasyon ve uyku problemlerinin fenomenolojisi ve temel mekanizmalarının transdiagnostik sistemlerde daha iyi anlaşılması, duygusal işlev bozukluğundan ayrışma spektrumuna bir kaymayı gerektirmektedir.

Anahtar Kelimeler: Ayrışma, anormal kişilik, psikopatoloji, uyku bozukluğu, somatizasyon

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Introduction

Psychiatric comorbidity, which is quite common, refers to the presence of two or more mental disorders or diseases in the same individual simultaneously.¹ Since individuals with comorbid disorders often experience greater symptom severity and a higher risk of functional impairment, comorbidity complicates accurate diagnosis and treatment due to the complex interplay of symptoms.² Specifically, somatization and sleep problems are among diagnostic categories with high comorbidity rates—ranging from 20% to 48%—which negatively impact quality of life.³

Somatization is a complex psychiatric phenomenon involving the manifestation of psychological distress through physical symptoms that are otherwise unexplained. Diagnosing somatization is challenging because it may be mistaken for physical illnesses, prompting patients to seek multiple medical opinions in search of an accurate diagnosis.⁴ Sleep is also a complex process that influences cognitive functioning, emotional regulation, and overall well-being.⁵ Sleep problems encompass a wide range of disorders, including insomnia, nightmares, and poor sleep quality.

Despite the high comorbidity between somatization and sleep problems, the categorical assessment approach tends to overestimate comorbidities across psychopathology. Categorical assessment, or symptom-based psychiatric nosology, focuses on diagnosing distinct disorders and can contribute to the perceived increase in comorbidity. This is because the rigid boundaries of diagnostic categories may lead to overlapping symptoms across multiple disorders, increasing the likelihood that individuals meet criteria for more than one diagnosis.⁶

Current approaches to psychopathology aim to address the limitations of comorbidity by adopting dimensional assessment frameworks and transdiagnostic models. Dimensional assessment within a hierarchical structure helps reduce comorbidity by moving beyond rigid diagnostic categories and concentrating on symptom severity and interplay. This approach enables a more nuanced understanding of individual patients, leading to more tailored and effective treatment plans.⁷ Transdiagnostic systems seek to identify commonalities across different mental health disorders, rather than focusing solely on specific diagnoses. These systems can help treat comorbidity by targeting underlying mechanisms and shared processes that contribute to multiple disorders.⁸

The research domain criteria and the hierarchical taxonomy of psychopathology (HiTOP) are two prominent transdiagnostic frameworks used to classify mental disorders. For example, HiTOP is designed to improve upon traditional systems by organizing disorders based on shared genetic vulnerabilities, environmental risk factors, and neurobiological abnormalities, rather than relying solely on symptom clusters.⁹ In this framework, comorbid symptoms and syndromes are encompassed by transdiagnostic constructs. For instance, distress and fear-related pathologies (e.g., depression, anxiety, phobias, and insomnia) are associated with the internalizing spectrum, while somatic symptoms and illness anxiety fall within the somatoform spectrum. Although

initially challenging, further research has supported treating these as two distinct spectra within HiTOP.¹⁰ At its highest level, HiTOP introduces a general factor of psychopathology, which comprises three super-spectra: externalizing (antagonistic and disinhibited behaviors), psychosis (detachment and thought disorder), and emotional dysfunction (internalizing and somatoform).⁹

Parallel to HiTOP, which addresses the challenges of comorbidity between somatization and sleep problems through the emotional dysfunction super-spectrum, the literature also explores the links between emotion dysregulation and these comorbid conditions.^{11,12} However, this should not lead to neglecting the connections between the comorbid conditions and other transdiagnostic constructs. In the present study, we aim to investigate the associations between the detachment spectrum within HiTOP and somatization, as well as sleep problems (e.g., insomnia, nightmares, and poor sleep quality). Detachment can manifest as emotional numbness, social withdrawal, and decreased engagement with one's environment, often observed in conditions like depression and anxiety disorders. Additionally, individuals may employ avoidance strategies as coping mechanisms, distancing themselves from distressing emotions or thoughts. The detachment spectrum specifically captures individual differences in sociability (ranging from high engagement to disinterest), volition (from enthusiastic goal pursuit to apathy), and affective expression (from highly expressive to restricted). It encompasses traits such as introversion and negative schizotypy (e.g., anhedonia, social withdrawal, avolition, anergia, affective flattening, and alogia), as well as negative symptoms of schizophrenia.¹³ Detached individuals may experience emotional anhedonia or depression and often tend to avoid social interactions, leading to withdrawal from others, whom they may view with suspicion.¹⁴

Detachment is considered the opposite pole of extraversion within the five-factor model¹⁵ and is also related to the concept of attachment.¹⁶ Theoretically, the detachment spectrum highlights the interconnectedness of psychiatric conditions, suggesting that detachment may serve as a common vulnerability factor. In transdiagnostic models of psychopathology, detachment is viewed as a core psychological construct that influences various mental health disorders.

Current Study

Research on the association between detachment, somatization, and sleep problems is limited, and the existing findings are contradictory and heterogeneous. Some studies have reported significant associations between the detachment spectrum (e.g., insecure attachment, low extraversion, anhedonia, depressivity, withdrawal, intimacy avoidance, perceptual dysregulation, and schizotypy) and somatization and sleep issues,¹⁷⁻²⁴ while others have found no significant links between detachment and these psychiatric conditions.²⁵⁻²⁷ Additionally, small sample sizes in some studies threaten the validity of the findings. These issues highlight the need for further research.

Furthermore, there is a gap in the literature, as most studies have focused on personality features of the detachment spectrum, whereas the concept of detachment encompasses a broader psychopathological spectrum, including psychotic and dissociative features. Since most scales designed to assess the HiTOP detachment spectrum rely on personality traits such as maladaptive extraversion,²⁸ we utilized the Measurement Inventory of Detachment Manifestations (MINDs) for a more comprehensive assessment.¹⁴

Detachment manifestations are a recently conceptualized transdiagnostic construct that includes broader psychopathology, such as personality features and dissociative phenomena. These manifestations involve two main patterns: self-focused and other-focused. The self-focused pattern includes subtypes such as self-body (e.g., avoiding perceiving one's own image or voice, often stemming from discomfort, self-criticism, or dissociation, which hampers self-acceptance and personal identity) and dissociative (interpersonal dysfunction caused by a lack of coherence or continuity among thoughts, memories, surroundings, actions, and identity). The other-focused pattern encompasses manifestations like social (avoiding interpersonal intimacy or social relations), family (avoiding intimacy with family members), physical (avoiding physical contact), verbal (avoiding conversations or using very short sentences), visual-auditory (avoiding seeing or listening to others), ethnic-racial (avoiding relationships with certain ethnic groups or races), collaborative (avoiding cooperation or collaboration), and feeling (avoiding discussing experienced feelings with others).¹⁴ The detachment manifestations are a recently conceptualized transdiagnostic construct that includes broader psychopathology (e.g., personality features, dissociations, etc.). Detachment manifestations consist of two big constructs, including the self-focused and the other-focused patterns. Self-body (when individuals avoid perceiving their own image or voice, often stemming from discomfort, self-criticism, or dissociation, hindering self-acceptance and personal identity) and dissociative (an interpersonal dysfunction caused by lack of coherence or continuity between thoughts, memories, surroundings, actions, and identity) manifestations are the subtypes of the self-focused pattern, while the other-focused pattern includes social (any avoidance of interpersonal intimacy or social relations with others), family (any avoidance of intimacy with family members), physical (any avoidance of physical contact with others), verbal (any avoidance of conversation with others or use of very short sentences), visual-auditory (any avoidance of seeing others and any avoidance of listening to others), ethnic-racial (any avoidance of relationships with certain ethnic groups and races), collaborative (any avoidance of collaboration and cooperation with others), and feeling (any avoidance of conversation with others about experienced feelings) manifestations.¹⁴

Finally, recent research on the relationship between detachment and nightmare disorders is scarce. Only one study reported a negative association between detachment and the occurrence of positive dreams.²⁹

In the present study, we aimed to address these gaps by applying a comprehensive scale to measure the detachment spectrum in a large sample. We also included nightmares as a criterion variable. Our first aim was to predict somatization and sleep problems-such as poor sleep quality, insomnia severity, nightmare frequency, and nightmare severity-based on the broad types of detachment manifestations, including both other-focused and self-focused patterns. Our second objective was to predict all criterion variables based on specific detachment subtypes-namely, social, family, physical, verbal, visual-auditory, ethnic-racial, collaborative, feeling, self-body, and dissociative manifestations.

Materials and Methods

Design and Sample

Iranian adults were invited to participate in an online survey from August to December 2023 through personal requests, phone calls, and social media apps such as Telegram, WhatsApp, and Instagram. We specifically targeted individuals aged 17 or older who had not used illegal drugs or psychiatric medications in the past four weeks to complete the questionnaires.

We calculated the statistical power and adequacy of the sample size for detecting a small to medium effect size of 0.15, with a power level of 0.99 and an alpha of 0.01 for multiple regression analyses involving ten predictor variables (see <https://www.danielsoper.com/statcalc/calculator.aspx?id=1>). This analysis indicated a minimum required sample size of 280 participants. However, we increased the sample size nearly fourfold to minimize all types of sampling errors, including alpha and beta errors, as well as the margin of error.³⁰

A total of 1,106 adults aged 17 to 73 consented to participate in this cross-sectional study. Among them, 333 (30%) reported previous experiences with counseling, psychotherapy, or medication. Participants completed the Persian versions of the MINDs,¹⁴ the Pittsburgh Sleep Quality Index,³¹ the Insomnia Severity Index,³² the Lucid Dream and Nightmare Frequency Scales (LDNFS),³³ the Disturbing Dream and Nightmare Severity Index,³⁴ and the somatization subscale of the Revised Symptom Checklist-90 (SCL-90-R).^{35,36} The MINDs subscales served as measures of the predictor variables, while the other instruments assessed the criterion variables. All participants provided signed, written informed consent. This study was approved by the Ethics Committee of Kermanshah University of Medical Sciences (approval number: IR.KUMS.REC.1402.125, date: 04.07.2023) and conducted in accordance with the Declaration of Helsinki.

Measures

MINDs: The questionnaire is a 62-item self-report inventory designed to assess detachment manifestations. It evaluates both self-focused and other-focused patterns. The self-focused pattern comprises two subcategories: self-body detachment (7 items: 12, 20, 27, 32, 45, 46, and 59) and dissociative detachment (17 items: 6, 10, 13, 16, 17, 18, 19, 21, 28, 30, 36, 37, 39, 44, 54, 58, and 61). The other-focused pattern includes eight subcategories: social (8 items: 1, 8, 29, 33, 40,

47, 52, and 55), family (6 items: 4, 9, 24, 43, 49, and 53), physical (3 items: 3, 31, and 57), verbal (4 items: 2, 23, 41, and 51), visual-auditory (9 items: 14, 15, 22, 25, 34, 35, 38, 48, and 62), ethnic-racial (3 items: 5, 50, and 56), collaborative (3 items: 7, 11, and 26), and feeling (2 items: 42 and 60). Except for six items-questions 1, 8, 24, 33, 52, and 55 -all items are scored directly, with responses rated on a Likert scale from 0 to 3 (0= completely false, 1= relatively false, 2= relatively true, 3= completely true). The score for each subscale is obtained by summing the item scores and dividing by the number of items, resulting in a subscale score ranging from 0 to 3. The initial validation study, including the Iranian population, reported acceptable reliability and validity for the questionnaire.¹⁴ In the present study, Cronbach's alpha coefficients for the subscales were as follows: self-body ($\alpha=0.78$), dissociative ($\alpha=0.88$), social ($\alpha=0.82$), family ($\alpha=0.75$), physical ($\alpha=0.66$), verbal ($\alpha=0.75$), visual-auditory ($\alpha=0.80$), ethnic-racial ($\alpha=0.75$), collaborative ($\alpha=0.78$), and feeling ($\alpha=0.60$). The internal consistency for the self-focused pattern was $\alpha=0.91$, for the other-focused pattern $\alpha=0.92$, and for the total scale items $\alpha=0.95$ -all indicating good reliability.

Pittsburgh Sleep Quality Index

This widely used measure assesses 7 subscales and a total sleep quality scale using 18 self-report items. The subscales include (i) the subjective quality of sleep (item 9), (ii) the delay in falling asleep (item 2 and the first part of item 5), (iii) the duration of sleep (question 4), (iv) sleep efficiency (manual calculation of some items), (v) the sleep disorders (the mean of item 5), (vi) using sleeping pills (question 6), and (vii) daily dysfunctions (the mean of items 7 and 8). Each subscale is scored from 0 to 3, with higher scores indicating poorer sleep quality. The initial validation supported good psychometric properties.³¹ Although scores above 5 are considered indicative of poor sleep quality in general, evidence in the Iranian population suggests a cut-off of 6.5 or higher.³⁷ The Persian version's psychometric properties are acceptable, and in our study, Cronbach's alpha was 0.76.

Insomnia Severity Index

The index contains 7 questions, which include questions to assess (i) the sleep onset dysfunction, (ii) sleep continuation due to frequent awakening, (iii) early awakening, (iv) dissatisfaction with sleep pattern, (v) daily performance dysfunctions, (vi) worry due to the sleep problem, and (vii) the negative impact on the quality of life. Items are rated from 0 to 4, with higher scores indicating more severe insomnia. Total scores range from 0 to 28, with a score of 15 or above indicating moderate to severe insomnia.³² The index has demonstrated acceptable validity and reliability in the Iranian population,³⁸ with a Cronbach's alpha of 0.88 in this study.

Nightmare and Lucid Dream Frequency Scale

This single-item scale includes seven categories to assess the frequency of nightmares. Grading ranges from "nothing" (score zero) to "several times a week" (score 7). Higher scores indicate a greater frequency of nightmares. The scale's developers have

reported retest reliability coefficients ranging from 0.75 to 0.89 across different samples.³³

Disturbing Dream and Nightmare Severity Index

This questionnaire comprises five items that assess the intensity of nightmares experienced by the individual. Given the variability in scoring across items, responses are scored from zero (no nightmare) to a maximum score of 14, depending on the item. Higher scores indicate greater nightmare severity. The authors of this scale have reported acceptable validity.³⁴ In this study, Cronbach's alpha for the index was 0.84.

Revised Form of Symptom Checklist-90

The SCL-90-R is a 90-item self-report questionnaire used to assess symptoms of mental disorders. It includes nine clinical subscales: depression (13 items), somatization (12 items), obsessive-compulsive disorder, anxiety, and psychoticism (10 items each), interpersonal sensitivity (9 items), phobic anxiety (7 items), hostility, and paranoid ideation (6 items each). Responses are rated on a Likert scale from 0 (no discomfort) to 4 (very severe discomfort). Initial validation studies have demonstrated acceptable reliability and validity for both the original and revised versions.^{35,36} The Persian version of the SCL-90-R has shown acceptable psychometric properties in the Iranian population.³⁹ In this research, we used only the somatization subscale, and the Cronbach's alpha was 0.90.

Statistical Analysis

No missing data were encountered, as responses to all questions in the online form were mandatory. First, we reported the means and standard deviations for all variables. Before conducting the main analyses, we examined whether the assumptions of parametric tests, such as data normality (Skewness and Kurtosis between -1 and +1 for most variables), were met. Subsequently, Pearson correlation coefficients were calculated to assess the relationships between detachment manifestations (both the two broad types and ten subtypes) and the criterion variables (somatization, sleep quality, insomnia severity, nightmare frequency, and nightmare severity). Multiple linear regression analyses were performed to predict each criterion variable.

In one set of regression models, the broad types of detachment-other-focused and self-focused patterns-served as predictor variables. In the other set, the ten subtypes of detachment-social, family, physical, verbal, visual-auditory, ethnic-racial, collaborative, feeling, self-body, and dissociative-were used as predictors. R^2 was computed to indicate the proportion of variance explained by each model. Standardized beta coefficients were also reported to quantify the associations between each detachment manifestation and the criterion variables. To control for potential confounding effects, additional regression models adjusted for gender and age.

All statistical analyses were conducted using IBM SPSS Statistics version 27.0 (IBM Corp., Chicago, USA, 2020). A significance level of $p \leq 0.05$ (two-tailed) was adopted for all tests.

Results

Supplementary Table 1 presents the demographic data of the full sample. The average age of participants was 32±9.6 years. Most participants were female (n=709, 64%), single (n=562, 51%), university-educated (n=917, 83%), employed (n=650, 59%), and residents of the western regions of the country (66%).

Supplementary Table 2 shows the descriptive statistics of the sample. The mean scores for all detachment subtypes ranged from 0.84 (self-body detachment) to 1.78 (feeling detachment). The mean scores for sleep disturbances ranged from 3.46 (nightmare frequency) to 9.76 (insomnia severity). The mean and standard deviation for somatization were 14.03 and 9.64, respectively.

Table 1 displays the correlation coefficients between all detachment manifestation types and both somatization and sleep disturbances. The broad types of detachment manifestations are significantly correlated with somatization (r from 0.30 to 0.48; all p≤0.001), poor sleep quality (r from 0.29 to 0.39; all p≤0.001), insomnia severity (r from 0.34 to 0.44; all p≤0.001), nightmare frequency (r from 0.25 to 0.31; all p≤0.001), and nightmare severity (r from 0.27 to 0.38; all p≤0.001).

Additionally, the subtypes of detachment manifestations are significantly correlated with somatization (r from 0.10 for feeling detachment to 0.48 for dissociative detachment; all p≤0.001),

poor sleep quality (r from 0.10 for feeling detachment to 0.38 for dissociative detachment; all p≤0.001), insomnia severity (r from 0.15 for feeling detachment to 0.42 for dissociative detachment; all p≤0.001), nightmare frequency (r from 0.11 for feeling and verbal types to 0.31 for dissociative detachment; all p≤0.001), and nightmare severity (r from 0.09 for feeling detachment to 0.37 for dissociative detachment; all p≤0.004).

Table 2 presents multiple linear regression models predicting the criterion variables (somatization, poor sleep quality, insomnia severity, nightmare frequency, and nightmare severity) based on the broad types of detachment manifestations, including self-focused and other-focused patterns. The results indicate that these broad types significantly predicted all criterion variables: somatization (R²=0.23; p<0.001), insomnia severity (R²=0.20; p<0.001), poor sleep quality (R²=0.15; p<0.001), nightmare severity (R²=0.14; p<0.001), and nightmare frequency (R²=0.10; p<0.001). Specifically, the self-focused pattern of detachment was a significant predictor of all these outcomes: somatization (β=0.53, p<0.001), insomnia severity (β=0.39, p<0.001), poor sleep quality (β=0.37, p<0.001), nightmare severity (β=0.36, p<0.001), and nightmare frequency (β=0.26, p<0.001). Conversely, the other-focused pattern did not significantly predict any of the criterion variables (β from -0.07 to 0.07; all p>0.05). The regression models adjusted for sex and age are presented in Supplementary Table 3.

Table 3 presents multiple linear regression models predicting the criterion variables (somatization, poor sleep quality, insomnia

Table 1. Correlations between the predictors and criterion variables

Predictors	Criterion variables									
	Somatization		Poor sleep quality		Insomnia severity		Nightmare frequency		Nightmare severity	
	r	p	r	p	r	p	r	p	r	p
Other-focused pattern	0.30	<0.001	0.29	<0.001	0.34	<0.001	0.25	<0.001	0.27	<0.001
Social detachment	0.26	<0.001	0.25	<0.001	0.28	<0.001	0.25	<0.001	0.28	<0.001
Family detachment	0.28	<0.001	0.29	<0.001	0.33	<0.001	0.24	<0.001	0.29	<0.001
Physical detachment	0.29	<0.001	0.24	<0.001	0.24	<0.001	0.25	<0.001	0.28	<0.001
Verbal detachment	0.15	<0.001	0.16	<0.001	0.20	<0.001	0.11	<0.001	0.10	<0.001
Visual-auditory detachment	0.26	<0.001	0.23	<0.001	0.28	<0.001	0.20	<0.001	0.22	<0.001
Ethnic-racial detachment	0.19	<0.001	0.21	<0.001	0.24	<0.001	0.14	<0.001	0.17	<0.001
Collaborative detachment	0.16	<0.001	0.15	<0.001	0.21	<0.001	0.15	<0.001	0.16	<0.001
Feeling detachment	0.10	<0.001	0.10	<0.001	0.15	<0.001	0.11	<0.001	0.09	0.004
Self-focused pattern	0.48	<0.001	0.39	<0.001	0.44	<0.001	0.31	<0.001	0.38	<0.001
Self-body detachment	0.42	<0.001	0.35	<0.001	0.39	<0.001	0.27	<0.001	0.32	<0.001
Dissociative detachment	0.48	<0.001	0.38	<0.001	0.42	<0.001	0.31	<0.001	0.37	<0.001

Table 2. Multiple regression models predicting the criterion variables based on the broad types of detachment manifestations

Predictors	Criterion variables									
	Somatization		Poor sleep quality		Insomnia severity		Nightmare frequency		Nightmare severity	
	β	p	β	p	β	p	β	p	β	p
Other-focused pattern	-0.07	0.073	0.03	0.402	0.07	0.059	0.07	0.078	0.03	0.768
Self-focused pattern	0.53	<0.001	0.37	<0.001	0.39	<0.001	0.26	<0.001	0.36	<0.001
R ²	0.23	<0.001	0.15	<0.001	0.20	<0.001	0.10	<0.001	0.14	<0.001

Table 3. Multiple regression models predicting the criterion variables based on the subtypes of detachment manifestations

Predictors	Criterion variables									
	Somatization		Poor sleep quality		Insomnia severity		Nightmare frequency		Nightmare severity	
	β	p	β	p	β	p	β	p	β	p
Other-focused pattern										
Social detachment	-0.04	0.272	0.02	0.629	0.03	0.459	0.09	0.021	0.08	0.049
Family detachment	-0.02	0.558	0.08	0.034	0.09	0.016	0.06	0.110	0.08	0.036
Physical detachment	0.02	0.627	-0.01	0.747	-0.08	0.033	0.07	0.065	0.07	0.063
Verbal detachment	-0.08	0.031	-0.05	0.231	-0.04	0.271	-0.12	0.003	-0.15	<0.001
Visual-auditory detachment	0.03	0.433	0.01	0.848	0.03	0.499	0.03	0.446	0.03	0.413
Ethnic-racial detachment	0.00	0.993	0.08	0.017	0.08	0.016	0.01	0.849	0.02	0.536
Collaborative detachment	-0.05	0.184	-0.03	0.357	0.01	0.802	-0.02	0.633	-0.02	0.580
Feeling detachment	-0.04	0.187	-0.03	0.388	-0.01	0.890	0.02	0.557	-0.02	0.579
Self-focused pattern										
Self-body detachment	0.16	<0.001	0.14	<0.001	0.18	<0.001	0.07	0.104	0.08	0.038
Dissociative detachment	0.45	<0.001	0.24	<0.001	0.24	<0.001	0.16	0.002	0.24	<0.001
R ²	0.25	<0.001	0.17	<0.001	0.21	<0.001	0.12	<0.001	0.17	<0.001

severity, nightmare frequency, and nightmare severity) based on the subtypes of detachment manifestations. The results indicate that detachment manifestations significantly predicted all criterion variables: somatization ($R^2=0.25$; $p<0.001$), poor sleep quality ($R^2=0.17$; $p<0.001$), insomnia severity ($R^2=0.21$; $p<0.001$), nightmare frequency ($R^2=0.12$; $p<0.001$), and nightmare severity ($R^2=0.17$; $p<0.001$). Specifically, dissociative ($\beta=0.45$, $p<0.001$), self-body ($\beta=0.16$, $p<0.001$), and verbal ($\beta=-0.08$, $p=0.031$) manifestations of detachment were significant predictors of somatization. Poor sleep quality was also significantly predicted by dissociative ($\beta=0.24$, $p<0.001$), self-body ($\beta=0.14$, $p<0.001$), ethnic-racial ($\beta=0.08$, $p=0.017$), and family ($\beta=0.08$, $p=0.034$) manifestations. Insomnia severity was significantly predicted by dissociative ($\beta=0.24$, $p<0.001$), self-body ($\beta=0.18$, $p<0.001$), family ($\beta=0.09$, $p=0.016$), ethnic-racial ($\beta=0.08$, $p=0.016$), and physical ($\beta=-0.08$, $p=0.033$) manifestations. Nightmare frequency was significantly associated with dissociative ($\beta=0.16$, $p=0.002$), verbal ($\beta=-0.12$, $p=0.003$), and social ($\beta=0.09$, $p=0.021$) manifestations. Finally, nightmare severity was significantly predicted by dissociative ($\beta=0.24$, $p<0.001$), verbal ($\beta=-0.15$, $p<0.001$), family ($\beta=0.08$, $p=0.036$), self-body ($\beta=0.08$, $p=0.038$), and social ($\beta=0.08$, $p=0.049$) manifestations of detachment.

Discussion

The present study aimed to predict dimensional measures of somatization, poor sleep quality, insomnia severity, nightmare frequency, and nightmare severity based on the transdiagnostic construct of detachment manifestations. The predictive models, which included the two broad types of detachment, explained 23% of the variance in somatization and 10-20% of sleep problems. However, we found that only the self-focused pattern of detachment (dissociative and self-body manifestations) was significantly related to both somatization and sleep problems.

Dissociative detachment is associated with somatization because both involve processing emotional and psychological distress through physical symptoms. Somatoform dissociation can manifest as a lack of integration of somatic experiences, leading to symptoms such as pain or paralysis without an identifiable medical cause. Additionally, individuals with dissociative disorders often exhibit high levels of somatization, with psychological trauma and stress expressed through bodily symptoms. This connection may also relate to underlying trauma-related factors, such as insecure attachment patterns.^{19,40} Furthermore, dissociative processes interfere with the integrative mechanisms of consciousness, memory, identity, or perception, contributing to somatic manifestations.¹⁹ Dissociative detachment has also been found to significantly influence sleep disorders; studies suggest that individuals experiencing dissociative symptoms tend to have higher levels of sleep problems.^{41,42} These symptoms can cause disturbances in sleep patterns, such as increased sleep intrusions during wakefulness, possibly contributing to depressive moods. Overall, there appears to be a complex relationship between dissociative experiences and sleep disturbances, underscoring their mutual influence. Dissociative experiences are also linked to higher distress levels in nightmares. Individuals who experience dissociative detachment may report more intense and distressing nightmares compared to those who do not. This association indicates that the psychological mechanisms underlying dissociation can exacerbate the frequency and emotional impact of nightmares.⁴¹ Regarding the relationship between self-body detachment and somatization, negative body image can lead to increased stress, anxiety, and emotional turmoil, which may manifest as physical symptoms. People with difficulties in self-acceptance may also be more attuned to their bodily sensations and thus report more somatic complaints as expressions of their psychological distress. Additionally, it is important to consider the potential

links between somatization and body dysmorphic disorder. The MINDs items related to self-body-such as “If I could, I would change my appearance or voice” and “I tear or delete photos of myself that I don’t like”-partially overlap with symptoms of body dysmorphic disorder. Some studies have reported a relationship between somatization and body dysmorphic disorder, as well as comorbidity between these diagnostic categories.^{43,44}

Individuals with negative body image may experience cognitive distortions and heightened dissatisfaction with their appearance, resulting in increased anxiety and low self-esteem. This emotional distress can negatively affect sleep quality and duration, leading to symptoms of insomnia.⁴⁵ Additionally, unhealthy coping strategies related to body image, such as avoidance or compulsive behaviors, may further disrupt sleep patterns.⁴⁶

We found that social and family types of detachment are predictors of nightmares. Social detachment may lead to nightmares due to the impact of social isolation on the brain’s dream processes. Isolated individuals might experience heightened interactions within their dreams, reflecting unmet social needs. In contrast, family detachment refers to the avoidance of ongoing and intimate communication with family members, which may stem from past traumatic experiences. Some studies have reported a relationship between childhood maltreatment and insecure attachment in adulthood.^{47,48} This disconnection from a vital support system-namely, the family-may threaten quality of life and sleep. Several studies have highlighted the association between poor family support and sleep problems.^{49,50}

We also found that ethnic-racial detachment is a significant predictor of insomnia severity and poor sleep quality. Given that Iran is a multi-ethnic and multicultural country, understanding some subcultures can be challenging for certain groups. Ethnic-racial detachment may contribute to insomnia due to the stressors and challenges faced by minority groups, which can adversely affect both mental and physical health, leading to sleep disturbances.

Finally, our results showed negative regression coefficients between verbal detachment and somatization, nightmare frequency, and nightmare severity. Although the bivariate correlations between these variables are positive, the negative associations in the regression models are likely due to suppressor effects.

The findings of this study have significant theoretical and clinical implications regarding the role of detachment manifestations in somatization and sleep disturbances. The predictive models underscore the importance of differentiating between self-focused and other-focused detachment patterns, with self-body and dissociative detachment being closely linked to somatic symptoms and sleep issues. These insights can inform therapeutic approaches, as targeting self-focused detachment may help alleviate both somatization and insomnia. Clinicians should consider assessing detachment manifestations, particularly in patients presenting with unexplained physical symptoms or sleep disturbances. The observed associations between social and family detachment and nightmares emphasize

the importance of addressing interpersonal relationships in treatment planning.

Additionally, recognizing the contribution of ethnic-racial detachment to insomnia can promote culturally sensitive interventions that address the unique stressors faced by minority groups. Overall, these findings support a transdiagnostic perspective, facilitating a more integrated understanding of mental health disorders and enhancing treatment efficacy.

Study Limitations

The present study is a pioneering effort to identify associations between detachment manifestations and two common mental health-threatening conditions across cultures: somatization and sleep problems. We used the MINDs, a recently developed dimensional questionnaire that uniquely covers all forms of detachment,¹⁴ to measure the transdiagnostic construct of detachment manifestations. We included a large sample to reduce sampling error and minimize bias.³⁰ Although knowledge about psychopathology primarily comes from Western populations,⁹ our analysis of data from a non-Western sample enhances the potential for cross-cultural generalizability. However, there are some limitations that future research should address.

The most significant limitation was the absence of a clinical sample with established psychiatric diagnoses. Including such a sample in future studies would increase the validity of the findings. Second, we employed a cross-sectional design and used self-report scales for data collection. A longitudinal design, along with data obtained from clinician-rated scales and clinical interviews, could improve the robustness and validity of the results. Third, self-report measures are susceptible to respondent biases. Future studies should aim to reduce these biases through semi-structured or structured interviews conducted by clinicians. Fourth, the LDNFS, used to measure nightmare frequency, relies on a single item, which may not capture all relevant nuances. Future research could utilize more comprehensive measurement tools to differentiate between trauma-related and idiopathic nightmares. Fifth, we did not examine participants’ trauma history or comorbid psychiatric conditions, which could potentially influence the findings. Future research should consider these confounding variables. Sixth, most participants were from the western regions of Iran, which may limit the generalizability of the results to other regions of the country. Finally, while online sampling offers convenience, it is subject to limitations such as online bias, which may affect representativeness. This approach may exclude individuals with limited internet access, further compromising diversity and potentially impacting the validity of the findings.

Conclusion

The current study found that only the self-focused pattern of detachment and its subtypes (dissociative and self-body manifestations) significantly predicted somatization, poor sleep quality, insomnia severity, nightmare frequency, and nightmare severity. Accordingly, detachment patterns related to

relationships with others do not appear to play a significant role in the psychopathology of somatization and sleep problems. We suggest that a deeper understanding of the phenomenology and underlying mechanisms of somatization and sleep disturbances within transdiagnostic frameworks requires a shift from focusing solely on emotional dysfunction to considering the detachment spectrum. However, the limitations discussed should be addressed in future research.

Ethics

Ethics Committee Approval: This study was approved by the Ethics Committee of Kermanshah University of Medical Sciences (approval number: IR.KUMS.REC.1402.125, date: 04.07.2023) and conducted in accordance with the Declaration of Helsinki.

Informed Consent: All participants provided signed, written informed consent.

Footnotes

Authorship Contributions

Concept: S.K., F.R., A.Z., B.F., H.K., Design: S.K., F.R., A.Z., B.F., H.K., Data Collection or Processing: S.K., A.Z., B.F., Analysis or Interpretation: S.K., H.K., Literature Search: S.K., F.R., A.Z., B.F., Writing: S.K., F.R., A.Z., B.F., H.K.

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Supplementary Table 1. Demographic data of the sample (n=1106)		
Variable	n	%
Sex		
Female	709	64.1
Male	397	35.9
Age groups		
17-45 years	996	90.1
46-65 years	107	9.7
>65 years	3	0.3
Education		
Under diploma	43	3.9
Diploma	146	13.2
Academic level	917	82.9
Job		
Employed	296	26.8
Self-employed	215	19.4
Housekeeper	139	12.6
College student	280	25.3
Other	176	15.9
Marital status		
Single	498	45.0
Married	608	55.0
Current psychotherapy or pharmacotherapy		
No	897	81.1
Yes	209	18.9
Previous psychotherapy or pharmacotherapy		
No	773	69.9
Yes	333	30.1
Geographical region		
Western	730	66.0
Other	376	34.0

Variables	Minimum	Maximum	Mean	SD	Skewness	Std. error	Kurtosis	Std. error
Somatization	0	48	14.03	9.64	0.80	0.07	0.28	0.15
Poor sleep quality	0	21	6.91	3.65	0.88	0.07	0.65	0.15
Insomnia severity	0	28	9.76	6.20	0.46	0.07	-0.44	0.15
Nightmare frequency	0	7	3.46	1.93	0.06	0.07	-0.67	0.15
Nightmare severity	0	34	6.77	6.31	1.27	0.07	1.81	0.15
Detachment manifestations								
Other-focused pattern	0	2.81	1.36	0.50	0.04	0.07	-0.21	0.15
Social detachment	0	3	1.08	0.60	0.15	0.07	-0.49	0.15
Family detachment	0	3	1.25	0.64	0.37	0.07	-0.34	0.15
Physical detachment	0	3	0.89	0.70	0.58	0.07	-0.20	0.15
Verbal detachment	0	3	1.51	0.68	-0.13	0.07	-0.45	0.15
Visual-auditory detachment	0	3	1.58	0.60	-0.12	0.07	-0.25	0.15
Ethnic-racial detachment	0	3	1.21	0.85	0.22	0.07	-0.87	0.15
Collaborative detachment	0	3	1.59	0.82	-0.02	0.07	-0.77	0.15
Feeling detachment	0	3	1.78	0.84	-0.27	0.07	-0.67	0.15
Self-focused pattern	0	2.84	0.85	0.53	0.62	0.07	0.23	0.15
Self-body detachment	0	3	0.84	0.63	0.75	0.07	0.29	0.15
Dissociative detachment	0	2.80	0.86	0.53	0.51	0.07	-0.00	0.15

SD: Standard deviation, Std.: Standard

Predictors	Criterion variables									
	Somatization		Poor sleep quality		Insomnia severity		Nightmare frequency		Nightmare severity	
	β	p	β	p	β	p	β	p	β	p
Sex-adjusted model										
Other-focused pattern	-0.05	0.176	0.04	0.240	0.08	0.023	0.10	0.009	0.05	0.171
Self-focused pattern	0.52	<0.001	0.36	<0.001	0.38	<0.001	0.25	<0.001	0.34	<0.001
R ²	0.23	<0.001	0.16	<0.001	0.19	<0.001	0.11	<0.001	0.14	<0.001
Age-adjusted model										
Other-focused pattern	-0.05	0.151	0.04	0.304	0.08	0.027	0.07	0.063	0.05	0.218
Self-focused pattern	0.52	<0.001	0.37	<0.001	0.39	<0.001	0.28	<0.001	0.35	<0.001
R ²	0.24	<0.001	0.16	<0.001	0.21	<0.001	0.11	<0.001	0.15	<0.001



The Relationship Between Sleep Spindle Characteristics and Obstructive Sleep Apnea Severity

Uyku İğcikleri Özellikleri ile Obstrüktif Uyku Apnesi Şiddeti Arasındaki İlişki

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Abstract

Objective: Obstructive sleep apnea (OSA) is a common condition that interrupts sleep due to repeated blockages in the airway. This study investigates whether sleep spindle characteristics in non-rapid eye movement (NREM) sleep stages can serve as reliable markers of OSA severity, aiding diagnosis and management.

Materials and Methods: In 2019, a cross-sectional study was conducted on 37 Qods Hospital Sleep Disorders Center patients. Overnight polysomnography was performed, and sleep spindle parameters-frequency (Hz), duration (seconds), and density (spindles/minute)-were manually analyzed in NREM N2 and N3 stages. Participants were classified based on OSA severity using the Apnea-Hypopnea Index (AHI): normal (AHI <5), mild (5-15), moderate (15-30), and severe (>30). The Epworth Sleepiness Scale (ESS) and Insomnia Severity Index (ISI) were used to assess daytime sleepiness and insomnia, respectively.

Results: Among the participants, 56.8% had severe OSA with an AHI greater than 30. The mean ISI score was 19.45 [standard deviation (SD) =6.24], indicating moderate to severe insomnia, and the mean ESS score was 10.18 (SD =5.29), reflecting moderate daytime sleepiness. The average sleep duration was 5.64 hours (SD =1.02), and the sleep efficiency was 75.36% (SD =10.96%). In NREM N2, spindle frequency was significantly lower in severe OSA (12.96 Hz, SD =0.05) than mild to moderate (13.05 Hz, SD =0.10; p=0.01, Cohen's d=0.65), and the duration was shorter (0.66 s, SD =0.09 vs. 0.80 s, SD =0.19; p=0.029). Spindle density correlated with Oxygen Desaturation Index (ODI; p=0.72, p=0.007). No significant NREM N3 differences were observed (p>0.05).

Öz

Amaç: Obstrüktif uyku apnesi (OSA), hava yolunda tekrarlayan tıkanıklıklar nedeniyle uykuyu kesintiye uğratan yaygın bir durumdur. Bu çalışma, hızlı göz hareketi olmayan (NREM) uyku aşamalarındaki uyku iğcikleri özelliklerinin OSA şiddetinin güvenilir belirteçleri olarak kullanılarak tanı ve tedavi yönetimine katkı sağlayıp sağlamayacağını araştırmaktadır.

Gereç ve Yöntem: 2019 yılında, Kudüs Hastanesi Uyku Bozuklukları Merkezi'nde tedavi gören 37 hasta üzerinde kesitsel bir çalışma gerçekleştirildi. Gece boyunca polisomnografi uygulandı ve NREM N2 ve N3 aşamalarında uyku iğcik parametreleri [frekans (Hz), süre (saniye) ve yoğunluk (iğcik/dakika)] manuel olarak analiz edildi. Katılımcılar, Apne-Hipopne İndeksi (AHI) kullanılarak OSA şiddetine göre sınıflandırıldı [normal (AHI <5), hafif (5-15), orta (15-30) ve şiddetli (>30)]. Gündüz uykululuk ve uykusuzluğu değerlendirmek için sırasıyla Epworth Uykululuk Ölçeği (ESS) ve Uykusuzluk Şiddet İndeksi (ISI) kullanıldı.

Bulgular: Katılımcıların %56,8'i 30'dan yüksek AHI değeri ile şiddetli OSA'ya sahipti. Ortalama ISI skoru 19,45 [standart sapma (SS) =6,24] idi, bu da orta ila şiddetli uykusuzluğu gösteriyordu, ve ortalama ESS skoru 10,18 (SS =5,29) idi, bu da orta derecede gündüz uykululuğu yansıtıyordu. Ortalama uyku süresi 5,64 saat (SS =1,02) ve uyku verimliliği %75,36 (SS =10,96) olarak bulundu. NREM N2'de, iğcik sıklığı şiddetli OSA'da (12,96 Hz, SS = 0,05) hafif ila orta dereceli OSA'ya (13,05 Hz, SS = 0,10; p=0,01, Cohen's d=0,65) göre anlamlı şekilde daha düşüktü ve süresi daha kısaydı (0,66 sn, SS =0,09 vs. 0,80 sn, SS =0,19; p=0,029). İğcik yoğunluğu Oksijen Desatürasyonu İndeksi (ODI; p=0,72, p=0,007) ile korelasyon gösteriyordu. NREM N3'te anlamlı bir farklılık gözlenmedi (p>0,05).

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Conclusion: The frequency and duration of sleep spindles in NREM N2 decreased with the severity of OSA, suggesting potential diagnostic value. However, no differences were observed in NREM N3, suggesting that this parameter requires further investigation in larger cohorts.

Keywords: Apnea/Hypopnea Index, obstructive sleep apnea syndrome, spindle frequency, Hypopnea Index

Sonuç: NREM N2'deki uyku içcikleri sıklığı ve süresi, OSA'nın ciddiyetiyle birlikte daha düşüktü, bu da potansiyel tanılabilir değeri olduğunu düşündürmektedir. Ancak, NREM N3'te herhangi bir fark gözlemlenmedi, bu da bu parametrenin daha büyük kohortlarda daha fazla araştırılması gerektiğini göstermektedir.

Anahtar Kelimeler: Apne/Hipopne İndeksi, obstrüktif uyku apnesi sendromu, içcik frekansı, Hipopne İndeksi

Introduction

Obstructive sleep apnea (OSA) is a common sleep disorder characterized by repeated upper airway blockages during sleep, leading to apnea or hypopnea.^{1,2} This can disrupt sleep and oxygenation, leading to daytime sleepiness and cognitive deficits.^{3,4} Epidemiological data indicate that moderate to severe OSA [Apnea-Hypopnea Index (AHI) ≥ 15] affects approximately 13% of men and 6% of women aged 30-70,⁵ with prevalence varying by gender and age.^{2,6}

Polysomnography (PSG), the definitive diagnostic tool for OSA, measures brain activity (EEG), eye movements (EOG), and oxygen saturation.^{7,8} Sleep spindles-oscillatory bursts of 11-15 Hz, lasting 0.5-2 seconds-occur predominantly in non-rapid eye movement (NREM) stage 2 sleep and are generated by interactions between the thalamic reticular nucleus and thalamocortical neurons.^{9,10} These spindles are linked to memory consolidation, learning, and sleep stability,^{11,12} and their characteristics are altered in neurodevelopmental and pathological states.^{13,14}

Patients with OSA exhibit reduced spindle activity during NREM stages N2 and N3, with frequency and density declining as disease severity increases.^{15,16} These spindle deficits have been further linked to cognitive impairment, particularly in memory consolidation.¹⁷ While some studies report partial recovery of spindle activity following continuous positive airway pressure (CPAP) therapy,¹⁸ others highlight significant variability in treatment responses, suggesting individual differences in neural recovery mechanisms.¹⁹ Considering these inconsistencies and the necessity of further research, this study was designed to investigate sleep spindle characteristics (frequency, duration, and density) in NREM N2 and N3 in different OSA severity levels to evaluate their diagnostic potential and elucidate their role in OSA pathophysiology.

Materials and Methods

This descriptive cross-sectional study was conducted on patients admitted to the Qods Hospital Sleep Disorders Center in 2019. The study was approved by the Ethics Committee of Qazvin University of Medical Sciences (approval number: IR.QUMS.REC.1397.103, date: 05.06.2018). After providing written informed consent, all patients referred for OSA assessment were included through a census sampling method.

Participants

The study included 37 patients evaluated for OSA. Patients were excluded if they had chronic conditions such as neurodegenerative diseases (e.g., Alzheimer's), uncontrolled cardiovascular diseases (e.g., heart failure), alcoholism,

substance use, sedative-hypnotic medication use, or had shift work. These exclusion criteria were established to minimize confounding factors affecting sleep architecture.

Demographic and clinical information was collected through a structured survey. The collected demographic data included age, gender, marital status, employment status, and education. Anthropometric data, including height and weight, were recorded, and body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2).

Sleep Assessments

Participants were evaluated for sleep disorders using validated tools: the Epworth Sleepiness Scale (ESS); a score of 10 or greater indicates excessive daytime sleepiness²⁰ the Insomnia Severity Index (ISI); a score of 8 or greater indicates insomnia,²¹ and the Pittsburgh Sleep Quality Index (PSQI); a score of 5 or greater indicates poor sleep quality.²¹

Polysomnography

All participants underwent overnight PSG following the 2018 American Academy of Sleep Medicine (AASM) standards.²² The PSG recorded EEG, EOG, EMG, and oxygen saturation (SpO_2), with apnea defined as a $\geq 90\%$ airflow reduction for ≥ 10 seconds and hypopnea as a $\geq 30\%$ reduction with $\geq 3\%$ desaturation or arousal.²³ OSA severity was classified according to AASM guidelines: AHI < 5 (normal), 5-15 (mild), 15-30 (moderate), > 30 (severe).²⁴ A certified sleep specialist, blinded to the patient's identity, manually analyzed the data.

Sleep Spindle Analysis

Sleep spindle characteristics (frequency, duration, and density) were manually extracted from EEG recordings in NREM stages N2 and N3 by a single experienced scorer blinded to OSA status.^{25,26} Frequency (Hz) was calculated as the oscillatory cycles per second within the 11-15 Hz range, duration (seconds) as the spindle length, and density as the number of spindles per minute.

Statistical Analysis

Data analysis was conducted using SPSS version 25. The normality of the data was assessed with the Shapiro-Wilk test. For normally distributed variables, parametric tests such as t-tests and ANOVA were applied. Non-parametric tests, including the Mann-Whitney U test, were used for non-normally distributed variables, and Bonferroni corrections were applied for multiple comparisons. Descriptive statistics included means and standard deviations (SD) for continuous variables and frequencies and percentages for categorical variables. Chi-square tests were used to evaluate differences among categorical variables, while Pearson's correlation was employed

to assess relationships between continuous variables. A p-value of less than 0.05 was considered statistically significant.

Results

In this study, 37 patients were examined at Qods Hospital Sleep Disorders Center. The demographic and clinical characteristics showed that 43.2% of the patients were female (n=16), 81.1% were married (n=30), and 51.4% had below-diploma education (n=19). As for employment status, 37.8% were housewives (n=14) and 32.4% were self-employed (n=12). 56.8% reported no medical conditions (n=21), and 24.3% had hypertension (n=9). The mean age was 47.27 years (SD =15.76), and the mean BMI was 30.97 kg/m² (SD =5.24). OSA severity varied among participants: 56.8% had severe OSA (n=21, AHI >30), 21.6% had moderate OSA (n=8, AHI 15-30), 10.8% had mild OSA (n=4, AHI 5-15), and 10.8% did not have OSA (n=4, AHI <5).²⁴ The mean ESS score was 10.18 (SD =5.29), indicating moderate daytime sleepiness, and the mean ISI score was 19.45 (SD =6.24), reflecting moderate to severe insomnia. Objective sleep parameters revealed a mean sleep duration of 5.64 hours (SD =1.02) with 75.36% efficiency (SD =10.96). The mean Respiratory Disturbance Index was 54.38 events/hour (SD =41.09), mean oxygen saturation (SpO₂) was 89.40% (SD =5.96), and minimum SpO₂ was 73.70% (SD =16.49).

Table 1 summarizes sleep spindle characteristics in NREM N2 and N3 stages, alongside ESS and ISI scores. Spindle density in NREM N2 (SNN2) averaged 720 spindles (SD =573.44), with a frequency (SFN2) of 12.98 Hz (SD =0.08) and duration (SDN2) of 0.70 seconds (SD =0.14). In non-rapid eye movement (NREM) stage N3, spindle density (SNN3: spindle number in NREM N3) was 73.72 (SD =55.68), frequency (SFN3) was 12.96 Hz (SD =0.12), and duration (SDN3) was 0.64 seconds (SD =0.07).^{9,12} Daytime sleepiness (ESS ≥10) affected 59.5% (n=22), and moderate to severe insomnia (ISI ≥15) affected 81% (n=30).

Table 2 compares spindle characteristics across OSA severity groups. In NREM N2, severe OSA patients had lower spindle frequency (SFN2 =12.96 Hz, SD =0.05) than mild-to-moderate [13.05 Hz, SD =0.10; p=0.01, Cohen's d=0.65, 95% CI (-0.12, -0.01)] and shorter duration (SDN2 =0.66 s, SD =0.09) than mild-to-moderate [0.80 s, SD =0.19; p=0.029, d=0.58, 95% CI (-0.25, -0.03)] after Bonferroni correction.²⁵ Spindle density (SNN2) trended lower in severe OSA (555.90, SD =489.39) versus mild-to-moderate (1071, SD =688.66; p=0.06). No significant NREM N3 differences were observed (p>0.05).

Table 3 demonstrates significant correlations between spindle characteristics and PSG indices. NREM N2 spindle density (SNN2) strongly correlated with Oxygen Desaturation Index (ODI; p=0.72, p=0.007) and heart rate (HR; p=-0.27, p=0.02).

Variable	Mean ± SD	Range
NREM N2 spindle density (SNN2)	720±573.44	50-2500
NREM N2 frequency (SFN2, Hz)	12.98±0.08	12.80-13.15
NREM N2 duration (SDN2, s)	0.70±0.14	0.45-1.00
NREM N3 spindle density (SNN3)	73.72±55.68	0-300
NREM N3 frequency (SFN3, Hz)	12.96±0.12	12.70-13.20
NREM N3 duration (SDN3, s)	0.64±0.07	0.50-0.80
ESS score	10.1±5.29	2-22
Percent of the subjects with daytime sleepiness (ESS ≥10)	59.5% (n=22)	-
ISI score	19.45±6.24	5-28
Percent of the subjects with moderate to severe insomnia (ISI ≥15)	81% (n=30)	-

SNN2: Spindle number in NREM stage 2, SFN2: Spindle frequency in NREM stage 2, SDN2: Spindle duration in NREM stage 2, SNN3: Spindle number in NREM stage 3, SFN3: Spindle frequency in NREM stage 3, SDN3: Spindle duration in NREM stage 3, ESS: Epworth Sleepiness Scale, ISI: Insomnia Severity Index^{9,12,21,22}

Variable	Normal (n=4)	Mild-to-moderate (n=12)	Severe (n=21)	p
SNN2	704±328.35	1071±688.66	555.90±489.39	0.06
SFN2 (Hz)	12.97±0.05	13.05±0.10	12.96±0.05	0.01*
SDN2 (s)	0.65±0.10	0.80±0.19	0.66±0.09	0.029*
SNN3	30.67±21.19	86.8±66.11	35.10±35.25	0.26
SFN3 (Hz)	13.00±0.00	12.94±0.11	12.97±0.14	0.78
SDN3 (s)	0.63±0.05	0.65±0.11	0.63±0.04	0.78

*Bonferroni-corrected p-values; p<0.05. Effect sizes: SFN2 [d=0.65, 95% CI (-0.12, -0.01)], SDN2 [d=0.58, 95% CI (-0.25, -0.03)].²⁵
SNN2: Spindle number in NREM N2, SFN2: Spindle frequency in NREM N2, SDN2: Spindle duration in NREM N2, SNN3, SFN3, SDN3: Same for NREM N3, ESS: Epworth Sleepiness Scale, ISI: Insomnia Severity Index CI: Confidence interval data from^{9,12,21,22}

Table 3. Correlations between spindle characteristics and polysomnography indices

Variable 1	Variable 2	ρ	p
SNN2	ODI	0.72	0.007*
SNN2	HR	-0.27	0.02*
SFN2	Mean SpO ₂	0.21	0.014*
SFN3	Hypopnea Index	-0.26	0.04*

* ρ : Pearson's correlation coefficient, $p < 0.05$ after Bonferroni correction.
Nonsignificant correlations (e.g., SNN3 with HR, $p = 0.91$) omitted for brevity.^{12,11}
ODI: Oxygen Desaturation Index, HR: Heart rate, SpO₂: Oxygen saturation, SNN2: Spindle density in NREM N2, SFN2: Spindle frequency in NREM N2

Table 4. Correlations between spindle characteristics and apnea types

Variable 1	Variable 2	ρ	p
SNN2	Obstructive	0.21	0.005*
SDN2	Hypopnea	-0.43	0.01*
SFN3	Obstructive	-0.19	0.04*

* ρ : Pearson's correlation coefficient, $p < 0.05$.
Nonsignificant correlations (e.g., SNN2 with Central, $p = 0.87$) were excluded.²⁰
SDN2: Spindle duration in NREM N2, SNN2: Spindle number in NREM N2, SFN3

N2 frequency (SFN2) correlated with mean SpO₂ ($p = 0.21$, $p = 0.014$). Nonsignificant findings (e.g., N3 correlations, $p > 0.05$) may be due to the limited sample size or stage-specific effects.^{12,27}

Table 4 shows correlations between spindle characteristics and apnea types. NREM N2 spindle density (SNN2) was positively correlated with obstructive apnea ($p = 0.21$, $p = 0.005$), while N2 duration (SDN2) was negatively correlated with hypopnea ($p = -0.43$, $p = 0.01$). N3 frequency (SFN3) was negatively correlated with obstructive apnea ($p = -0.19$, $p = 0.04$).²⁰

Discussion

In this study, NREM N2 sleep spindle frequency and duration decreased significantly with increasing OSA severity, reflecting disrupted thalamocortical function.¹¹ Patients with severe OSA exhibited lower N2 spindle frequency (SFN2=12.96 Hz vs. 13.05 Hz in mild/moderate, $p = 0.01$) and shorter duration (SDN2=0.66 s vs. 0.80 s, $p = 0.029$). This confirms prior observations that spindle activity is reduced in breathing in individuals with sleep disorders.^{27,28} These changes may reflect repeated apneic episodes impairing thalamic reticular nucleus and thalamocortical neuron interactions, which generate spindles.^{10,29} Spindle density during NREM N2 (SNN2) showed a trend towards reduction in cases of severe OSA ($p = 0.06$), aligning with Carvalho et al.'s¹¹ findings of decreased slow spindles in moderate OSA.

The strong correlation between N2 spindle density and oxygen desaturation index (ODI; $p = 0.72$, $p = 0.007$) in this study indicates that spindles may serve as an indicator of hypoxic stress in OSA.³⁰ In addition, the negative correlation with

heart rate (HR; $p = -0.27$, $p = 0.02$) indicates cardiovascular effects on sleep architecture.²⁶ However, NREM N3 spindle characteristics showed no significant differences across severity groups ($p > 0.05$), possibly due to limited spindles in deeper sleep or our small sample size ($n = 37$).^{9,13} This stage-specificity contrasts with Ondze et al.²⁸ findings, who noted spindle reductions in N2 and slow-wave sleep in the breathing of those with mild sleep-disordered.

In the context of the recent literature, our results confirm Himanen et al.²⁷ observation of persistently slow spindle frequency in OSA patients²⁸ and extend this by quantifying duration reductions, a finding consistent with the Yetkin and Aydogan³¹ post-CPAP therapy. Several studies, including Parker et al.³² study, link spindle deficits to cognitive impairments in other disorders, suggesting a parallel mechanism in OSA where hypoxia and arousals disrupt memory consolidation.¹² Unlike some reports of spindle recovery with treatment, our cross-sectional study lacks post-CPAP data, limiting direct comparisons.³¹

Clinically, these findings suggest NREM N2 spindle metrics (frequency, duration, density) could enhance OSA diagnosis as non-invasive, cost-effective markers alongside PSG.³³ A lower SFN2 or SDN2 might indicate severe OSA, guiding treatment prioritization, while longitudinal monitoring of spindle recovery could assess CPAP efficacy.³¹ Statistically, effect sizes (e.g., $d = 0.65$ for SFN2) underscore moderate to substantial differences, though nonsignificant N3 results warrant caution in overgeneralization.¹¹

Study Limitations

Limitations include a small sample size ($n = 37$), which restricts statistical power and generalizability, particularly regarding N3 findings.⁹ Moreover, although the single-scorer manual spindle analysis was blinded, it may have still introduced bias compared to automated methods.¹⁰ Furthermore, limited access to post-treatment information might have limited insights into therapeutic impacts, a gap noted in broader OSA research.²⁶ Nonsignificant correlations (e.g., N3 with ODI, $p > 0.05$) may pinpoint sample constraints or reduced spindle occurrence in deeper sleep, highlighting the need for larger studies.³⁰ Future research should explore automated spindle detection, post-CPAP spindle changes, and their cognitive implications in diverse populations.^{26,31}

Conclusion

In summary, NREM N2 spindle reductions highlight their potential as OSA severity markers, bridging neurophysiological and clinical disciplines. These findings contribute to understanding sleep architecture disruption in OSA and suggest practical diagnostic applications, which require validation in studies with larger cohorts.

Ethics

Ethics Committee Approval: The study was approved by the Ethics Committee of Qazvin University of Medical Sciences

(approval number: IR.QUMS.REC.1397.103, date: 05.06.2018).

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Footnotes

Authorship Contributions

Surgical and Medical Practices: A.A.S., Concept: Z.Y., A.H.S.J., S.A., M.S., S.J., A.A.S., Design: Z.Y., A.H.S.J., S.A., A.A.S., Data Collection or Processing: Z.Y., A.H.S.J., A.A.S., Analysis or Interpretation: Z.Y., A.A.S., Literature Search: Z.Y., A.H.S.J., S.A., M.S., S.J., A.A.S., Writing: Z.Y., A.H.S.J., A.A.S.

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Workaholism and Sleep Disorders in Employees: The Moderator Roles of Workaholism in the Relationships between Insomnia and Affective Symptoms

Çalışanlarda İşkoliklik ve Uyku Bozuklukları: Uykusuzluk ve Afektif Belirtiler Arasındaki İlişkilerde İşkolikliğin Düzenleyici Rolü

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Abstract

Objective: Sleep disorders are a growing concern in occupational health due to their strong associations with emotional distress and impaired functioning. Employees with severe insomnia symptoms are at increased risk for affective symptoms such as anxiety and depression. However, this relationship may vary depending on work-related behavioral patterns. Workaholism, a compulsive drive to work excessively, may act as a moderator, intensifying the impact of sleep disturbances on affective symptoms and vice versa. This study aimed to assess sleep disorder risk among employees, examine differences in the severity of sleep disorders based on workaholism levels, and investigate the moderating roles of workaholism in the relationships between insomnia severity and affective symptoms.

Materials and Methods: The sample consisted of 459 day-working employees (68.41% female, $M_{age}=41.14$, standard deviation =10.90) who completed measures of demographics, workaholism, and sleep disorders.

Results: Results showed that 40.31% were at risk for at least one sleep disorder, and 28.98% for multiple. Compared to employees with lower workaholism, those with higher workaholism had significantly higher scores of breathing-related sleep disorder, insomnia, narcolepsy, restless legs/periodic limb movement disorder, and circadian rhythm sleep disorder. Moderation analysis revealed that workaholism significantly moderated the relationship between insomnia severity and affective symptoms, but not vice versa. As workaholism increased, the relationship between insomnia severity and affective symptoms became stronger.

Conclusion: These findings suggest a high prevalence of sleep disorders among employees and that workaholism can exacerbate the affective burden of insomnia. Targeted interventions addressing both sleep

Öz

Amaç: Uyku bozuklukları duygusal zorluk ve işlevsellikteki bozulmalar ile güçlü ilişkileri nedeniyle iş sağlığı alanında giderek artan bir endişe kaynağına dönüşmüştür. Şiddetli uykusuzluk çeken çalışanlar, anksiyete ve depresyon gibi afektif belirtiler açısından yüksek risk altındadır. Ancak, bu etkilerin işle ilgili davranışsal örüntülere bağlı olarak değişebileceği söylenebilir. Bu bağlamda, aşırı çalışma dürtüsü ile karakterize olan işkoliklik, uykusuzluğun afektif belirtiler ve afektif belirtilerin uykusuzluk üzerindeki etkilerini potansiyel olarak yoğunlaştıran bir düzenleyici değişken olarak işlev görebilir. Bu çalışmanın amacı, çalışanlar arasında uyku bozukluğu riskini değerlendirmek, işkoliklik düzeylerine göre uyku bozuklukları şiddetindeki farklılıkları incelemek ve uykusuzluk şiddeti ve afektif belirtiler arasındaki ilişkilerde işkolikliğin düzenleyici rolünü araştırmaktır.

Gereç ve Yöntem: Çalışmanın örneklemini gün içinde çalışan ve demografik özellikler, işkoliklik ve uyku bozukluğu ölçeklerini dolduran 459 kişiden (%68,41 kadın, ortalama yaş =41,14, standart sapma =10,90) oluşmaktadır.

Bulgular: Sonuçlar, çalışanların %40,31'inin en az bir uyku bozukluğu, %28,98'inin ise birden fazla uyku bozukluğu açısından risk altında olduğunu göstermiştir. İşkolikliği düşük olanlara kıyasla yüksek olan bireylerde solunumla ilişkili uyku bozukluğu, uykusuzluk, narkolepsi, huzursuz bacak/periodyodik uzuv hareketi bozukluğu ve sirkadiyen ritim uyku bozukluğu puanları anlamlı derecede yüksek bulunmuştur. Düzenleyici değişken analizi, işkolikliğin uykusuzluk şiddeti ile afektif belirtiler arasındaki ilişkiyi anlamlı olarak düzenlediğini ancak afektif belirtiler ile uykusuzluk şiddeti arasındaki ilişkiyi düzenlemediğini göstermiştir. İşkoliklik artıkça uykusuzluk şiddeti ile afektif belirtiler arasındaki ilişki güçlenmektedir.

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health and workaholism may be critical for improving employee well-being.

Keywords: Sleep disorders, insomnia, affective symptoms, workaholism, moderation

Sonuç: Bu bulgular, çalışanlar arasında uyku bozukluğu yaygınlığının yüksek olabileceğine işaret etmekte ve işkolikliğin uykusuzluğun afektif yükünü daha da ağırlaştırabileceğini ortaya koymaktadır. Hem uyku sağlığını hem de işkolikliği hedef alan müdahalelerin, çalışanların refahını artırmak için kritik öneme sahip olduğu görülmektedir.

Anahtar Kelimeler: Uyku bozuklukları, uykusuzluk, afektif belirtiler, işkoliklik, düzenleyici

Introduction

Insufficient sleep results a to wide range of adverse outcomes affecting cognitive functioning, mood regulation, neurobehavioral performance, physiological health, and overall disease risk, thereby making it a significant public health concern.¹ Disruptions in sleep duration, timing, or quality may stem from various causes, among which sleep disorders represent one of the most substantial contributors.² Sleep disorders can manifest as difficulties initiating or maintaining sleep, excessive daytime sleepiness, or unusual movements while sleep.³ Given their diverse and serious consequences, identifying and addressing sleep disorders is essential for promoting overall health and well-being across multiple domains, including the workplace.

Modern work environments, characterized by high demands and ever-increasing pressures, are growingly impacted by health-related challenges, such as sleep disorders, which extend well beyond traditional physical illnesses.^{4,5} The findings of 2008 Sleep in America Poll (Sleep, Performance and the Workplace) reported that 37% of employees working 30 hours or more were at-risk for at least one disorder, while 9.6% of them were as at-risk for multiple sleep disorders.⁶ Similarly, a nationwide study of Turkish adult workforce by Firat et al.⁷ found the prevalence rates of parasomnia, poor sleep, obstructive sleep apnea, insomnia, excessive sleepiness, and restless leg syndrome as 19.2%, 17.0%, 9.1%, 6.1%, 5.2%, and 2.7%, respectively. In occupational settings, sleep disturbances have been consistently linked to impaired performance outcomes such as increased presenteeism, absenteeism, and occupational accidents with the associated economic burden estimated to range from US \$322 (in 1995) to US \$1,967 (in 2010) per employee.⁸ Likewise, poor sleep quality was a risk factor for absenteeism, while poor sleep quality, excessive sleepiness, and parasomnia were risk factors for delay to work in the study by Firat et al.⁷ Among sleep disorders, insomnia have emerged as critical public health problem^{9,10} impacting workplace settings.¹¹

The most recent research defines insomnia as a sleep disorder characterized by difficulties in initiating and sustaining sleep, as well as experiencing early morning awakenings.¹² An estimated 10% of adults meet the diagnostic criteria for insomnia disorder, while another 20% report experiencing intermittent symptoms of insomnia.⁹ Although often situational or episodic, insomnia becomes chronic in over half of those affected, particularly those with heightened vulnerability following initial episodes triggered by stress, health issues, irregular schedules, or jet lag.¹³ Insomnia may also increase the risk of various adverse health outcomes, such as physical illnesses (e.g.,

cardiovascular problems and metabolic disorders) and mental disorders (e.g., major depression and anxiety disorders).¹⁴ Furthermore, in workplace settings, insomnia significantly impairs occupational functioning and productivity, increases absenteeism and the risk of accidents and long-term disability, and imposes substantial direct and indirect economic burdens on individuals and the healthcare system.^{7,9,11,15,16} Given the wide-ranging impacts of insomnia on health and workplace functioning, it is important to consider psychosocial factors, such as workaholism, that may exacerbate or sustain insomnia and sleep problems.

The concept of workaholism, defined as an uncontrollable and excessive commitment to work, has garnered attention as a significant workplace problem.¹⁷ Over the past two decades, research has increasingly conceptualized workaholism negatively, as an addiction characterized by a compulsive motivation to work and excessive cognitive and time investment.¹⁸ A recent meta-analysis, including data from 23 countries, found a 14.1% prevalence rate for workaholism.¹⁹ Although workaholism is often socially endorsed in high-achievement contexts, its underlying compulsive nature is associated with elevated levels of stress, burnout, and negative mood states.²⁰ Previous research has consistently linked workaholism to a range of negative outcomes, including impaired physical and mental health, reduced life satisfaction, strained interpersonal relationships, and adverse organizational consequences.¹⁹ A number of studies have also revealed that higher levels of workaholism are associated with more sleep problems.²¹⁻²⁴ In one study examining different types of workers (workaholics, positive workers, compulsive workers, and hard workers), workaholics were shown to have significantly more sleep disturbances, including morning fatigue, falling asleep while driving, and reduced sleep duration during both weekdays and weekends, compared to other types of workers.²⁵ Using a longitudinal design covering 10 work days, Menghini and Balducci²⁶ found that employees experienced increased systolic and diastolic blood pressure, heightened emotional exhaustion, and greater sleep disturbances on workdays with higher symptoms of workaholism.

It can be argued that workaholism contributes to sleep problems through behavioral, cognitive, and physiological pathways. Working excessively increases job demands and reduces recovery time, resulting in physical and emotional exhaustion that disrupts sleep.^{22,25} In addition, compulsive work-related thoughts cause heightened arousal and stress, making it difficult to fall asleep or stay asleep.²² Finally, chronic stress from workaholism may also dysregulate the hypothalamic-pituitary-adrenal axis, a key stress-response system, resulting

in prolonged physiological arousal that interferes with sleep quality and increases sleep latency.^{23,25}

Given that affective symptoms of depression and anxiety are reciprocally associated with insomnia^{27,28} and that workaholism is significantly associated with a range of psychiatric disorders,²⁹ it is possible to argue that workaholism may moderate the relationship between insomnia severity and affective symptoms. It was hypothesized that when stress and burnout caused by workaholism interact with negative affect and emotion dysregulation resulting from insomnia, these factors may exacerbate affective consequences of insomnia. Similarly, when stress and burnout caused by workaholism interact with affective symptoms such as anxiety and depression, these factors may amplify the effects of affective symptoms on insomnia. These bidirectional interactions may result in a vicious cycle of worsening insomnia and affective symptoms in employees with high levels of workaholism.

In summary, the present study had three primary aims. The initial aim was to screen for sleep disorders risk among Turkish employees, as sleep disorders are prevalent and have a substantial impact on both work performance and the well-being of employees. The second aim was to investigate differences in the severity of sleep disorders among employees based on their workaholism scores. The third aim was to examine the moderator role of workaholism in the relationships between insomnia severity-affective symptoms and affective symptoms-insomnia severity. The moderation models were depicted in Figure 1.

The specific hypotheses regarding the differences in the severity of sleep disorders among employees based on their workaholism scores were:

1. Individuals with high workaholism would have statistically higher breathing-related sleep disorders scores than individuals with low workaholism.
2. Individuals with high workaholism would have statistically higher insomnia scores than individuals with low workaholism.
3. Individuals with high workaholism would have statistically higher narcolepsy scores than individuals with low workaholism.
4. Individuals with high workaholism would have statistically higher restless legs/periodic limb movement disorder scores than individuals with low workaholism.
5. Individuals with high workaholism would have statistically higher circadian rhythm sleep disorder scores than individuals with low workaholism.
6. Individuals with high workaholism would have statistically higher sleepwalking scores than individuals with low workaholism.
7. Individuals with high workaholism would have statistically higher nightmare disorder scores than individuals with low workaholism.
8. Individuals with high workaholism would have statistically higher impact of sleep complaints on daily functioning scores than individuals with low workaholism.

The specific hypotheses regarding the moderator role of workaholism in the relationship between insomnia severity and affective symptoms were:

9. Higher insomnia severity would predict higher affective symptoms.

10. Higher workaholism would predict higher affective symptoms.

11. Higher interaction of insomnia severity and workaholism would predict higher affective symptoms.

Finally, the specific hypotheses regarding the moderator role of workaholism in the relationship between affective symptoms and insomnia severity were:

12. Higher affective symptoms would predict higher insomnia severity.

13. Higher workaholism would predict higher insomnia severity.

14. Higher interaction of affective symptoms and workaholism would predict higher insomnia severity.

Materials and Methods

Participants

Data for the present study were collected from 459 employees who were working during the day and were over the age of 18. They were recruited using a convenience sampling method via the internet and social media. The mean age of the sample was 41.14 (standard deviation (SD) =10.90). Three hundred fourteen participants (68.41%) identified as female, while 145 participants (31.59%) identified as male. Regarding marital status, 100 participants (21.79%) were single and not in a relationship, 49 participants (10.6%) were single but in a relationship, and 310 participants (67.5%) were married. Only 40 participants (8.7%) were students. Finally, 315 participants (68.6%) reported having a moderate-income level. The demographic details and sleep-related behaviors of the sample can be seen in Table 1.

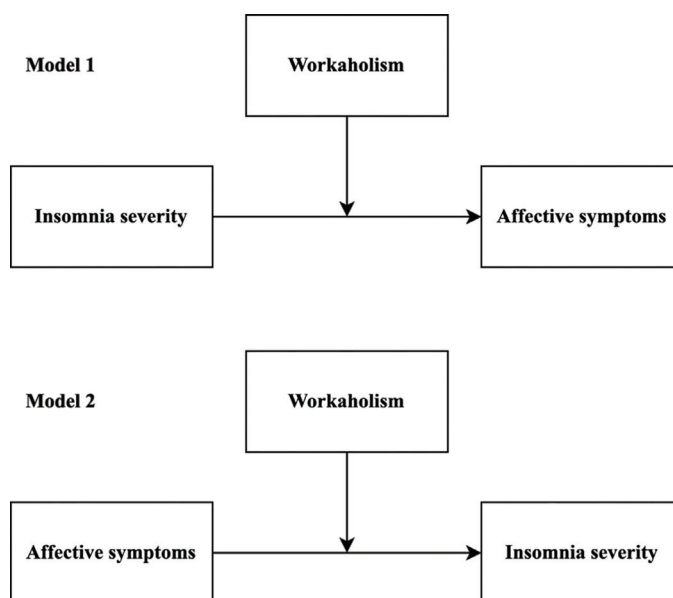


Figure 1. The hypothesized moderation models

Table 1. The demographic details of the sample		
	M	SD
Age	40.14	10.90
Work experience (years)	16.21	11.08
Black tea after 18:00 (cup)	1.77	1.73
Coffee after 18:00 (cup)	0.52	0.68
Gender	n	%
Female	314	68.41
Male	145	31.59
Student		
Yes	40	8.71
No	419	91.29
Education		
Primary school	1	0.21
Secondary school	3	0.65
High school	18	3.92
Associate's degree	16	3.48
Bachelor's degree	296	64.48
Master's degree	105	22.87
Doctorate degree	20	4.35
Marital status		
Single/no relationship	100	21.78
Single/in a relationship	49	10.67
Married	310	67.53
Perceived socio-economic status		
Very low	6	1.30
Low	69	15.03
Medium	315	68.62
High	67	14.59
Very high	2	0.43
Smoking		
No	286	62.31
Yes	173	37.69
Exercise frequency (day)		
0	206	44.88
1	57	12.42
2	75	16.34
3	80	17.43
4	13	2.83
5	18	3.92
6	2	0.44
7	8	1.74
Bedtime screen time (minutes)		
0	134	29.19
10	87	18.95
20	64	13.94
30	93	20.26
40	17	3.70
50	10	2.18
60	39	8.50
>60	15	3.28

SD: Standard deviation, M: Mean

Demographic Information Form

A demographic information form was utilized to gather data pertaining to the characteristics of the participants, including their age, gender, student status, educational level, work experience, marital status, perceived socioeconomic status, smoking status, consumption of black tea and coffee after 18:00, exercise frequency, and bedtime screen time.

DUWAS Workaholism Scale

The DUWAS Workaholism Scale was developed by Schaufeli et al.³⁰ by combining items of the Work Addiction Risk test³¹ and the Workaholism Battery³². The scale had a two-factor structure (working excessively and working compulsively) with 17 items. The items are rated on a 5-point likert type scale (1 =totally disagree, 5 =totally agree). Higher scores on the subscales indicate a higher level of working excessively or working compulsively. Sample items from the scale include "I seem to be in a hurry and racing against the clock" and "I feel obliged to work hard, even when it is not enjoyable". The scale was adapted into Turkish by Doğan and Tel.³³ The Turkish version also had a two-factor structure and the Cronbach's alpha coefficients were 0.85 for total scale, 0.76 for working excessively, and 0.74 for working compulsively. In the present study, the DUWAS Workaholism Scale was used to assess workaholism levels of the employees and the Cronbach's alpha coefficient was 0.91.

SLEEP-50 Questionnaire

The SLEEP-50 questionnaire was developed by Spoormaker et al.³⁴ to detect sleep disorders listed in the DSM-IV-TR. The questionnaire includes 52 items evaluating breathing-related sleep disorders, insomnia, narcolepsy, restless legs/periodic limb movement disorder, circadian rhythm sleep disorder, sleepwalking, nightmares, factors influencing sleep, the impact of sleep complaints on daily functioning, subjective sleep quality, and subjective sleep duration. The items are rated on a 4-point likert scale 1 =not at all, 4 =very much. Higher scores on the subscales indicate higher severity of the sleep problem. Sample items from the questionnaire include "I often snore loudly at night", "I have difficulty falling asleep at night", "I find myself falling asleep unexpectedly during the day", "I feel the need to move my legs while trying to sleep", "I go to bed at very different times", "I sometimes walk around while I'm asleep", "I frequently wake up from unsettling dreams", "I drink alcoholic beverages during the evening", and "I feel tired at getting up".

The cut-off values assessing sleep disorders were: a score of 15 and more from breathing-related sleep disorders subscale, a score of 19 and more from the insomnia subscale, a score of 7 and more from the narcolepsy subscale, a score of 7 and more from the restless leg/periodic limb movement disorder subscale, a score of 8 from the circadian rhythm sleep disorder subscale, a score of 7 and more from the sleepwalking subscale, and a score of 9 and more from the nightmare disorder subscale. In addition, a score of 15 and more from the impact of sleep complaints on daily functioning subscale is required to detect any sleep disorder. The questionnaire also provides scores

for affective disorder (measured by four items pertaining to depression and anxiety symptoms), sleep state misperception, and hypersomnia. The questionnaire was adapted to Turkish by Yildirim et al.³⁵ The Cronbach's alpha coefficients and test-retest correlations of the subscales were between 0.52 and 0.83, and 0.51 and 0.80, respectively. In the present study, the SLEEP-50 questionnaire was used to detect probable sleep disorders in the sample and assess affective symptoms (measured by the affective disorder subscale) and insomnia severity. Cronbach's alpha coefficients of the subscales were 0.73 for breathing-related sleep disorder, 0.85 for insomnia, 0.68 for narcolepsy, 0.80 for restless legs/periodic limb movement disorder, 0.65 for circadian rhythm sleep disorder, 0.80 for sleepwalking, 0.53 for nightmare, 0.85 for the impact of sleep complaints on daily functioning, and 0.79 for affective disorder.

Procedure

The ethical approval of the present study was granted by Atılım University Human Research Ethics Committee (date: 07.06.2024, approval number: 88177). The participants were recruited using convenience sampling. Data for the present study was collected online between June and September 2024 via the SurveyMonkey platform. All participants were provided with an overview of the study's objectives, information on the voluntary nature of their participation, their right to withdraw from the study, and specific details regarding how their information would be used for research purposes. They were also assured that their data would be kept completely confidential. Their consent was obtained through the informed consent form. The duration of the study was approximately 15 minutes.

Statistical Analysis

The analyses for the present study were conducted using the SPSS 25 (for data editing and moderation analyses) and JASP 19.3.0 (for correlation analysis, reliability coefficients and standardized moderation estimates). The relationship between study variables was examined with Pearson correlations. The severity of sleep disorders based on workaholism levels (categorized as low and high based on the mean) was assessed using Student and Welch's t-tests and while homogeneity of variances was examined by Levene's test. The assumptions were evaluated for linearity (scatter plots), univariate normality [skewness (-2.0,

2.0) and kurtosis (-7.0, 7.0) values], multivariate normality (Mardia's test), homoscedasticity (residuals vs. predicted plot), and multicollinearity [tolerance (>0.01) and variance inflation factor (<10) values]. PROCESS macro was used to examine the moderator role of workaholism in the relationship between insomnia severity-affective symptoms and affective symptoms-insomnia severity.³⁶ To assess the moderation effect, the required sample size to reach 0.80 statistical power was calculated as 395 with parameters of small effect size ($f^2=0.02$), a single tested predictor and a total of three predictors. The reliabilities of the scales used were examined with Cronbach's alpha coefficients.

Results

Sleep Descriptives Based on SLEEP-50 Questionnaire

Based on the subscale-specific cut-off values and a score of 15 and more on the impact of sleep complaints on daily functioning subscale, 13.07% of the sample had high risk for breathing-related sleep disorders, 20.48% for insomnia, 0.44% for sleep state misperception, 32.68% for narcolepsy, 21.35% for restless legs/periodic limb movement disorder, 2.61% for circadian rhythm sleep disorder, 0.65% for sleepwalking, 1.09% for nightmare disorder, and 4.58% for hypersomnia. The majority of the participants were free from poor sleep practices, such as too light/too noisy bedroom, alcohol or smoke at night, and the use of sleep/other medications. The details of sleep descriptives are shown in Table 2.

Differences Between Sleep Disorder Severities Based on Workaholism Levels

Four students and three Welch t-tests (in cases of inequality of variances) were conducted. Compared to the low workaholism group, the high workaholism group had significantly higher scores of breathing-related sleep disorder, $t(457)=2.85$, $p<0.001$, $d=0.27$; insomnia, $t(451.32)=4.46$, $p<0.001$, $d=0.42$, narcolepsy, $t(457)=2.47$, $p<0.001$, $d=0.23$; restless legs/periodic limb movement disorder, $t(444.78)=4.72$, $p<0.001$, $d=0.44$, and circadian rhythm sleep disorder, $t(457)=2.53$, $p<0.001$, $d=0.24$. Finally, the high workaholism group ($m=15.35$, $SD=4.15$) had significantly higher scores on the impact of sleep complaints on daily functioning than the low workaholism group ($m=13.66$, $SD=3.73$), $t(457)=4.58$, $p<0.001$, $d=0.43$. The details of the comparisons can be seen in Table 3.

Table 2. The risks for sleep disorders and poor sleep practices of the sample based on the SLEEP-50 Questionnaire (n=459)

	n	%
Sleep disorders		
Breathing-related sleep disorders	60	13.07
Insomnia	94	20.48
Sleep state misperception	2	0.44
Narcolepsy	150	32.68
Restless legs/periodic limb movement disorder	98	21.35
Circadian rhythm sleep disorder	12	2.61
Sleepwalking	3	0.65
Nightmares	5	1.09

Table 2. Continued

	n	%		
Hypersomnia	21	4.58		
Any sleep disorder	185	40.31		
More than one sleep disorders	113	28.98		
	(Not at all =0)	(A little =1)	(Rather much =2)	(Very much =3)
Poor sleep practices				
Bedroom condition - too light	357 (77.79%)	87 (18.95%)	15 (3.27%)	0 (0.00%)
Bedroom condition - too noisy	392 (85.40%)	62 (13.51%)	5 (1.09%)	0 (0.00%)
Alcohol at night	274 (59.70%)	169 (36.82%)	14 (3.05%)	2 (0.44%)
Smoke at night	294 (64.05%)	83 (18.08%)	66 (14.38%)	16 (3.49%)
Sleep/other medication	421 (91.72%)	34 (7.41%)	0 (0.00%)	0 (0.00%)

Table 3. The differences in sleep disorders severity based on workaholism levels (n=459)

Sleep disorder	Test	High workaholism M (SD)	Low workaholism M (SD)	t	df	Cohen's d
Breathing-related sleep disorders	Student	13.12 (3.28)	12.27 (3.04)	2.85**	457.00	0.27
Insomnia	Welch	17.80 (4.77)	15.94 (4.13)	4.46***	451.32	0.42
Narcolepsy	Student	7.62 (2.11)	7.11 (2.29)	2.47*	457.00	0.23
Restless legs/periodic limb movement disorder	Welch	6.60 (2.44)	5.62 (2.00)	4.72***	444.78	0.44
Circadian rhythm sleep disorder	Student	4.99 (1.44)	4.66 (1.41)	2.53*	457.00	0.24
Sleepwalking	Welch	3.16 (0.47)	3.26 (1.06)	-1.33	307.41	-0.13
Nightmare disorder†	Student	10.86 (1.92)	10.46 (1.63)	1.89	294.00	0.22
Impact	Student	15.35 (4.15)	13.66 (3.77)	4.58***	457.00	0.43

*p<0.05, **p <0.01, ***p<0.001. †n=296
SD: Standard deviation

Correlations Between The Study Variables

The results showed that workaholism had small to moderate correlations with subjective sleep quality ($r=-0.22$, $p<0.001$), subjective sleep duration ($r=-0.13$, $p<0.001$), insomnia severity ($r=0.29$, $p<0.001$), and affective symptoms ($r=0.35$, $p<0.001$). Employees with higher levels of workaholism were more likely to have lower sleep quality, shorter sleep duration, and higher insomnia severity and affective symptoms. Moreover, there was a strong correlation ($r=0.68$, $p<0.001$) between insomnia severity and affective symptoms, indicating that employees with higher insomnia severity also tend to have higher affective symptoms. The correlations between the variables can be seen in Table 4.

Moderation Analysis

Two moderator analyses were conducted to assess the moderator roles of workaholism in the relationship between insomnia severity-affective symptoms and affective symptoms-insomnia severity. All assumptions of linear regression (i.e., linearity, normality, homoscedasticity, and multicollinearity) were met for both analyses. Despite being the subscales of the SLEEP-50, insomnia severity and affective symptoms were not highly correlated ($r>0.80$), which did not lead to a problem of discriminative validity or multicollinearity.³⁷ In the

analysis regarding the insomnia severity-affective symptoms, ten multivariate outliers identified using Mahalanobis distance were excluded from further analyses, resulting in a final sample of 449 participants. The first model accounted for 47.30% of the variance in affective symptoms, $F(3,445)=133.28$, $p<0.001$. Insomnia severity [$\beta=0.59$, $B=0.31$, standard error (SE) =0.02, $t(445)=16.16$, $p<0.001$, 95% confidence interval (CI) (0.27, 0.34)] and workaholism [$\beta=0.18$, $B=0.04$, $SE=0.01$, $t(454)=5.15$, $p<0.001$, 95% CI (0.03, 0.06)], and the interaction of insomnia severity and workaholism [$\beta=0.13$, $B=0.00$, $SE=0.00$, $t(445)=3.49$, $p<0.001$, 95% CI (0.00, 0.01)] were significant predictors of affective symptoms. The moderation model 1 can be seen in Figure 2. The conditional effect of workaholism in the relationship between insomnia severity and affective symptoms was significant, $\Delta R^2=0.01$, $F(1, 445)=12.20$, $p<0.01$, suggesting that workaholism moderated the relationship. The interactions were probed using the pick-a-point method for workaholism (-1 SD, mean, and 1 SD); however, no statistical significance transition points were found within the observed range of the moderator variable found using the Johnson-Neyman method. Conditional effects of workaholism on the relationship between insomnia severity and affective symptoms can be seen in Table 5.

Table 4. The correlations between subjective sleep quality, sleep duration, insomnia severity, workaholism, and affective symptoms

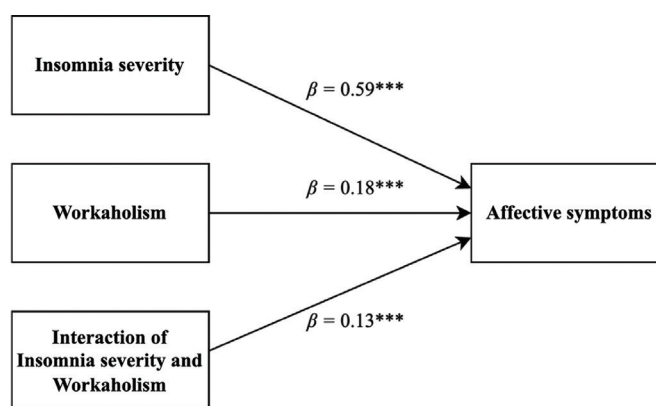
Variables	1	2	3	4	5
(1) Subjective sleep quality	-				
(2) Subjective sleep duration (hours)	0.42***	-			
(3) Insomnia severity	-0.61***	-0.34***	-		
(4) Workaholism	-0.22***	-0.13**	0.29***	-	
(5) Affective symptoms	-0.56***	-0.29***	0.68***	0.35***	-
M	6.48	6.99	16.89	45.79	7.93
SD	2.10	1.10	4.56	10.36	2.31
Minimum	0.00	3.00	8.00	14.00	4.00
Maximum	10.00	10.00	32.00	70.00	16.00

** p<0.01, *** p<0.001
SD: Standard deviation, M: Mean

In the analysis regarding the affective symptoms-insomnia severity, three multivariate outliers identified using Mahalanobis distance were excluded from further analyses, resulting in a final sample of 456 participants. The second model accounted for 46.8% of the variance in insomnia severity, $F(3, 452) = 132.41, p < 0.001$. Only affective symptoms [$\beta = 0.66, B = 1.32, SE = 0.08, t(452) = 16.78, p < 0.001, 95\% CI (1.16, 1.47)$] was a significant predictor of insomnia severity. The conditional effect of workaholism in the relationship between affective symptoms and insomnia was not significant, $\Delta R^2 = 00, F(1, 452) = 0.10, p = 0.75$, meaning that workaholism did not moderate the relationship. The moderation model 2 can be seen in Figure 3.

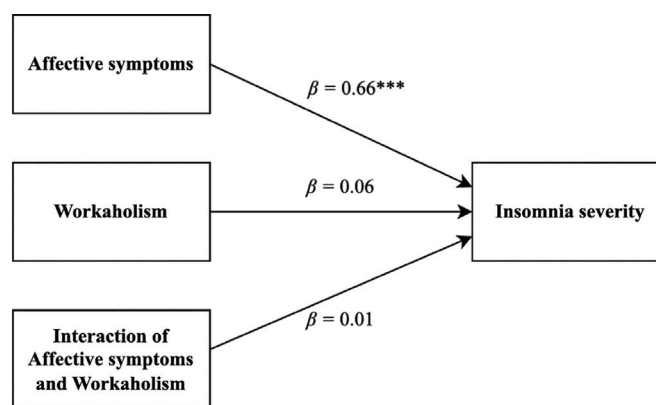
Discussion

The present study examined the risk of sleep disorders among employees, the differences in the severity of sleep disorders based on workaholism levels, and the moderator role of workaholism in the relationships between insomnia severity-affective symptoms and affective symptoms-insomnia severity. The results indicated that 40.31% of the employees were at risk for at least one sleep disorder, while 28.98% of them were at risk for more than one. Comparing the present findings with the results of the 2008 American Sleep Poll⁶ and the study by Firat et al.⁷, the risk rate for any single sleep disorder was found to be similar; however, the prevalence of employees at risk for more than one sleep disorder was notably higher in the present study. This discrepancy may stem from methodological differences, particularly due to the tools used to assess sleep disorder risk. The present study used the SLEEP-50, while the 2008 American Sleep Poll⁶ and Firat et al.⁷'s studies utilized several scales such as the Epworth Sleepiness Scale or the STOP Questionnaire. Similarly, compared to the studies using the SLEEP-50 with college and medical students^{35,38,39}, we found different percentages for probable sleep disorder caseness in all sleep disorders covered. This difference could be due to the nature of the samples used by the studies. For example, the sample of Yildirim et al.³⁵ was affected by previous earthquakes



Note. ***p < .001

Figure 2. The moderator role of workaholism in the relationship between insomnia severity and affective symptoms



Note. ***p < .001

Figure 3. The moderator role of workaholism in the relationship between affective symptoms and insomnia severity

Table 5. The conditional relationship between insomnia severity and affective symptoms at values of workaholism

Workaholism	β	B	SE	t	p	LLCI	ULCI
-1 SD	0.45	0.24	0.03	8.25	<0.001	0.18	0.30
Mean	0.59	0.31	0.02	16.16	<0.001	0.27	0.34
1 SD	0.71	0.37	0.02	15.51	<0.001	0.32	0.42

SD: Standard deviation, SE: Standard error, LLCI: Lower limit confidence interval, ULCI: Upper limit confidence interval

in the Van region and increased post-traumatic stress disorder rates.³⁵

Regarding the differences in sleep disorder scores based on workaholism levels, employees with higher workaholism levels had significantly higher scores for breathing-related sleep disorder, insomnia, narcolepsy, restless legs/periodic limb movement disorder, circadian rhythm sleep disorder and the impact of sleep problems on daily functioning but not for sleepwalking and nightmare disorder, supporting the hypotheses 1, 2, 3, 4, 5, and 8. The insignificant results for sleepwalking and nightmare disorder scores may be due to the low number of these cases in our sample. This broad spectrum of sleep pathology in the employees suggests that workaholism may interfere with general sleep quality and disrupt multiple physiological and behavioral components of the sleep system. Workaholics' long working hours and compulsive behavior may lead to less recovery time and increased stress, which in turn contribute to sleep problems.²⁵ Moreover, higher workaholism was associated with poor sleep quality, shorter sleep duration, higher insomnia severity, and increased affective symptoms. It is possible that workaholics' persistent thoughts about work, even when trying to rest, may lead to increased sympathetic arousal and cognitive activation, making it harder to fall asleep.^{22,23,25} Overall, the findings supported previous studies indicating higher sleep disturbance severity in workaholics and associations between workaholism and sleep problems.²¹⁻²⁶

The moderator analyses revealed that workaholism moderated the relationship between insomnia severity-affective symptoms, but not the relationship between affective symptoms-insomnia severity, partially supporting our hypotheses. In the first model, both insomnia severity and workaholism were significant predictors of affective symptoms which are in align with previous research^{20,27,29,40} and supported the hypotheses 9 and 10. Insomnia may contribute to increased depressive and anxiety symptoms by disrupting emotional regulation, heightening negative emotionality, and impairing cognitive and physiological functioning, particularly through daytime distress and hyperarousal.^{27,41} Similarly, workaholism may lead to higher depressive and anxiety symptoms by promoting chronic stress, emotional avoidance, and compulsive overworking that disrupts psychological well-being.²⁹ Moreover, the association between insomnia severity and affective symptoms was stronger in higher levels of workaholism, supporting the hypothesis 11. As insomnia severity increased, employees with higher levels of workaholism experienced a more pronounced increase in

affective symptoms compared to those with lower levels of workaholism. In other words, workaholism may amplify the adverse affective consequences of insomnia. This finding could be explained by the fact that the added stress or the impaired psychological capital with excessive work behaviors^{42,43} may exacerbate the negative affective impact of insomnia severity. In the second model, affective symptoms significantly predicted insomnia severity, but workaholism and the interaction of affective symptoms and workaholism did not, supporting only the hypothesis 12. There could be several explanations for these findings. Regarding the main effect of workaholism, it is possible that workaholism may lead to stress or burn-out, but these effects may already be mediated by affective symptoms in the model, leaving affective symptoms as the stronger independent predictor. Therefore, future studies could examine a model in which affective symptoms mediate the relationship between workaholism and insomnia severity. Furthermore, regarding the moderator role of workaholism in the relationship between affective symptoms-insomnia severity, it may be possible that the relationship is relatively stable and resistant to modulation by behavioral and cognitive tendencies associated with workaholism, such as compulsive drive, intrusive work-related thoughts, excessive work investment, and difficulty detaching from work.

These insights have practical implications for workplace interventions aimed at promoting employee health. To improve sleep health, workplaces can introduce sleep education programs that raise awareness about the importance of sleep hygiene, screen for common sleep disorders, and provide guidance on healthy sleep practices.⁴⁴ Offering flexible work hours and limiting after-hours communication may also support employees in maintaining consistent sleep schedules. Workaholism, functioning as an internal stressor akin to external job demands, may contribute to emotional exhaustion, but psychological detachment can serve as a protective factor by facilitating recovery and mitigating the distress associated with prolonged work-related effort.²⁶ Organizations might consider implementing strategies to reduce workaholic behaviors, such as promoting work-life balance, encouraging regular breaks, and fostering a culture that values rest and recovery.^{18,45} Additionally, providing resources for stress management and mental health support, such as meditation awareness training and counseling based on self-validation, can help mitigate the affective consequences associated with sleep disorders in workaholic individuals.^{18,46}

Study Limitations

Several limitations of the present study should be acknowledged. Firstly, the sample had a gender imbalance, with fewer male participants than female participants. Additionally, the study only included employees working during the daytime. Future research should aim to recruit more balanced samples in terms of both gender and work schedules to improve representativeness. Secondly, the reliance on convenience sampling may have led to selection bias, which constrains the generalizability of the findings to broader populations. Thirdly, the cross-sectional design of the study provides only a snapshot of the data, making it difficult to draw conclusions about causal relationships or developmental trajectories. To address this, future studies would benefit from employing experimental and longitudinal designs to more rigorously examine the associations among workaholism, affective symptoms, and insomnia severity. Fourthly, we used the affective disorders subscale of the SLEEP-50, which included only four items to measure symptoms of anxiety and depression. In future studies, more comprehensive measures such as the Beck Depression Inventory or the Beck Anxiety Inventory may be utilized to assess affective symptoms. Finally, the SLEEP-50 Questionnaire, based on the DSM-IV-TR, was used to screen for sleep disorders in this study. The SLEEP-50 provided relatively high-risk rates for some sleep disorders, such as narcolepsy and restless legs/periodic limb movement disorder. The items in the narcolepsy subscale largely coincide with symptoms of hypersomnolence. These symptoms are not unique to narcolepsy and can also be present in conditions such as sleep deprivation, stress, depression, and work-related fatigue. There is a strong need for further assessment of the psychometric properties of the Turkish version of the SLEEP-50, particularly by addressing limitations of the study by Yildirim et al.³⁵ Future research could incorporate diagnostic clinical interviews to provide a more accurate identification of individuals with sleep disorders rather than using self-report measures.

Conclusion

In conclusion, the present study showed that employees may be at significant risk for sleep disorders and employees with higher levels of workaholism have higher scores for breathing-related sleep disorders, insomnia, narcolepsy, restless legs/periodic limb movement disorder, circadian rhythm sleep disorder and the impact of sleep problems on daily functioning. Furthermore, it was found that workaholism can exacerbate affective symptoms associated with insomnia. By addressing the relationship between workaholism, sleep health, and emotional well-being, employers can enhance overall employee productivity and reduce the long-term risk of burn-out.

Ethics

Ethics Committee Approval: The ethical approval of the present study was granted by Atılım University Human Research Ethics Committee (date: 07.06.2024, approval number: 88177).

Informed Consent: Informed consent was obtained from all individual participants included in the study.

Footnotes

Authorship Contributions

Concept: Y.N., K.K.T., Design: Y.N., K.K.T., Data Collection or Processing: Y.N., K.K.T., Analysis or Interpretation: Y.N., K.K.T., Literature Search: Y.N., K.K.T., Writing: Y.N., K.K.T.

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Chronotype, Insomnia, and ADHD: A Discussion on the Confounding Effect of Shift Work in Medical Students

Kronotip, İnsomnia ve DEHB: Tıp Fakültesi Öğrencilerinde Vardiyalı Çalışmanın Karıştırıcı Etkisine Dair Bir Tartışma

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Dear Editor,

I was intrigued by the article by Kocakaya and Öztürk, titled “Do Chronotype and Insomnia in Medical Students Provide Insights into Attention Deficit Hyperactivity Disorder?” published in your journal. This study aimed to investigate the relationship between attention deficit hyperactivity disorder (ADHD) symptoms, insomnia, and chronotype among medical students. The findings revealed that depression, anxiety, a history of self-harm, and insomnia may serve as predictors of ADHD, whereas chronotype was not identified as an independent predictor of ADHD symptoms.

One well-established feature of ADHD is that sleep disturbances are more common in individuals with ADHD than in the general population. The most frequently observed sleep problems associated with ADHD include insomnia, sleep-related breathing and movement disorders, and circadian rhythm sleep-wake disorders. Specifically, insomnia affects approximately 40-80% of adults with ADHD.¹ However, the results regarding chronotype preferences in ADHD vary, as some studies suggest that patients lean more toward an evening chronotype, while others indicate that they are mostly intermediate types.² Recently, an increasing number of researchers have postulated that sleep disorders may be predictors of ADHD.³

The findings of Kocakaya and Öztürk align well with the existing literature. In their study, 87.2% of individuals identified as high risk for ADHD reported experiencing insomnia, and the ADHD group displayed a greater prevalence of delayed circadian rhythms. A notable strength of their study is the exclusion

of participants with bipolar disorder or those currently using antidepressants, as this helped to minimize the potential confounding effects of mood disorders. However, the study does not specify whether participants who engage in shift work were included.

Accurate identification and evaluation of sleep disorders (particularly insomnia) and chronotype preferences require that individuals have appropriate conditions for sleep in accordance with the diagnostic and statistical manual of mental disorders, 5th edition diagnostic criteria. Additionally, the current sleep issue must not be better explained by another sleep disorder and should not occur only during the course of another sleep disorder. The current data indicate that individuals who begin shift work are more susceptible to sleep disorders, decreased sleep duration, increased depression and stress levels, and disrupted circadian rhythms, with these effects often intensifying over time.⁴ Furthermore, shift workers frequently struggle to adhere to sleep hygiene recommendations and typically lack the conditions necessary for restorative sleep.⁵

Given that the sample in Kocakaya and Öztürk’s study consisted of third-, fourth-, and fifth-year medical students-who often work night shifts as part of their training-ascertaining whether shift workers were included in the study becomes crucial. The inclusion of individuals who work shifts and who experience alternating schedules may have affected the study outcomes. The discussion should emphasize that individuals working shifts may experience insomnia and could show variations in chronotype preferences and circadian rhythms, potentially leading to additional sleep problems that might exacerbate ADHD symptoms.

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