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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 Desatürasyon İndeksi (ODI; ρ=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 iğcikleri sıklığı ve süresi, OSA'nın ciddiyetiyle birlikte daha düşüktü, bu da potansiyel tanısal 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, iğcik 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 sleepiness[20] 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 ≥90% airflow reduction for ≥10 seconds and hypopnea as a ≥30% reduction with ≥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/m^2 (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 (SpO_2) was 89.40% (SD =5.96), and minimum SpO_2 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).

Table 1. Sleep spindle characteristics, ESS, and ISI

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]

Table 2. Spindle characteristics by OSA severity

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, SpO_2: 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.'s.[11] 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 patients[28] and extend this by quantifying duration reductions, a finding consistent with the Yetkin and Aydogan[31] 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).
Informed Consent: It was obtained.

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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