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ARAŞTIRMA MAKALESİ RESEARCH ARTICLE

Severity of Obstructive Sleep Apnea and Its Impact on Retinal Ganglion Cell Complex and Retinal Nerve Fiber Layer

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Abstract

Aim: To evaluate whether systemic hypoxia associated with obstructive sleep apnea syndrome (OSA) is related to structural changes in the retinal ganglion cell complex (GCC) and retinal nerve fiber layer (RNFL).

Materials and Methods: SThis cross-sectional, observational case–control study included 49 OSA patients and 20 age- and sex-matched healthy controls with a normal apnea–hypopnea index (AHI <5). Participants were aged 30–65 years. Exclusion criteria included systemic or ocular disease, prior ocular surgery, smoking, and alcohol use. Only participants with refractive errors between −5.00 and +3.00 diopters were included. All subjects underwent overnight polysomnography. Spectral-domain optical coherence tomography (SD-OCT; Optovue RTVue) was performed in the morning (09:00–11:00) to measure average and sectoral GCC and RNFL thickness. Recorded sleep parameters included AHI, mean oxygen saturation (SpO^2), nadir SpO^2, duration of sleep with SpO^2 between 80–90%, and the percentage of total sleep time spent in this range.

Results: No statistically significant differences were observed between the OSA and control groups for average or sectoral GCC and RNFL thickness (all $p>0.05$). Correlation analyses showed no significant associations between GCC or RNFL thickness and AHI, mean SpO^2, nadir SpO^2, duration of SpO^2 at 80–90%, or the percentage of sleep time spent in this range (all $p>0.05$). Sector-based analyses revealed no focal retinal thinning.

Conclusion: GCC and RNFL thickness were not significantly associated with OSA severity or nocturnal hypoxemia parameters. These findings suggest that global hypoxic burden alone may not be sufficient to drive early retinal neurodegeneration. However, given the limited statistical power of the study, the results should be interpreted with caution. Larger, well-powered longitudinal studies are needed to confirm these findings.

Keywords: *Obstructive sleep apnoea, Ganglion cell complex, Retinal nerve fibre layer, Hypoxia, Optical coherence tomography*

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Obstrüktif Uyku Apnesinin Şiddeti ve Bunun Retina Ganglion Hücre Kompleksi ile Retina Sinir Lifi Tabakası Üzerindeki Etkisi

Özet

Amaç: AObstrüktif uyku apne sendromunda (OUAS) kronik sistemik hipoksinin retina gangliyon hücre kompleksi (GCC) ve retina sinir lifi tabakası (RNFL) kalınlıkları ile ilişkisini değerlendirmek.

Gereç ve Yöntem: Bu kesitsel, gözlemsel vaka-kontrol çalışmasına, 49 OSA hastası ve normal apne-hipopne indeksi (AHI <5) olan, yaş ve cinsiyet açısından eşleştirilmiş 20 sağlıklı kontrol katılımcısı dahil edildi. Katılımcıların yaşları 30–65 arasındaydı. Dışlama kriterleri arasında sistemik veya göz hastalıkları, daha önce geçirilmiş göz ameliyatı, sigara kullanımı ve alkol kullanımı yer alıyordu. Sadece −5,00 ile +3,00 diyoptri arasında refraktif kusuru olan katılımcılar çalışmaya dahil edildi. Tüm deneklere gece boyunca polisomnografi uygulandı. Ortalama ve sektörel GCC ve RNFL kalınlığını ölçmek için sabah (09:00–11:00) spektral alan optik koherens tomografi (SD-OCT; Optovue RTVue) yapıldı. Kaydedilen uyku parametreleri arasında AHI, ortalama oksijen satürasyonu (SpO^2), en düşük SpO^2, SpO^2'nin %80–90 arasında olduğu uyku süresi ve toplam uyku süresinin bu aralıkta geçirilen yüzdesi yer almıştır.

Bulgular: OUAS ve kontrol grupları arasında GCC ve RNFL kalınlıkları açısından anlamlı fark saptanmadı. Ayrıca GCC ve RNFL kalınlıkları ile AHI ve oksijen satürasyon parametreleri arasında anlamlı ilişki bulunmadı.

Sonuç: GCC ve RNFL kalınlığı, OUAS şiddeti veya gece hipoksemi parametreleriyle anlamlı bir ilişki göstermedi. Bu bulgular, genel hipoksik yükün tek başına erken dönem retina nörodejenerasyonunu tetiklemek için yeterli olmayabileceğini düşündürmektedir. Ancak, çalışmanın istatistiksel gücünün sınırlı olduğu göz önüne alındığında, sonuçlar dikkatli bir şekilde yorumlanmalıdır. Bu bulguları doğrulamak için daha geniş kapsamlı ve istatistiksel gücü yeterli uzunlamasına çalışmalar gereklidir.

Anahtar Kelimeler: *Obstrüktif uyku apnesi, Ganglion hücre kompleksi, Retina sinir lifi tabakası, Hipoksi, Optik koherens tomografi*

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INTRODUCTION

Obstructive sleep apnoea (OSA) is a common sleep disorder characterized by recurrent upper airway collapse during sleep, resulting in intermittent hypoxia and sleep fragmentation. It affects approximately 13% of middle-aged men and 5% of women, and chronic nocturnal oxygen desaturation is known to contribute to metabolic, cardiovascular, and neurocognitive morbidity (1). Increasing evidence suggests that OSA may also have important ocular implications. Intermittent hypoxemia and hemodynamic fluctuations associated with OSA can induce oxidative stress, inflammation, and endothelial dysfunction, ultimately impairing optic nerve head perfusion and predisposing to retinal nerve fiber layer (RNFL) thinning (1).

OSA has been associated with several ophthalmic conditions, including floppy eyelid syndrome, retinal vascular occlusions, and glaucoma. When the optic nerve is involved, hypoxia-related vascular dysregulation and impaired autoregulation may lead to retinal ganglion cell apoptosis, resulting in progressive RNFL loss (2). These mechanisms support a potential link between OSA and glaucomatous optic neuropathy, independent of intraocular pressure elevation.

OSA is known to be associated with a wide range of systemic disorders, including cardiovascular, metabolic, and neurocognitive diseases, reflecting its multisystem impact (3). Several epidemiological studies have examined the relationship between OSA and glaucoma. A large meta-analysis including 46 studies demonstrated that OSA is associated with an approximately 40% increased risk of glaucoma, even after adjustment for confounding factors (4). In addition to clinical data, optical coherence tomography (OCT)-based imaging studies have provided further structural evidence

supporting this association. Reduced peripapillary RNFL thickness has been reported in OSA patients compared with controls, particularly in those with moderate to severe disease, suggesting a role of chronic intermittent hypoxia in optic nerve vulnerability (1, 2).

Despite these observations, findings regarding OSA-related RNFL changes remain inconsistent. While some studies have reported focal or sectoral RNFL thinning, others have failed to demonstrate significant structural differences between OSA patients and healthy individuals (1). Such discrepancies may be attributed to variations in study design, disease severity, duration of hypoxic exposure, and imaging methodology, indicating that the retinal impact of OSA has not yet been fully clarified.

Another important issue is whether OSA-related retinal alterations are reversible with treatment. Continuous Positive Airway Pressure(CPAP) is the first-line therapy for OSA and effectively reduces nocturnal hypoxia. Previous studies have shown that CPAP therapy may lead to improvements in RNFL thickness and visual function, particularly in patients with more severe disease (5). These findings suggest that at least some OSA-related retinal changes may be partially reversible with timely intervention.

Based on the conflicting evidence in the literature, the present study was designed to evaluate the relationship between OSA severity and RNFL and GCC thickness prior to treatment initiation. By correlating OCT-derived retinal measurements with Polysomnography (PSG) parameters, we aimed to clarify whether increasing OSA severity is associated with detectable structural retinal changes and to better characterize the link between OSA and optic nerve vulnerability.

MATERIALS AND METHODS

This cross-sectional, observational case–control study was conducted between 1 June and 1 December 2022 to evaluate the relationship between OSA severity and RNFL and ganglion cell complex (GCC) thickness before treatment. The study was approved by the XXX Clinical Research Ethics Committee (Approval No. 2022-5, dated 9 October 2022), and written informed consent was obtained from all participants. The study included 49 newly diagnosed, untreated OSA patients and 20 age- and sex-matched healthy controls with Apnea–Hypopnea Index (AHI) <5 confirmed by full-night PSG. Participants were aged 30–65 years, had Best Corrected Visual Acuity (BCVA) ≥20/20, and a spherical equivalent between –5.00 and +3.00 Diopter (D). Only non-smokers without alcohol use were included. Individuals with systemic disease, ocular disease, previous ocular surgery or trauma, glaucoma or suspected glaucoma, Intraocular Pressure (IOP) >20 mmHg, poor-quality OCT images, or use of medications affecting IOP or retinal structures were excluded. PSG was performed using the Alice 6 system (Philips Respironics). OSA severity was classified according to AASM criteria as mild (AHI 5–14.9), moderate (AHI 15–29.9), or severe (AHI ≥30). Recorded parameters included AHI, mean SpO2, lowest SpO2, duration of SpO2 between 80–90%, and the percentage of total sleep time spent within this range.

All participants underwent a comprehensive ophthalmological examination between 09:00 and 11:00 by a single masked ophthalmologist. Spectral-domain optical coherence tomography (SD-OCT) imaging was performed using the RTVue system (Optovue Inc., Fremont, CA, USA). Average peripapillary RNFL thickness and average macular GCC thickness were used for analysis, as no significant sectoral differences were observed. Only high-quality OCT images were included; images with a signal strength index (SSI) <40, motion artifacts, segmentation errors, or poor centration were excluded. Data were analyzed using SPSS version 28.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± SD. Group comparisons were performed using the Mann–Whitney U test, and associations between OCT and PSG parameters were assessed using Spearman's correlation analysis. A post hoc power analysis (G*Power 3.1) demonstrated a statistical power of 47% to detect a medium effect size ($r = 0.3$) at $\alpha = 0.05$.

This study protocol was reviewed and approved by the Non-Interventional Clinical Research Ethics Committee of Mardin Artuklu University (Approval No. 2022-5, dated 9 October 2022).

RESULTS

A total of 49 patients with newly diagnosed OSA and 20 healthy control subjects were included in the study. The demographic and clinical characteristics of the participants are presented in Table 1.

Table 1. A total of 49 patients with newly diagnosed OSAS and 20 healthy controls were included in the study. The demographic and clinical characteristics of the study population is summarized.

Parameter	Total (n=69)	OSAS Group (n=49)	Control Group (n=20)	P value
Age (years), mean ± SD	46.3 ± 10.1	47.1 ± 9.8	44.0 ± 10.5	0.28
Female, n (%)	44 (63.3%)	31 (63.3%)	13 (65.0%)	0.89
BMI (kg/m²), mean ± SD	32.5 ± 6.4	33.6 ± 5.9	29.8 ± 7.1	0.03*
AHI, mean ± SD	28.0 ± 23.6	37.8 ± 22.1	3.2 ± 1.5	<0.001*
Mean SpO2 (%), mean ± SD	90.6 ± 2.8	89.9 ± 2.9	92.5 ± 2.1	0.001*
Lowest SpO2 (%), mean ± SD	76.8 ± 10.1	73.4 ± 9.8	85.0 ± 6.1	<0.001*
80–90% SpO2 duration (min), mean ± SD	92.9 ± 91.7	118.5 ± 95.6	25.1 ± 15.4	<0.001*
% Sleep time at 80–90% SpO2, mean ± SD	24.4 ± 22.2	31.0 ± 22.5	6.9 ± 5.8	<0.001*

BMI: Body Mass Index; AHI: Apnea-Hypopnea Index; SpO2: Oxygen saturation.

The mean age of the study population was 46.3 ± 10.1 years (range: 30–65 years), and 63.3% of the participants were female. The mean body mass index was 32.5 ± 6.4 kg/m², with values ranging from 19.0 to 48.5 kg/m². None of the participants had a history of systemic comorbidities such as diabetes mellitus, hypertension, renal failure, or other chronic systemic diseases. Correlation analyses demonstrated no significant associations between AHI and RNFL or GCC thickness in either eye. Similarly, mean oxygen saturation, lowest oxygen saturation, total duration of desaturation between 80% and 90%, and the percentage of time spent within this saturation range were not significantly correlated with RNFL or GCC measurements (all $p > 0.05$). These results are presented in Table 2.

Table 2. *The correlation analysis between sleep parameters and OCT measurements.*

Parameter	GCC OD (μm)	GCC OS (μm)	RNFL OD (μm)	RNFL OS (μm)
AHI	r = -0.08, p = 0.51	r = -0.10, p = 0.43	r = -0.12, p = 0.35	r = -0.09, p = 0.49
Mean SpO2 (%)	r = 0.11, p = 0.39	r = 0.09, p = 0.47	r = 0.08, p = 0.51	r = 0.10, p = 0.44
Lowest SpO2 (%)	r = 0.14, p = 0.27	r = 0.12, p = 0.34	r = 0.15, p = 0.23	r = 0.13, p = 0.31
80–90% SpO2 duration (min)	r = -0.07, p = 0.56	r = -0.09, p = 0.47	r = -0.11, p = 0.39	r = -0.10, p = 0.45
% Sleep time at 80–90% SpO2	r = -0.09, p = 0.48	r = -0.11, p = 0.38	r = -0.10, p = 0.44	r = -0.12, p = 0.34

GCC: Ganglion Cell Complex; RNFL: Retinal Nerve Fiber Layer; OD: Right Eye; OS: Left Eye; BMI: Body Mass Index; AHI: Apnea-Hypopnea Index; SpO2: Oxygen Saturation.

The mean AHI was 28.0 ± 23.6, with values ranging from 1.2 to 109.0. According to AASM criteria, 20 participants were classified as normal (AHI <5) and served as the control group, while 10 participants had mild OSA, 14 had moderate OSA, and 25 had severe OSA. The mean oxygen saturation during sleep was 90.6% ± 2.8%, and the lowest recorded oxygen saturation was 76.8% ± 10.1%. Participants spent an average of 92.9 ± 91.7 minutes with oxygen saturation between 80% and 90%, corresponding to 24.4 ± 22.2% of total sleep time. When patients with severe OSA were compared directly with the control group, no statistically significant differences were observed in average GCC thickness (97.65 ± 9.14 µm vs. 95.31 ± 2.44 µm, p = 0.32) or average RNFL thickness (102.47 ± 13.32 µm vs. 105.17 ± 7.97 µm, p = 0.56). The mean GCC thickness was 97.5 ± 7.6 µm in the right eye and 97.5 ± 8.1 µm in the left eye. The mean RNFL thickness was 104.9 ± 12.1 µm in the right eye and 107.0 ± 10.9 µm in the left eye.

Sectoral analyses of RNFL and GCC thickness did not reveal any statistically significant differences across OSA severity groups (p > 0.05), as summarised in Table 3.

Table 3. *Comparison of OCT parameters between OSAS and Control groups*

Parameter	OSAS (n=49) Mean ± SD	Control (n=20) Mean ± SD	p-value
Average GCC (µm)	97.03 ± 8.97	95.31 ± 2.44	0.32
Average RNFL (µm)	103.94 ± 12.36	105.17 ± 7.97	0.56

DISCUSSION

In this study, we evaluated the relationship between OSA severity, nocturnal hypoxemia parameters, and retinal structural measurements. No significant associations were found between polysomnography-derived indices, including AHI and oxygen saturation parameters, and OCT-derived GCC and RNFL thickness. In addition, retinal structural parameters did not differ significantly between OSA patients and healthy controls or within the severe OSA subgroup. These findings suggest that systemic hypoxemia alone may not be sufficient to explain early retinal structural changes.

Instead, local microvascular or perfusion-related factors may play a more important role. Our cross-sectional study did not find a significant correlation between the severity of OSA and the thickness of the RNFL or GCC. Several prior studies, however, have reported structural retinal changes in OSA. For instance, Shiba et al. found that nasal RNFL thickness decreases as OSA severity increases (6). Likewise, a meta-analysis by Yu et al. concluded that OSA patients had significantly thinner average RNFL than healthy controls, particularly in cases of moderate to severe disease (7). In contrast, other cross-sectional investigations have not observed consistent retinal thinning. Ferrández et al. reported no significant difference in GCC (ganglion cell layer + inner plexiform layer) thickness between OSA patients and controls (8) , although OSA patients in that study did show reduced retinal sensitivity on perimetry. Similarly, Moyal et al.(2018) found no significant differences in peripapillary or macular perfusion density between OSA patients and healthy individuals (9). Taken together, these conflicting results highlight the variability of findings across cross-sectional studies and suggest that the impact of OSA on retinal structure may be influenced by differences in study design, disease duration at the time of evaluation, comorbid conditions, or measurement techniques.

Intermittent hypoxemia is a hallmark feature of OSA and has been proposed as a potential mechanism for neural stress. Repeated cycles of oxygen desaturation and reoxygenation at night are thought to generate oxidative stress and inflammatory mediators. Experimental work by Kaur et al. demonstrated that hypoxia–ischemia in the retina can induce overexpression of hypoxia-inducible factor (HIF-1α) and downstream targets such as vascular endothelial growth factor (VEGF) and nitric

oxide synthase (10). Excess VEGF may disrupt the blood–retinal barrier, while increased nitric oxide and glutamate release have been associated with excitotoxicity and calcium influx, potentially leading to retinal ganglion cell (RGC) injury. These pathways have therefore been discussed in the literature as contributors to RGC loss during chronic intermittent hypoxia. RGCs are considered particularly sensitive to even short-term oxygen deprivation – Kergoat et al. reported that acute, transient hypoxic stress was sufficient to cause measurable RGC damage. Acute hypoxia tends to produce rapid energy failure and cell death, whereas chronic hypoxia might allow partial cellular adaptation. It has been hypothesized that prolonged low-grade hypoxia could trigger protective metabolic or mitochondrial adaptations in retinal neurons (11). Ferrández et al. suggested this as a possible explanation for why some OSA patients in their cross-sectional study did not exhibit RNFL thinning. In the context of our own cross-sectional findings, the absence of significant RNFL/GCC thinning despite OSA may indicate that intermittent hypoxia alone was not strongly associated with detectable retinal structural changes. The balance between potentially injurious effects of intermittent hypoxia and possible compensatory mechanisms may vary between individuals, but longitudinal studies would be needed to clarify this.

The relationship between OSA and glaucoma has been widely debated, with some epidemiological studies reporting a higher prevalence of glaucoma in patients with severe OSAS, potentially related to hypoxia-induced vascular dysregulation rather than intraocular pressure elevation (12). In this context, OSA has been proposed as a contributing factor to an optic neuropathy resembling normal-tension glaucoma, possibly mediated by vascular insufficiency and oxidative stress (13).

However, the present study focused primarily on early retinal structural changes rather than glaucomatous damage. In our cohort, none of the participants demonstrated manifest glaucoma, and no significant differences in RNFL or GCC thickness were observed across OSA severity groups. These findings suggest that OSA alone may not be sufficient to induce detectable structural retinal changes, at least in a cross-sectional setting. Consistent with previous reports, including Ferrández et al. (8), the relationship between OSA and ocular involvement appears to be heterogeneous and likely influenced by additional systemic and microvascular factors.

Interestingly, our cross-sectional results indicated that local retinal vascular factors may have a closer association with retinal status than systemic oxygen metrics. It has been hypothesised that ischaemia at the level of the retinal microcirculation could have a stronger influence on retinal ganglion cells (RGCs) than intermittent systemic hypoxaemia alone. Recent optical coherence tomography angiography (OCTA) studies have reported microvascular alterations in eyes with OSA, lending support to this idea. Ucak and Ünver (2020), for example, demonstrated reduced vessel densities in the superficial capillary plexus and radial peripapillary capillaries in OSA patients, which correlated with disease severity (14). Findık et al. observed significantly higher resistance in the posterior ciliary arteries that supply the optic nerve in OSA patients, which was accompanied by thinner RNFL measurements and a higher frequency of optic nerve ischaemic changes in those with severe OSA (12). Similarly, other investigators have described associations between reduced retinal perfusion and thinner retinal layers. In healthy individuals, a positive correlation has been documented between retinal capillary perfusion and inner retinal thickness (15), suggesting that chronic blood flow reduction may contribute to retinal thinning. Within this context, it is plausible that OSA-related ocular changes are more strongly linked to cumulative microvascular insufficiency rather than to systemic hypoxaemia indices such as AHI or average oxygen saturation. This interpretation may help explain why we did not observe direct correlations between systemic polysomnographic parameters and GCC/RNFL thickness in our study: these systemic metrics may not fully capture the nuances of ocular perfusion at a single time point.

Our observations are consistent with previous

cross-sectional studies suggesting that retinal vascular impairment may be an important mediator of structural changes in eyes with OSA (13). Indeed, OCTA has been identified as a valuable tool for detecting early microvascular alterations in glaucoma and other optic neuropathies, and it may also provide useful information in the assessment of OSA patients who could be at risk of optic nerve compromise (16).

The methods we used to assess retinal structure are worth noting. We employed the Optovue SD-OCT, which includes a dedicated GCC analysis. The GCC represents the combined thickness of the RNFL, the ganglion cell layer (GCL), and the inner plexiform layer (IPL) in the macula (17). This metric is clinically useful because glaucoma and other optic neuropathies tend to affect these inner retinal layers earlier and more prominently than the outer layers. By measuring the GCC in addition to the peripapillary RNFL, our aim was to provide a more comprehensive evaluation of RGC integrity. This constitutes an advantage over earlier OCT devices that reported only peripapillary RNFL thickness.

In our cross-sectional cohort, neither average RNFL thickness nor macular GCC thickness demonstrated significant reduction or correlation with OSA severity metrics. This finding may appear unexpected given the numerous mechanisms proposed for hypoxia-related retinal injury. One possible explanation is that many of our patients were newly diagnosed and had not yet developed long-standing OSA, meaning that structural degeneration may require a longer disease duration to become detectable. Another explanation is that subtle functional changes might precede structural alterations, which were not captured in our measurements. Importantly, our study design provides only a snapshot of baseline retinal status in untreated OSA, and does not allow conclusions about disease progression or causality.

Another important consideration in the interpretation of our findings is the difference in body mass index (BMI) between the groups. Increased BMI has been associated with systemic inflammation, endothelial dysfunction, and impaired microvascular circulation, all of which may potentially influence ocular structures and retinal integrity. Previous studies have suggested that obesity-related vascular dysregulation may affect retinal nerve fiber layer thickness and ocular perfusion. Therefore, the observed BMI difference in our cohort may have acted as a confounding factor, potentially masking or modulating the relationship between OSA severity and retinal parameters and increasing the risk of residual confounding. Although this variable was acknowledged as a limitation, its potential impact should be considered when interpreting the results. Future studies with better-controlled BMI distribution or adjusted analyses are warranted to clarify the independent effects of OSA on retinal structures.

Several limitations of this cross-sectional study should be acknowledged. The duration of OSA prior to diagnosis was unknown, preventing estimation of cumulative hypoxic exposure, which may partly explain the absence of consistent retinal structural changes. Retinal function was not assessed using visual field or electrophysiological tests, and subtle functional impairment may therefore have been missed. The relatively low statistical power (47% for a medium effect size) increases the risk of a type II error, and the lack of significant associations should be interpreted with caution. In addition, higher BMI in the OSA group represents a potential confounding factor, as obesity may independently affect vascular and ocular circulation. Finally, the limited sample size, particularly after stratification by OSA severity, and the reliance on static OCT measurements may have reduced sensitivity for detecting small or dynamic retinal changes, highlighting the need for larger longitudinal studies incorporating structural, functional, and vascular assessments.

The strengths of this study include the use of standard polysomnography for the confirmation of OSA in all participants, the performance of OCT measurements by a single masked examiner, and the inclusion of a treatment-naïve patient population.

CONCLUSION

In conclusion, this cross-sectional study did not demonstrate a significant association between OSA severity and retinal neurodegenerative parameters, including RNFL and GCC thickness. These findings suggest that systemic hypoxaemia or global hypoxic burden alone may be insufficient to explain early retinal structural changes. Retinal involvement may vary among individuals and could be influenced by additional factors, particularly local microvascular or ocular perfusion abnormalities. However, given the limited statistical power of the study, these results should be interpreted with caution. Larger, well-powered longitudinal studies incorporating both structural and functional retinal assessments are needed to confirm these findings and to further clarify the underlying pathophysiological mechanisms.

Author's contribution: Conceptualization: YD, BÇ, İBD; Supervision: YD, YA, SU; Writing: YD; Review: YD, MÖ; Editing: YD, İBD.

Conflict of interest: The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics approval: The study protocol was reviewed and approved by the Non-Interventional Clinical Research Ethics Committee of Mardin Artuklu University (Approval No. 2022-5, dated 9 October 2022). All participants provided written informed consent before enrolment, in accordance with the principles of the Declaration of Helsinki.

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