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Does Pediatric OSAS with Adenotonsillar Hypertrophy Impact on the Choroidal Thickness of Children?

Adenotonsiller Hipertrofinin Neden Olduğu Pediatrik OSAS'ın Koroid Kalınlığı Üzerine Etkisi

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ÖZ

Amaç: Adenotonsiller hipertrofiye bağlı obstrüktif uyku apne sendromu (OUAS) olan çocuklarda koroid kalınlığındaki değişiklikleri normal kontrol grubundaki değerlerle karşılaştırıp değerlendirmek.

Araçlar ve Yöntem: Tek merkezli, vaka kontrol çalışmasına adenotonsiller hipertrofiye bağlı 39 pediatrik OUAS hastası ve 39 yaş ve cinsiyet uyumlu sağlıklı kontrol dahil edildi. Koroid kalınlıkları, gelişmiş optik koherens tomografi kullanılarak değerlendirildi. Koroid kalınlıkları subfoveal, nazal ve temporal bölge olarak ölçüldü. Sferik eküvalan, göz içi basıncı ve aksiyel uzunluk değerleri de ölçüldü.

Bulgular: Çalışma ve kontrol gruplar arasında subfoveal, nazal ve temporal bölgenin bölgenin koroid kalınlığı açısından istatistiksel olarak anlamlı bir fark yoktu. İki grup arasında Sferik eküvalan, göz içi basıncı ve aksiyel uzunluk değerlerinde de istatistiksel olarak anlamlı bir fark bulunmadı.

Sonuç: Bu sonuçlar adenotonsiller hipertrofinin çocuklarda koroid kalınlığı üzerinde anlamlı derecede bir etkiye neden olmadığını göstermiştir.

Anahtar Kelimeler: Adenotonsiller hipertrofi; Koroid kalınlığı; Pediatrik Obstrüktif Uyku Apne Sendromu

ABSTRACT

Purpose: To determine the changes in choroidal thickness of children with obstructive sleep apnea syndrome (OSAS) as a result of adenotonsillar enlargement versus normal controls.

Materials and Methods: This study was consist of 39 pediatric OSAS individuals and 39 age,sex and body mass index-matched healthy children. Choroidal thicknesses of the patients and normal controls were analyzed by enhanced depth imaging optical coherence tomography. The choroidal thicknesses were measured in the subfoveal, nasal, as well as temporal areas. The spherical equivalent, intraocular pressure, and then axial length were additionally measured.

Results: Statistically notable difference in the choroidal thicknesses were not found in any areas between the groups. No statistically significant differences were found in between spherical equivalent, intraocular pressure, and axial length values of the two groups.

Conclusion: These results show that adenotonsillar hypertrophy does not result in a major impact on choroidal thickness of children.

Key Words: Adenotonsillar hypertrophy; Choroidal thickness; Pediatric obstructive sleep apnea syndrome

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INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is identified by a recurring partial and/or complete collapse of the upper airway during sleep.[1] Recurrent airway obstruction leads to oxygen desaturation, hypercapnia, and intra-thoracic pressure changes. This status affects hemodynamic, autonomic, humoral, and neuroendocrine regulation[2]. OSAS patients have complaints such as breath holding, restless sleep, mouth breathing, sleep pauses, snoring, and enuresis. Adenotonsillar hypertrophy has been accepted as the main pathophysiological cause in children with OSAS[3]. Polysomnography (PSG) during the night in the laboratory has been identified as the gold standard for the diagnosis of OSAS[1]. However, this method is time-consuming, expensive, and difficult to apply to children. For this reason, the diagnosis of pediatric OSAS can usually be made based on physical findings and parental reports of nocturnal symptoms[4].

The choroid layer is an almost full vascularized tissue located between the retina and the sclera, and have a critical role in the normal ocular function. It provides oxygen and nutrients under a highly regulated blood flow to the outer retina; it also protects photoreceptors and secretes growth factors[5]. A structurally and functionally normal choroid is essential for retinal health. Hypoxia secondary to recurrent apneic attacks in OSAS causes blood pressure and hemodynamic changes[6]. Severe hypoxia causes increased vascular resistance which can endanger retinal perfusion and oxygenation. Therefore, there may be structural changes in the choroid tissue[7].

Spectral-domain optical coherence tomography (SD-OCT) is a reliable method that allows high resolution in vivo imaging of retinal microstructures. SD-OCT is useful in many ocular diseases to describe the disease process[8]. The correct in vivo morphological evaluation of the choroid without SD-OCT is quite difficult. Enhanced depth imaging optic coherence tomography (EDI-OCT), a relatively new technique, uses longer wavelength light. Thus, more valuable information about choroid morphology is obtained in normal healthy people and patients with various diseases[9-11]. In previous studies, alterations in choroidal thickness were shown in adults with OSAS[6,8,12,13]. However, there are few studies on choroid thickness in children with OSAS. For this reason, we aimed to evaluate the changes in choroidal thickness in kids with pediatric OSAS and adenotonsillar enlargement.

MATERIAL and METHODS

Patients

This study was approved by the local Clinical Research Ethics Committee at the Faculty of the Medicine, Ahi Evran University. (Approval No. 2018-23/191). This study was conducted by the tenets of the Declaration of Helsinki, and written informed consent was obtained from the parents of the subjects. A total of 78 children (39 OSAS patients and 39 controls) aged between 5 and 12 years were included in the study. The OSAS group was composed of children who had been diagnosed as having adenoid and/or tonsil hypertrophy in the otolaryngology clinic and whose parents described them as having sleep apnea. Nocturnal obstructive symptoms such as snoring, chronic nasal obstruction, open mouth sleeping ,oral respiration and interrupted breathing during sleep were asked to parents. The video recordings taken by the parents were evaluated. The patients' adenoids and tonsils were assessed using the flexible nasopharyngoscope. Nasopharyngeal adenoid tissue volume, occluding the chona, was graded into four classes base on the criteria by Cassano[14]. Tonsil hypertrophy was graded according to the Brodsky grading scale[15]. This study involved the patients with grade 3 or 4 adenoids and patients with 3+ or 4+ tonsil hypertrophy. It was carried out the nocturnal pulse oximetry in the patient and control group. Desaturation was characterized as a 4% or more low blood oxygen saturation values and desaturation cluster in kids with pediatric OSAS was described as 5 or more blood oxygen desaturation in a 10 to 30 minute period. All of the patients and controls analysed at least six-hour assessments with nocturnal pulse oximetry, and those patients owned 3 or more clusters of desaturation, and experienced at least 3 nocturnal desaturations lower than 90%, were diagnosed with pediatric OSAS[16]. We excluded patients with nasal septal deviation, regular drug use, head and neck malformations, allergic symptoms, passive smokers, Patients with SE over ±2.0

D, visual acuity below 0.8, ocular disorder or past trauma/surgery, obesity, diabetes mellitus, a systemic disorder such as hypertension or Behçet's disease, and the use of any ocular or systemic medication were excluded from the study.

Ophthalmologic examination

A full ophthalmic examination was performed for all participants, and a detailed history was taken. The ophthalmic examination included best-corrected visual acuity with the Snellen chart, anterior segment examination with the slit lamp, intraocular pressure (IOP) measurement with an air puff tonometer, dilated fundus examination, and choroidal thickness (right eyes) measurement in OCT with ED. Spherical equivalent (SE), IOP, and axial length (AL) measurements were carried out by optical low coherence reflectometry (Lenstar, Haag-Streit, Switzerland) were recorded for each patient and normal control.

EDI-OCT Scan Protocol

The EDI-OCT scan protocol has been previously described elsewhere[10]. The EDI mode of a spectral-domain OCT (Software version 6.3.3.0, Heidelberg Engineering Inc., Heidelberg, Germany) was used in this study. The choroidal thickness was determined as the distance between the outer reflective *retinal pigment epithelium* (RPE) layer and the inner sclera border. All choroidal depth measurements were performed manually by the same ophthalmologist (RK) (Figure 1). The sections were measured horizontally across the fovea at 500-µm intervals. The digital calipers provided by the Heidelberg Spectralis software were used for the vertical choroidal thickness measurements. Choroidal thickness measurements were taken of the subfoveal, 1500 µm nasal, and 1500 µm temporal to the center of the fovea.

Statistical Analysis

The data were analyzed with the SPSS software, version 20.0. The Kolmogorov–Smirnov test was used to determine whether the data were normally distributed. The statistical methods used were the independent t-test, chi-square, and Pearson's correlation test. The results with p-value less than 0.05 ($p < 0.05$) were accepted as statistically significant.

RESULTS

There were 78 participants in the study, 39 (50.0%) patients and 39 controls (50.0%). The age distribution of the patient group was 7.31 ± 2.02 years and the age distribution of the control group was 7.33 ± 2.08 years. Fifteen (38.5%) of the patient group were female and 24 (61.5%) were male; 16 (41.0%) of the control group were female and 23 (59.0%) were male. No significant difference was determined between the patients and controls in terms of sex ($\chi 2 = 0.054$; $p = 1.000$), mean age($t = 0.055$; $p = 0.956$) or body mass index (BMI) ($p = 0.433$). ($p>0.05$, Table 1).

Table 1. Demographic data of groups

	Control group (n=39)	OSAS group (n=39)	p value
Age (years)	7.33±2.08	7.31 ± 2.02	0.956*
Male/Female	23/16	24/15	1.000**
BMI (kg/m2)	15.97±1.72	15.91±1.34	0.433

*Independent-samples test, **Chı-Square Test, SD=Standard deviation, BMI: Body Mass Index, OSAS: Obstructive sleep apnea syndrome

Table 2. Choroidal thickness, Intraocular pressure, and axial length in the Study and Control Groups

	Control	OSAS	p* value
SFCT (µm)	321.74± 61.45	316.49± 61.22	0.706
TCT (µm)	313.10± 57.31	310.44± 62.68	0.845
NCT (µm)	252.95± 59.52	253.64± 62.63	0.960
SE (D)	0.30± 0.72	0.06± 0.74	0.174
IOP (mm Hg)	16.09± 2.69	15.92± 2.33	0.774
AL (mm)	22.79± 0.65	22.67± 0.85	0.514

Values are expressed as mean ± SD, * Independent-Samples Test, Subfoveal choroid thickness (SFCT), Temporal choroid thickness (TCT), Nasal choroid thickness (NCT), spherical equivalent (SE), D (Diopter), Intraocular pressure (IOP), and axial length (AL)

Table 2 shows the outcomes of the between-groups comparison of subfoveal choroidal thicknesses (SFCT), nasal choroidal thicknesses (NCT), and temporal choroidal thicknesses (TCT) measurements. The NCT

was significantly lower compared to SFCT and TCT in both the control group and the OSAS group. The SFCT, NCT, and TCT of the patients and controls had no significant differences. The measurement of the subfoveal choroidal thickness is shown in Figure 1.

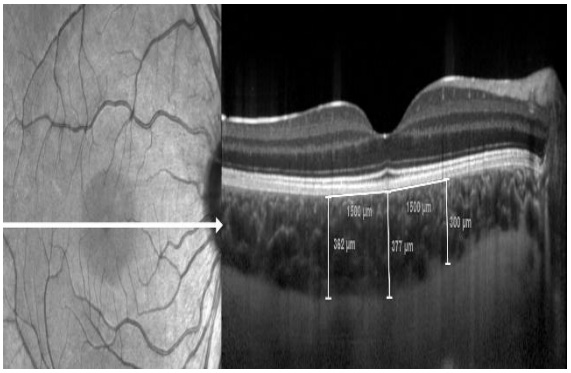

Figure 1. Choroidal thickness measurement in a patient using EDI-OCT**.**

Also, there were no significant differences in the means of SE, AL, and IOP values between the OSAS patients and control groups. According to Table 3,

Table 3. Choroid thickness and gender

	Female	Male	p* value
SFCT (µm)	318.80±49.06	315.04±68.71	0.855
TCT (µm)	316.46±59.20	306.67±65.71	0.641
NCT (µm)	257.93±56.17	250.95±67.39	0.740

Values are expressed as mean ± SD, ∗Independent-samples test. Subfoveal choroid thickness (SFCT), Temporal choroid thickness (TCT), and Nasal choroid thickness (NCT)

there were no significant differences between TCT, SFCT, and NCT values by sex. When the relationships between age and choroid thicknesses were evaluated, there was no correlation between age and TCT ($r = 0.024$; $p = 0.883$), age and NCT ($r = 0.209$; $p = 0.201$), and age and SFCT ($r = 0.033$; $p = 0.844$). Also, there were no significant relationships between the grade of adenotonsillar hypertrophy and duration of the disease and choroid thicknesses (Table 4).

Table 4. Adenoid grade, tonsil grade, duration, and choroid thickness

	Adenoid Grade	Tonsil Grade	Duration
SFCT Rho*	0,071	-0,296	0,196
p**	0,666	0,067	0,232
TCT Rho*	-0,017	-0,202	0,263
p**	0,918	0,218	0,105
NCT Rho*	0,025	-0,112	0,183
p**	0,88	0,497	0,264

*Rho Spearman Correlation Coefficient, **Pearson's correlation test, SFCT: Subfoveal choroid thickness, TCT: Temporal choroid thickness, NCT: Nasal choroid thickness

DISCUSSION

OSAS is a sleep-related breathing disorder that involves a decrease or complete cessation of airflow despite the continous effort of breathing. Most OSAS patients snore loudly when airflow is reduced or blocked. The prevalence of OSAS is 4% in males and 2% in females in the middle-aged population[17]. As age increases, the prevalence also increases and is estimated to be around 28-67% for older men and 20-54% for older women[17]. The prevalence of OSAS is known to be between 1% and 5% in children[18].

In adults, a large and elongated soft palate, a large tongue, elonge uvula, and large tonsils are among the most common causes of airway obstruction in OSAS. The main feature of OSAS in children is increased upper airway resistance during sleep. 80 percent of OSAS and respiratory sleep disorders in children are due to adenotonsillar hypertrophy[2]. The lymphoid tissue of the Waldeyer's ring rapidly develops between the ages of 3 and 6, when OSAS peaks.

Pediatric OSAS is not just a common simple respiratory problem. It is now noticed that it is a major problem that affects other systems and may result in morbidity. Adenotonsillar hypertrophy lead to the partial or complete obstruction of the upper airway and is an independent risk factor of sleep-related disorder breathing ranges from simple snoring to OSAS. Obesity is another risk factor, such as pediatric adenotonsillar hypertrophy, for the OSAS progression in the pediatric population.

Obesity is another risk factor of similar significance to in pediatric population. Intermittent airway obstruction by adenotonsillar hypertrophy causes hypoxia and decreases oxygen saturation. Recurrent hypoxia and reoxygenation episodes can produce oxidative stress, inflammation, vascular endothelium injury and poor response to vasodilator agents such as nitric oxide[2,3].

Many studies have reported that OSAS causes decreases in neurocognitive, behavioral, and quality of life scores[18]. It is shown that OSAS can cause depression, poor quality of life, attention deficit hyperactivity disorder in children. Recent studies showed that OSAS may be related to several cardiovascular risk factors such as hypertension, dyslipidemia insulin resistance, and impaired glucose tolerance[19-21]. Pediatric OSAS lead to metabolic changes, sleep disruption, growth retardation, pulmonary hypertension, and cor pulmonale in children[2]. Moreover, children with OSAS are more likely than OSAS adults to have hypertension and metabolic syndrome[1]. In addition, normotensive glaucoma, primary open-angle glaucoma, retinal nerve fiber layer (RNFL) thinning, and visual field defects have been reported more frequently in OSAS [22-24]. Karakucuk et al.[25] showed a positive correlation between IOP levels and the severity of OSAS. Moghimi et al.[26] reported that OSAS patients had worse visual field indices and lower nerve fiber layer parameters than controls.

For proper retinal function, the retina should be supported with a choroid layer with normal function and structure[6,12,13]. The choroid mainly supports the outer layers of the retina, which contains photoreceptors. Changes in the thickness of the choroid layer and blood flow can cause photoreceptor death or dysfunction. Various ocular diseases such as retinitis pigmentosa, central serous chorioretinopathy, diabetic retinopathy, and glaucoma cause changes in choroidal thickness[27]. Based on this, many studies investigating choroid thickness in OSAS patients have been conducted. Kucuk et al.[28] found that OSAS patients' IOP, RNFL, subfoveal, and parafoveal choroid thicknesses were not significantly different from the control group. Bayhan et al.[6] showed that choroidal thickness decreased only in the nasal region in severe OSAS patients. They also determined that there were no significant changes in mild and moderate OSAS patients in all choroidal regions. On the contrary, Ozge et al.[29] reported that the thickness of the choroid increased in the nasal region only in OSAS patients. He et al.[30] showed that subfoveal choroidal thickness was significantly lower in OSAS than in the control group. They also observed that the reduction in choroidal thickness was more pronounced in severe OSAS patients.

There are conflicting findings of choroidal thickness in adult OSAS patients. However, there are not enough studies on choroid thickness in children with OSAS. A better understanding of the normal thickness characteristics of the choroid in childhood will assist in the diagnosis of choroidal abnormalities associated with eye disease, as well as prevent the development of refractive errors and choroid-associated abnormal growth of the eye[27]. OCT is a non-invasive tool used to visualize the posterior regions of the ocular fundus and generally to evaluate retinal diseases. However, the traditional SD-OCT device is not sufficient for imaging the choroid due to reasons such as wavelength-related light scattering and loss of signal in the image path. EDI-OCT is used in the evaluation of disease activity and treatment effectiveness in choroidal diseases as it allows detailed visualization of the choroid[11]. In this study, the choroid was evaluated noninvasively in three regions (subfoveal, nasal, and temporal) using EDI-OCT. There were no significant differences in the AL, IOP, SF, TCT, SFCT, and NCT parameters in the OSAS patients when compared with the controls. Cakabay et al.[3] reported that severe adenoid hypertrophy did not make a significant difference in choroid thickness, SF, IOP, and AL compared to control. Bayraktar et al.[31] showed significantly thinner choroidal thicknesses 1.5 mm and 3.0 mm from nasal to the fovea in OSAS patients when compared with the controls. Yenigun et al.[2] determined that SFCT, TCT, and NCT were decreased in OSAS patients. These results reveal that the contradictory results in choroidal thickness in adult OSAS patients are also observed in pediatric patients.

In this study, we aimed to investigate changes in choroidal thickness in children with obstructive

symptoms due to severe adenotonsillar hypertrophy. Polysomnography (PSG) is the gold standard method to assess the severity of obstruction. Due to the difficulty of application, the use of PSG in clinical practice in children is limited. For this reason, we evaluated the children according to physical findings, parental reports of nocturnal symptoms and nocturnal pulse oximetry.

In conclusion, our findings revealed that adenotonsillar hypertrophy did not cause a change in choroid thickness. The duration of obstructive symptoms in children is less than in adults. Therefore, we think that intact choroidal blood flow and normal intraocular pressure protect the choroid in children with obstructive symptoms.

Conflicts of Interest

The authors declare that they have no conflict of interest.

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