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Central corneal thickness in children with adenotonsillary hypertrophy

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ABSTRACT

Aim: We aimed to detect whether the central corneal thickness value in children with advanced phase ATH (it is indicated with OSAS at large extent) becomes different from that of children of the same age.

Materials and Method: Prepubertal, nonobese, 6-12 years of age children were included in the study. The first group consisted of 15 patients (mean age, 8.13 ± 1.6 years; body index, 19.43 ± 2.11), and the second group consisted of 42 patients (mean age, 8.65 ± 2.7 years; body mass index 20.93 ± 5.71). The control group consisted of 31 subjects. Comprehensive otolaryngologic examinations of all children were done by an otolaryngologist. Following tonasal endoscopy, the levels of adenoid hypertrophy were graded from 1 towards 4 according to the criteria of Cassano et al., i.e., grade 1 as 25%, grade 2 as 25-50 %, grade 3 50-75%, and grade 4 over 75% were evaluated as adenoid hypertrophy making airway obstruction. Tonsillary hypertrophy was graded using the Brodsky scale. Tonsillar size was graded as follows: grade 1, small tonsils confined to the tonsillar pillors; grade 2, tonsils extended just outside the pillors; grade 3, tonsils extended outside the pillors but did not meet at the midline; grade 4, large tonsils met at the midline. The patients whose adenoid and tonsil hypertrophies became grade 3 and 4 were evaluated as advanced level ATH, and they were included in the study. After the complications of all the patients were evaluated, visual acuity, measurement of intra eye-pressure and biomicroscopyand fundus treatment were performed. Central corneal thickness (CCT) of the patients was measured under topical anesthesia using an ultrasonic packmetry device.

Results: There was no statistically significant distinction among all groups according to the results obtained by Duncan's multiple comparison procedure (p>0.01).

Conclusions: We could not find a statistically significant distinction between the corneal thickness and ATH. Further studies in more advanced age groups or using a wider range of patients series will test whether this result is also observed in the children who have been exposed to the disease for longer periods of time.

Key words: *Adenoid hypertrophy, cornea, children, packmetry*

Introduction

Sleep-disordered breathing (SDB) is a highly prevalent condition in children that is characterized by snoring, witnessed apnea, unrefreshing sleep, and excessive daytime sleepiness. SDB has entities ranging from severe simple snoring to Obstructive Sleep Apnea Syndrome (OSAS) [1,2].Adenotonsillar Hypertrophy (ATH) is the most common cause of OSAS in children. It is reported that the prevalence of snoring in children is approximately 12% and the prevalence of pediatric OSAS is approximately 1-3%. Children with OSA experience recurrent periods of elevated upper airway re-

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sistance during sleep due to partial or complete upper airway obstruction, which results in snoring, episodic oxyhemoglobin desaturation, hypercapnia, and repeated arousals [1-3]. Adult patient groups with OSAS are associated with floppy eyelid syndrome, visual field defects, retinal vein occlusion, central serous chorioretinopathy, optic nerve dysfunctions ophthalmological disorders such as open-angle glaucoma and papilla oedeme.

Untreated glaucoma is an optical nerve pathology that can lead to permanent damage of the retinal nerve fiber layer and lead to visual field loss, which over time can progress to blindness. Although according to some studies, the glaucoma frequency in patients with OSAS is similar to that of the wide community [4], in most of the studies OSAS and Glaucoma relationship has been pointed [5,6]. In the calculation of intraocular pressure, which is known as the primary risk factor for glaucoma development, the process of measuring central corneal thickness is significant [7,8]. However, studies in recent years have emphasized that the central cornea thickness value is also important in glaucoma pathogenesis [9,10]. Thus, there are also studies specifying thin cornea as the major risk factor for severe ocular hypertension and primer open-angle glaucoma [10,11].

The cornea is an avascular transparent tissue that is the eye's most outer layer, forming 70% of the total refractive power of eye [12]. Since the central cornea is avascular, it obtains the necessary oxygen from the atmosphere and receives its metabolic needs such as nourishment through diffusion from capillaries located around the cornea, the tears and aqueous humor (a fluid in the anterior portion of the eye) CCT can be associated with factors including race, age, sex, dry eye, anti-glaucoma drugs, contact lens wearing, corneal diseases and systemic diseases such as diabetes mellitus [13-16]. On the other hand, it has been reported that CCT is higher in OHT(ocular hypertension) than in POAG(primer open-angle glaucoma), pseudo-exfoliation glaucoma (PSXG) and the normal population [17]. Although no change was specified in the CCT values of the patients with OSAS, there are studies confirming an increase in thickness [18].

With this study we aimed to determine whether values of the central cornea thickness in children with advanced Adenotonsillar Hypertrophy (substantially accompanying OSAS) are different from those of the same-age control group of children.

Materials and Methods

The patients included in our study were selected among prepubertal, non-obese, 6-12 year-old children, who in the previous two years had obstructive complaints reported by themselves and/or their parents, such as snoring, mouth breathing and pausing of breath during sleep. All parents of children gave informed consent. This study was also approved by the Ethics Committee of the Institute.

An otolaryngologist performed detailed otolaryngologic exams on patients. We graded adenoid hypertrophy levels from 1 to 4 based on the criteria of Cassano et al. [19] by nasal endoscopy. Grade-1 25%, Grade-2 25-50%, Grade-3 50-75%, Grade-4 75% and over were classified as adenoid hypertrophy causing obstruction of the airways. Tonsiller hypertrophy was assessed using a Brodsky scale [20]. Tonsillar size was graded as follows: grade 1, small tonsils confined to the tonsillar pillars; grade 2, tonsils extended just outside the pillars; grade 3, tonsils extended outside the pillars but did not meet at the midline; grade 4, large tonsils met at the midline. Patients with adenoid and/or tonsil hypertrophy graded 3 and 4 were evaluated as advanced ATH.

In the group Grade 1-2, ATH children with head and neck malformation and no recognized chronicle disease were excluded from this study. In this study, the patient group grade 3-4 with ATH included 60 children (left and right eyes, a total of 120 eyes) and the control group with ATH included 35 children (left and right eyes, total 70 eyes) without upper and lower airway obstruction and no infection.

Parents of children classified in Grade 3-4 with advanced ATH hypertrophy were asked to complete the OSA-18 quality of life survey. The survey comprises 18 items in 5 domains of sleep disturbance, physical suffering, emotional distress, daytime problems, and caregiver concerns. A point scale was used ranging from 1 to 7 to assess the severity of the symptoms related to each item. The total scores, ranging from 18 to 126, were recorded and were classified as mild (<60); moderate (>60, <80), or severe (>80) in each patient group [21].

Ophtalmologic Examination

After complaints of all patients and their histories were recorded, visual acuity, intraocular pressure measurements, biomicroscopy and fundus examinations were performed. The central corneal thickness of patients was measured with ultrasonic pachymeter (Pacline Pachymeter, Opticon, Italy) under topical anaesthesia. The cornea was anesthetized with topical proparacaine HCl 0.5% (Alcaine, Alcon, USA). Measurements were carried out with the tip of the probe targeted to the centre of the pupil and oriented perpendicular to the cornea while the participant looked at a fixed target. Three consecutive measurements were made at the center of the cornea of each eye and the mean value was calculated. All measurements were performed by the same person, and the average of 3 values obtained for each eye was recorded. Patients with value of refracting error outside of the -2,00 D and over, +2,00 D interval were excluded from the study. Topical or systemic treatment areas were not included in the study. Patients who had an eye surgery history or were contact lens users, as well as patients with glaucoma, uveitis or corneal pathology were also not included in this study.

Statistical Analysis

All collected data were tabulated in a spreadsheet using Microsoft Excel for Windows, software version 2003 (Microsoft Corp., Seattle, WA, USA). All analyses were conducted using IBM SPSS statistical software, version 20 (SPSS Inc. Chicago IL, USA). Subjects were divided into three groups: those without ATH, those with moderate (60< OSA-18 survey scoring <80) and severe (OSA-18 survey scoring >80) disease. Data from the three groups were compared using one-way ANOVA for continuous data, and Fisher's exact test for categorical data, when appropriate. Duncan's multiple comparison procedure was employed for pairwise comparisons. The correlation (r) between OSA-18 survey variables and ophthalmologic variables in the moderate/severe OSAS group was evaluated using Pearson's correlation coefficient. A p-value less than 0.01 was considered to be statistically significant.

Results

The patients were divided into two groups according to the test scores of the OSA-18 survey: The first group (between 60 and 80 paints; middle severity OSAS) consisted of 15 patients (mean age, 8.13 ± 1.6 years; body index, 19.43 ± 2.11), and the second group (over 80 points severe OSAS) consisted of 42 patients (mean ages, 8.65 ± 2.7 years; body mass index 20.93 ± 5.71). In the scoring of OSA – 18 survey, no patient could take a point under 60 (mild OSAS). The control group consisted of 31 subjects (mean age 8.59 ± 2.0 years; body mass index and age of each three study groups not statistically different, p>0.01). Both eyes' corneal thickness parameters between control groups and moderate or severe OSA patients groups were compared. We could not detect statistically significant between any of the groups with respect to the corneal thickness parameters, according to the results obtained by Duncan's multiple comparison procedure (p>0.01) (Table 1).

Discussion

It is well-known that ATH is most frequent cause of upper airway obstruction and OSAS in children. OSAS is characterized by recurrent apnea occurring as a result of partial or total obstruction of upper airway during sleep, mild and severe hypoxemia and increased vascular resistance occurring as a result of hypopnea attacks

Table 1. Mean CCT values in study group and control group.

	Control	Moderete OSA Group	Severe OSA Group	P Value
Number Of Subject	31	15	42	
Number Of Males	18 (58%)	6 (40%)	25 (59%)	
Mean Age	8.59± 2,0 years	8.13 ± 1,6 years	8.65 ± 2,1 years	P>0.01
BMI (kg/m2)	20.11±2.71	19.43±2,11	20.93.±5.71	P>0.001
Right Eye CCT	530,7±26.5	534.2 ±31.7	528.9±35.7	P>0.001
Left Eye CCT	533.1±32.1	532.3± 33.8	531.6±28.4	P>0.001

*All values above are expressed as mean ± S(standard deviation)

[1,2]. Although nocturnal polysomnography is regarded as the golden standard in the diagnosis of OSAS, this technique is high-priced and time-consuming, which is why for patients with ATH the OSA-18 survey technic is performed instead. It is a reliable, tested and approved survey that can be used safely for children with OSAS occurring as a result of adenotonsillar hypertrophy [21]. In the evaluation of OSAS and its severity among children with ATH, OSA-18 survey obtained ultra test–retest reliability, validity, and responsiveness [22]. Therefore, we used the OSA-18 survey to diagnose OSAS and evaluate its severity.

OSAS is defined as the major risk factor for cardiovascular and neurological diseases. Hypoxia, developing as a result of apnea and hypopnea in patients with OSAS, causes stimulation of stress mechanisms due to alterations in the cerebral blood flow and it also causes neurohumoral and autonomic activation and release of proinflamatuar cytokines, which affects optical nerve circulation and is accompanied by loss of ganglion cells [23]. In addition, in patients with OSAS, as a result of intermittent hypoxia reactive oxygen species (ROS) may be generated in a high concentration [24]. ROS stimulate peroxidation of proteins, lipids or fatty acids found in the cell membrane, damage corneal stromal tissue [25] and cause necrosis in bovine corneal endothelial cells [26].

In patients with OSAS, increased sympathetic activity raises the endothelin level, which is an endogenic vasoconstructor agent, and it also decreases the levels of nitric oxide, a known as vasodilator, which results in a composed vasoconstriction that damages the microvascularities. [27].

The most emphasized of the ocular complications of OSAS has been glaucoma. In our country for the research of probable early findings of glaucoma in patients with OSAS, laser polarimeter is used to measure the thickness of retina nerve fiber layer. During this study it was observed that the retina nerve fiber layer is thinning and it has been indicated that thinning arises as a result of hypoxia and vasoconstruction occurring in OSAS patients [28]. A similar result was reported by Akbulut et.al, although they were measuring the retina nerve fiber layer indirectly by using confocal laser scanning ophthalmoscopy [29]. During this study (Akbulut M et al.) the central corneal thickness was also measured, which has recently been considered a significant parameter glaucoma, but no difference was found between the patients and normal individuals. In another study, an increase in the prevalence of glaucoma was observed in patients with OSAS in consequence of the breakdown of the autoregulation mechanism of blood flow in the optic nerve head or secondary damages such as arterial hypertension and atherosclerosis induced by this syndrome [5,30]. In studies with more extended patient groups with OSAS, the glaucoma frequency was found to be higher than in the normal population [4]. There are also studies reporting that patients with OSAS have a tendency to ocular complications such as non-arteritic anterior ischemic optic neuropathy and floppy eyelid syndrome [31,32]. In such cases, the direct effects of hypercoagulopathy and anoxia on the optic nerve are the cause of OSAS-dependent ischemia reperfusion injury. Hypoxia and hypercapnia episodes seen in patients with OSAS cause significant changes in the corneal epithelium, stromada and endothelium. Development of stromal changes such as corneal thinning due to stromal edema, acidosis and neovascularization entail endothelium dysfunction depending on the increased oxidative stress in cells [33,34].

In some previous studies the relationship of OSAS with glaucoma was emphasized and central corneal thickness was only used for evaluating the intraocular pressure value [29,35,36]. Corneal thickness is affected by many factors such as age, gender and race, and it is a significant parameter in the diagnosis and tracking of glaucoma [16,37]. Increased corneal thickness may cause a false calculation of intraocular pressure as too high while a thin cornea may cause a false calculation of intraocular pressure as too low [38,39]. The Ocular Hypertension study group considers the central corneal thickness value as an independent risk factor for glaucoma progression [10]. Furthermore, Shildkrot et al. emphasized the need to measure the corneal thickness of patients with glaucoma more than once [40]. With such emphasis on the central corneal thickness measurements in recent times, it is now necessary to evaluate the normal corneal thickness evaluation in different communities and to determine other parameters affecting corneal thickness. Values of corneal thickness

change according to the nations. In the Turkish society women have values in average 552.2+35.9 μm, whereas men's average value is 552+.35.4 μm [41], while in China, the respective values are 575 ± 31 μm, and 574 ±33 and in Japan they are 511.1 ± 32.5 μm and 518.3 ± 33.2 μm. In Caucasian societies the CCT value is approximately 506 ± 39 μm in all groups, while in the Korean population it is 530.9 μm [41,42].

Toit et al. specified that the CCT measurement should be performed at least 2 hours after waking in the morning because of the probable swollen eyes immediately after a night's sleep, which might increase central corneal thickness [43]. We have followed this recommendation.

Contrary to the studies that found no relationship between age and CCT [10,44,45], Nomura et al. noted a tendency to corneal thinning in older men; however, Cho et al. noted that they found corneal thinning mostly in female groups.

In our country, Altınok et al. reported that there is no agreement on the relationship between age, intraocula pressure, spherical equivalence of refractive error, systemic disorders, menopause and CCT [41].

It is indicated that metabolic and hormonal changes in body cause a variance in corneal thickness by making a change in pump function with the help of Na-K ATPase in corneal endothelial cells. The most common examples of that are the studies on metabolic stress in diabetic patients that causes an increase in central corneal thickness. Thus, studies of children with type 1 diabetes indicate an increase in corneal thickness [46,47].

The cause of corneal oedema due to hypoxia is still a matter of debate. Some researchers working on mountainers stated [48] that hypoxia activates anaerobic glycolysis for energy production in epithelial cells and increases lactate production in the stroma, making a water flow through osmosis and stroma. It is also stated that increased lactate concentration in the stroma breaks the endothelium pump function and this causes an increase in stromal hydration. There are studies indicating that chronical hypoxia causes an increase in the intracellular nitric oxide and calcium levels as well as mitochondrial ROS production and a decrease in antioxidant enzyme levels such as superoxide dismutase, which causes a reduction in cellular vitality.

Ekinci et al. evaluated central corneal thickness parameters on 107 patients and found out a negative correlation between CTT and Apnea–Hypopnea Index (AHI) as for according to min. SpO2 a weak positive correlation [18]. We investigated if adenotonsillar hypertrophy (known as childhood OSAS) presenting with sleep disorder and metabolic stress in children makes any change in the corneal thickness. At the end of our study we did not find any statistically significant result in terms of corneal thickness between the healthy children and patient children groups. The reason for that may be the early diagnosis. Different results may be obtained by studying the same parameters in children who have been exposed to hypoxia for longer periods.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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