Original Article

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Auditory Functions of Patients with Obstructive Sleep Apnea Syndrome

Auditory Functions in OSAS

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Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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

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ABSTRACT

Objective: Obstructive sleep apnea syndrome (OSAS) is a respiratory disease occurring during sleep. In order for the hearing system to be able to function normally, the inner ear and cochlear nerve require healthy oxygen support. The purpose of this study was to assess the hearing functions of patients with OSAS and to reveal the relations between polysomnographic parameters and hearing test results.

Materials and Methods: The study was performed with 35 patients diagnosed with moderate or severe OSAS with polysomnography, and a control group consist of 30 individuals. The STOP-Bang questionnaire was used to establish the control group. Detailed otoscopic examinations were administered to all subjects by the same otolaryngologist, followed by tympanogram, pure tone audiometry and transient evoked otoacoustic emissions (TEOAE) tests.

Results: We determined a mild sensorineural hearing loss in patients with OSAS. When the different frequencies were evaluated separately, hearing threshold values in the patients with OSAS were significantly higher compared to the control group at 500, 1000, 2000, 4000 and 8000 Hz in the both ears. TEOAE test reproducibility values in both ears were significantly lower in the study group compared to the control group.

Conclusion: The hearing system is affected to varying degrees in patients with OSAS. In the event that hearing loss is detected in patients presenting to otolaryngology clinics due to snoring, then assessing these subjects in terms of risk of OSAS may important in terms of reducing mortality and morbidity that may develop at later stages in association with OSAS.

Key words: Obstructive sleep apnea syndrome; Hearing; Pure tone audiometry; Transient evoked otoacoustic emissions

Introduction

Obstructive sleep apnea syndrome (OSAS) is a respiratory disease occurring during sleep. It is characterized by recurring upper airway obstructions and results in high levels of morbidity and mortality by affecting several systems, particularly the cardiovascular and neurological systems. It is seen in 4% of men and 2% of women in the middle age group [1]. Apnea-hypopnea attacks throughout the night and resulting intermittent hypoxia play a key role in the development of various complications. Oxidative stress occurs as a result of chronic hypoxia, and vascular endothelial functions can be compromised. In addition, the vasa nervorum can be damaged as a result of hypoxia. Irreversible peripheral neuropathy develops in association with intermittent hypoxia [2].

In order for the hearing system to be able to function normally, the inner ear and cochlear nerve require healthy oxygen support. Conditions capable of leading to hypoxia can cause various levels of hearing loss by impairing the auditory system [1,2].

The purpose of this study was to assess the hearing functions of patients with OSAS and to reveal the relations between polysomnographic parameters and hearing test results.

Methods

Ethical approval was granted from local ethical committee with approval number 2017:5:4. Patients diagnosed with moderate or severe OSAS (Apnea-Hypopnea Index>15) based on the results of polysomnography tests performed in the XXXX, were included in the study. Informed consent was obtained from all patients. The STOP-Bang questionnaire (SBQ), developed by Chang et al., [3] was used to establish the control group. The SBQ is extensively employed as an accurate screening test for OSAS. The questionnaire comprised 4 yes/no questions concerning habitual snoring,

tiredness/sleepiness, observed apnea, and high blood pressure. Each affirmative positive receives a score of 1. A further score of 1 is added for each of the following clinical characteristics observed: body mass index >35 kg/m2, age >50 years, neck circumference >40 cm, and male gender. Scores of 3 or more from a maximum possible total of 8 are considered to represent a high risk in terms of OSAS. The control group was established from patients shown by the questionnaire to be at low risk in terms of OSAS. Detailed otoscopic examinations were administered to all subjects by the same otolaryngologist, followed by tympanogram, pure tone audiometry (PTA) and transient evoked otoacoustic emissions (TEOAE) tests. Subjects with a history of hearing loss, with hearing loss in the family, with a history of ototoxic drug use or with chronic diseases such as hypertension and diabetes were excluded from the study.

Polysomnography: Full polysomnography analysis was performed using the Compumedics E-series Sleep System (Compumedics Sleep, Melbourne, Vic., Australia). Electroencephalography (EEG), electrooculography, electromyography and electrocardiography tests were carried out concurrently. Surface electrodes were attached in order to measure EEG channels, right and left electro-oculographies and submental electromyography. Nasal or oral respiratory flow was calculated using airflow. Inductive plethysmography bands were used to monitor thoracic and abdominal respiratory movements, as well as body position. Arterial oxygen saturation for pulse oximetry was measured from the fingertip. Apnea was defined as continuous cessation of airflow greater than 10 seconds in duration, while hypopnea was defined as a reduction of airflow or 30% or more with a duration greater than 10 seconds in duration accompanied by oxygen desaturation value of ≥ 3% or reduced thoracic wall movement. The apnea-hypopnea index (AHI) was defined as the total number of apnea and hypopnea events per hour of sleep.

Tympanogram: These tests were performed with Interacoustics AT 235h tympanometer. Subjects with type B and C tympanograms were excluded from the study for the purpose of excluding pathological conditions in the middle ear.

Pure-tone Audiometry: These tests were administered to all subjects by an experienced audiologist using a Maico MA53 device inside a sound-proofed chamber. Subjects were directly exposed to a range of different frequencies and amplitudes. Subjects were instructed to lift their hands or press a button as soon as they perceived the sound tone. The lowest response level achieved was adopted as the threshold value for each separate frequency. Air thresholds were calculated at frequencies of 250, 500, 1000, 2000, 4000 and 8000 Hz, while bone threshold values were recorded at 500, 1000, 2000, 4000 Hz. The hearing thresholds recorded in the study group at each separate frequency were compared with the equivalent value measured in the control group. Also mean hearing thresholds for both groups were calculated by dividing the thresholds at frequencies 500, 1000 and 2000 Hz by 3.

Transient Evoked Otoacoustic Emissions: All TEOAE tests were carried out by an experienced audiologist in a sound-proofed chamber with a Vivosonic Integrity Evoked Potentials System device (Vivosonic, Inc., Toronto, Canada). A click stimulus was employed. The signal/noise ratio (SNR) was calculated at four distinct frequencies (1, 2, 3 and 4 kHz). The SNR values recorded in the study and control groups were measured for each frequency and subsequently subjected to comparison analysis. The SNR represents the difference between emission amplitude and noise floor. In addition, signal amplitude values and test reproducibility values were recorded for each subject.

Power Analysis: The preliminary outcome of the study was the difference in the hearing thresholds. In our preliminary study, the standard deviation was determined as 5.9 in the study group and 5.1 in the control group. The expected difference in the hearing threshold was at least 10 dB. Accordingly, with

35 patients in study and 30 individuals in control group, the power of the study was calculated as 99%, with alpha error of 0.05 using Russ Lenth Piface Java Module.

Statistical Analysis:

SPSS 17.0 software (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Data were expressed as mean±standard deviation. The Shapiro-Wilk test was used to determine distribution of data. Student's T test was used for comparisons in the case of normal distribution and the Mann-Whitney U test under non-parametric conditions. For correlation analysis, Pearson's or Spearman's correlation tests were used according to the normality of the data. $p<0.05$ was regarded as significant for all tests.

Results

The study was performed with 35 patients aged 39-48 (44.4±3.9) diagnosed with moderate or severe OSAS at polysomnography tests, and a control group consist of 30 individuals aged 28-51 (43.1±2.4) identified as low risk in terms of OSAS based on the SBQ. The study group consisted of 20 men and 15 women, and the control group of 17 women and 13 men. There was no statistically significant difference between the two groups in terms of age or sex. There was no statistically significant difference between body mass index of the control group (27.5±2.2) and study group (28.3±1.9) ($p=0.06$). Mean AHI in the study group was 37.7±21.9. Demographic characteristics and polysomnography data for individuals in the study and control groups are shown in Table 1.

Mean hearing thresholds based on audiogram results in the study group were 23.7±5.9 in the right ear and 22.9±6.6 in the left ear, compared to control group values of 12.3±5.1 in the right ear and 9.4±4.6 in the left ear (Table 2). Mean hearing thresholds in both ears were significantly higher compared to

the control group (p<0.001, Mann- Whitney U test). There was no difference in terms of bone thresholds between groups. Also no air-bone gap was observed at PTA tests.

When frequencies were examined separately, hearing thresholds at both ears in the study group at 500, 1000, 2000, 4000 and 8000 Hz were significantly higher compared to the control group (Figure 1).

Evaluation of TEOAE values revealed no statistically significant variation between SNR values and signal amplitudes in the two groups. However, test reproducibility values in the study group were 60±12.6 in the right ear and 58.1±18.6 in the left ear, compared to 70.1±8.1 in the right ear and 68.4±6.5 in the left ear in the control group. Test reproducibility values in both ears were significantly lower compared to the control group (right ear p<0.001, left ear p<0.05, Mann Whitney U test). TEOAE test parameters are summarized in Table 3.

When hearing thresholds were compared with polysomnography data, no significant correlation was observed between AHI, SpO2, total sleep time (TST), sleep time at an oxygen saturation less than 90% (TST90) or mean desaturation values and hearing thresholds and TEOAE values.

Discussion

We determined a mild sensorineural hearing loss in patients with OSAS. When the different frequencies were evaluated separately, hearing threshold values in the patients with OSAS were significantly higher compared to the control group at 500, 1000, 2000, 4000 and 8000 Hz in the both ears. No statistically significant difference was observed in TEOAE test results between the patient and control groups in terms of SNR or amplitude values. However, test reproducibility values in both ears were significantly lower in the study group compared to the control group. Additionally, no correlation

was observed between hearing threshold levels and the polysomnographic parameters AHI, spO2, TST, TST90 and mean desaturation values.

OSAS is a widespread respiratory disease characterized by snoring and breathing interruption attacks during sleep. It can cause complications in several systems in association with ischemia and intermittent hypoxia developing due to recurring apnea attacks. Few studies have investigated the effects of OSAS on the hearing system, and the results of those studies are inconsistent.

Hwang et al. [4] reported that the presence of OSAS does not affect hearing thresholds. In their study of patients with OSAS, Martines et al. [5] reported that the hearing thresholds of patients with OSAS at extended high frequency were significantly higher than those of patients with simple snoring. However, they also reported that mean hearing thresholds between frequencies of 250 and 8000 Hz did not differ between individuals with OSAS and simple snoring. At the TEOAE test, significantly lower SNR values were determined only at frequencies of 3000 and 4000 Hz between the severe OSAS and simple snoring groups. In contrast to that study, we compared patients with OSAS with healthy individuals at no risk of OSAS by establishing a control group based on the Stop Bang questionnaire. Our result showed significant elevation in hearing thresholds at frequencies of 1000-8000 Hz in patients with OSAS compared to the control group. We observed no difference after the TEOAE test between the groups in terms of SNR and amplitude values.

Casale et al. [6] reported that mean hearing thresholds were within normal limits in patients with OSAS, but that these were significantly higher than those of the control group. Additionally, when individual frequencies were analyzed, hearing thresholds at 4000 Hz were significantly higher than those of the control group. In the TEOAE test, they observed significantly lower reproducibility and SNR values in patients with OSAS compared with the control group. Similarly, Matsumura et al. [7] observed no

significant difference between groups in terms of mean hearing thresholds but determined significantly higher hearing thresholds at high frequencies compared to the control group. Ballaccihino et al. [8] determined significantly high hearing loss in individuals at risk of OSAS with the Stop Bang questionnaire and significantly low SNR values using the TEOAE test. Ekin et al. [1] reported that hearing thresholds were not affected at low and high frequencies in individuals with OSAS and/or simple snoring compared to the control group, but observed significantly high hearing threshold at extended high frequencies. When subjects with simple snoring and the OSAS were compared, however, they determined no difference in hearing thresholds at any frequencies.

The subject of how OSAS affects the hearing system is still controversial. Steiner et al. [9] reported that blood plasma viscosity increases in patients with OSAS and that this compromises microcirculation. Bernand et al. [10] also reported that this hyperviscosity can lead to impairment of the hearing system. In contrast, Casale et al. [6] reported that the adverse effect of OSAS on the hearing system may be due to intermittent hypoxia and ischemia associated with this. Ekin et al. [1] suggested that hearing loss was the result of exposure to constant noise deriving from snoring, rather than of hypoxia.

The most important limitation of our study is that extended high frequencies were not studied at audiogram tests. The fact that no investigation was made of the pathogenesis of hearing loss in OSAS is another limitation.

In conclusion the hearing system is affected to varying degrees in patients with OSAS. In the event that hearing loss is detected in patients presenting to otolaryngology clinics due to snoring, then assessing these subjects in terms of risk of OSAS may important in terms of reducing mortality and morbidity that may develop at later stages in association with OSAS.

Compliance with Ethical Standards

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FIGURE LEGEND

Figure 1: Audiological results of study and control group

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TABLES

Table 1: Comparison of dermographic properties and polisomnographic parameters of OSAS and control groups.

	CONTROL (n=30)	STUDY (n=35)	P
Age	43.1±2,4	44.4±3.9	P=0,16
Gender (M/F)	13/17	20/15	P=0.06
BMI	27,5± 2,2	28,3± 1,9	P=0,06
AHI		37.7± 21.9	
SpO2		87,9± 4	
TST		345±53.5	
Desaturation Ratio		6,2±2.7	
TST90		146±106.1	

BMI: Body mass index
AHI: Apnea/hypopnea index
TST: Total sleep time
TST90: Sleep time at an oxygen saturation less than 90%

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Table 2: Mean hearing levels for each frequency

	RIGHT EAR			LEFT EAR		
Frequency (Hz)	Patients	Control		Patients	Control	
250	17±8.3	15.2±6.9	P=0.48	18.3±11.6	13.4±7.1	P=0.10
500	22.3±10.6	12.8±6.6	P<0.05*	21.9±11.3	10.8±4.7	P<0.01*
1000	23.6±12.3	12.2±5.6	P<0.01*	22.1±11.5	9.6±5.5	P<0.01*
2000	24.9±12.5	10.4±5.1	P<0.01*	23.3±12	8±5.4	P<0.001*
4000	28.5±15.1	14±6.9	P<0.001*	30.4±15	13.2±6.9	P<0.001*
8000	29.3±16.6	19.4±10.7	P<0.05*	36.2±17.2	22±17.9	P<0.01*
Mean	23.7±5.9	12.3±5.1	P<0.001*	22.9±6.6	9.4±4.6	P<0.001*

* Statistically significant (Mann Whitney U test)

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Table 3: TEOAE SNR levels

	RIGHT EAR			LEFT EAR		
Frequency (Hz)	Patients	Control		Patients	Control	
1000	5.3±3.2	8.5±4.5	P=0.01	6.7±3.2	9.7±4.5	P=0.01
2000	5.5±4.3	8±2.9	P=0.09	6.8±3.2	8.2±3.3	P=0.21
3000	7.1±2.8	7.5±4.4	P=0.62	5.4±3.4	5.9±3.1	P=0.46
4000	6.2±3.1	4±3.8	P=0.01	5.4±3.3	6.4±5.6	P=0.77
Reproducibility	60±12.6	70.1±8.1	P<0.01*	58.1±18.6	68.4±6.5	P<0.05*

*: Statistically significant (Mann Whitney U test)

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