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Is There a Relationship Between Obstructive Sleep Apnea (OSA) and Hearing Loss?

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Background: Obstructive sleep apnea (OSA) is a common disorder with an estimated prevalence in the general population of 2–5%. Its main clinical features are loud snoring and breathing stoppage during sleep. Ischemia could be a consequence of noise-induced hearing loss because cochlear oxygen tension is reduced during and after noise exposure. In this study, we evaluated auditory function in patients affected by OSA and simple snoring.

Material/Methods: A total of 66 participants (male to female ratio: 40:26) were included in the study, of which 21 were in the control group, 18 were in the simple snoring group, and 27 were in the OSA patient group. Polysomnography and audiometric examination were performed in all participants.

Results: The mean ages of the participants in the control, simple snoring, and OSA groups were 39.14±9.9, 37.28±8.2, and 41.56±8.99 years, respectively. There were no statistically significant differences among groups regarding age or sex; however, there were statistically significant differences among groups in body mass index, apnea-hypopnea index scores, mean saturation, and duration under 90% saturation. In addition, statistically significant differences were found between the patient group and the control and simple snoring groups concerning the mean saturation, duration under 90% saturation, and the extended high frequency of hearing.

Conclusions: These data show that snoring may cause hearing loss at extended high frequencies.

MeSH Keywords: **Hearing Loss • Sleep Apnea, Obstructive • Snoring**

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Background

Obstructive sleep apnea (OSA) is a prevalent breathing disorder in sleep that influences roughly 4% of middle-aged males and 2% of middle-aged females [1]. It consists of intermittent hypoxia (IH) causing blood hypoxemia, hypercapnia, sleep fragmentation, increased respiratory tries, and enhanced sympathetic activity. The hypoxemia results in peripheral nerve damage by harming the vasa nervorum. In the early stages of ischemia, mechanisms to reduce peripheral neuropathy are activated, but these become insufficient over time, and obvious neuropathy is inevitable in chronic hypoxemia [2-4]. The transduction mechanism of the inner ear and transmission of nerve impulses along the auditory path are highly dependent on the oxygen supply; few studies, with non-uniform results, have considered the possibility that obstructive apnea-hypopnea at night can interfere with the processes of generation and transmission of nerve impulses at the level of the auditory system [5,6]. Hearing loss causes are embryonic developmental anomalies (e.g., microtia, anotia, and external auditory canal atresia), genetic (e.g., nonsyndromic and syndromic), infectious (e.g., acute otitis media and otitis media with effusion), noise exposure, otosclerosis, trauma, ototoxicity, acoustic neuroma, autoimmune, central auditory processing disorder, presbycusis, and Meniere disease [7]. Experimental and clinical studies have shown that ischemia could be a consequence of noise-induced hearing loss, because cochlear oxygen tension is reduced during and after noise exposure [8,9]. Anoxia or reperfusion leads to cochlear dysfunction by inflammatory factors, among which nitric oxide can affect cochlear outer hair cells [10,11].

We evaluated the auditory function in patients affected by obstructive sleep apnea syndrome (OSAS) and simple snoring to determine if these conditions were associated with whether this situation associated with either exposure to noise or hypoxia.

Material and Methods

The study was approved by the local ethics committee in accordance with the Helsinki Declaration, and written informed consent was received from the patients and control subjects before being enrolled into the study. The patient and control cohorts were recruited at the Pulmonary Medicine and Otorhinolaryngology Department. Audiometric examination was analyzed at the Otorhinolaryngology Department.

Patient selection

A total of 66 participants (male-to-female ratio: 40: 26) were included in the study, of which 21 were in the control group, 18 were in the simple snoring group, and 27 were in the OSA

subjects group. Polysomnography and audiometric examination were performed in all participants. The exclusion criteria for the studied subjects were age >60 and <20 years, chronic diseases (e.g., diabetes mellitus, chronic kidney failure, congestive heart failure, hepatic illness, hypertension, and hypercholesterolemia), tinnitus, middle ear disease, major neurological or psychiatric disease, brain tumor or vestibular schwannoma, vertigo, hypogonadism/menopause, family history of hearing loss, history of prior ear surgery, occupational noise exposure, autoimmune diseases, cancer, history of acoustic trauma, conductive hearing loss, exposure to ototoxic substances, pregnancy, head and neck radiation exposure, history of smoking, ongoing infection-inflammation, and being on any medication.

Polysomnography

Sleep-disordered breathing events were scored manually by the same examiner according to the 2012 American Academy of Sleep Medicine criteria and sleep stages were scored according to standard criteria with 30-s epochs. Overnight polysomnography was performed using the 16-channel Embla system (Medcare Inc., Iceland) with continuous sleep technician monitoring. The system consists of 4 channels of electroencephalography (EEG) (with electrode placements at C4-A1, C3-A2, O2-A1, and O1-A2) and 2 channels of EOG, recording submental EMG, oronasal airflow, thoracic and abdominal movements, pulse oximeter oxygen saturation, tibial EMG, body position, electrocardiogram readings, and tracheal sound. Apnea was defined as the complete cessation of airflow lasting more than 10 s. Hypopnea was defined as a reduction >30% in airflow lasting more than 10 s accompanied by >4% desaturation and/or arousal. The average number of episodes of apnea and hypopnea per hour of sleep was measured as the AHI. Patients were diagnosed with OSA if the apnea-hypopnea index (AHI) was ≥ 5 .

Audiometric examination

All of the cases provided their medical history, and underwent otoscopy, tympanogram, and a pure tone audiometric examination. The following audiometry parameters were measured for each participant: low-frequency audiometry (LFA) of 250 Hz to 2 kHz; high-frequency audiometry (HFA) of 4 kHz to 8 kHz; and extended high-frequency audiometry (EHFA) of 10 kHz to 16 kHz. Audiometry was performed by an expert audiologist blinded to the study. We counted the average threshold at each frequency for the 2 ears of each subject. We averaged the thresholds at 250 Hz, 500 Hz, 1 kHz, and 2 kHz as LFA; thresholds at 4 kHz, 6 kHz, and 8 kHz as HFA; and thresholds at 10 kHz, 12 kHz, 14 kHz, and 16 kHz as EHFA, to obtain the averaged pure tone hearing level (PTA) for each subject.

Table 1. Demographic characteristics of the groups.

Groups		BMI	Duration under %90 saturation	Mean saturation	Age	AHI	Sex	
							Female (n)	Male (n)
Control (n=21)	Mean	29.81	.00	94.24	39.14	.00	10	9
	Std. deviation	3.12	.00	2.05	9.91	.00		
Simple snoring (n=18)	Mean	25.35	.03	92.68	37.28	2.17	9	9
	Std. deviation	6.08	.12	1.83	8.23	1.43		
Patients (n=27)	Mean	32.53	29.35	88.51	41.56	24.46	7	20
	Std. deviation	8.38	36.72	6.54	8.99	23.62		
P Value		.002	<0.001	<0.001	.298	<0.001	.18	.18

Statistical analyses

Statistical analyses were performed using SPSS 20 software. Continuous data are expressed as means ± standard deviation (SD). Statistical comparisons were performed using one-way ANOVA. To determine the relationships between these variables in each group separately, Pearson’s correlation coefficients were calculated. AHI, BMI, variables in patients, and the simple snoring and control groups were assessed. Results were considered statistically significant when the p value was less than or equal to 0.05.

Results

We included 66 participants, 27 of whom were in the OSA group (6 patients with severe OSA, 8 patients with moderate OSA, and 13 patients with mild OSA), 18 in the simple snoring group, and 21 in the control group. The mean age of the control group was 39.14±9.91 years, that of the simple snoring group was 37.28±8.23 years, and that of the OSA group was 41.56±8.99 years (the demographic features of the groups are given in Table 1). There were no statistically significant differences among the groups concerning age or sex; however, there were statistically significant differences among the groups regarding the BMI, AHI scores, mean saturation, and duration under 90% saturation. There were statistically significant differences among the OSA and control and simple snoring groups concerning the duration under 90% saturation and mean saturation; however, there were no differences between the simple snoring and control groups. The simple snoring and control and OSA groups did not differ in terms of low-frequency or high-frequency, and the low-frequency and high-frequency hearing all groups were normal.

Although there was not a significant difference in duration under 90% saturation and mean saturation between control and

simple snoring subjects, there was a significant difference in hearing of extended high frequencies. There was a significant difference in duration under 90% saturation and mean saturation between simple snoring subjects and OSA subjects, but there was no difference in hearing of extended high frequencies (Tables 2–4).

The duration under 90% saturation, mean saturation, and age were correlated with any frequency of hearing. AHI was not associated any frequency of hearing. The BMI, duration under 90% saturation, mean saturation, age, and AHI were correlated with each other, but there was no correlation between AHI and age.

Discussion

There have been few studies in patients with OSA using hearing tests. Huwang et al. [12] studied 34 OSA subjects and 190 control subjects and reported finding no significant positive association with PTA (averaged pure-tone threshold)-low or PTA-high for all subjects.

In a study by Sardesai et al. [13], the bed partners of snorers demonstrated a unilateral high-frequency pattern of hearing loss consistent with noise-induced hearing loss. Furthermore, the affected ear in every case was the one that was chronically exposed to snoring noise. We found extended high-frequency hearing loss in both ears in OSA and simple snoring subjects. We found a statistically significant positive correlation of hearing of all frequencies with the duration under 90% saturation, age, and BMI, and a negative correlation with the mean saturation. Although there were significant differences between OSA and simple snoring subjects in duration under 90% saturation, mean saturation, and AHI scores, we did not find a significant difference in extended high-frequency hearing loss. These findings suggest that hearing loss in these

Table 2. Differences between control and simple snoring group's frequencies and exact P values.

Groups		Right low frequencies	Left low frequencies	Right high frequencies	Left high frequencies	Right extended high frequencies	Left extended high frequencies
Control (n=21)	Mean	14.14	15.00	21.52	20.28	22.43	22.00
	SD*	3.32	3.58	6.64	5.99	8.90	6.98
Simple snoring	Mean	17.39	16.11	21.28	24.61	39.67	39.78
	SD*	8.70	8.12	15.74	15.83	20.41	20.17
P value		0.12	0.57	0.95	0.25	0.001	0.001

* Standart deviation.

Table 3. Differences between control and OSA subjects's frequencies and exact P values.

Groups		Right low frequencies	Left low frequencies	Right high frequencies	Left high frequencies	Right extended high frequencies	Left extended high frequencies
Control (n=21)	Mean	14.14	15.00	21.52	20.28	22.43	22.00
	SD*	3.32	3.58	6.64	5.99	8.90	6.98
OSA subjects	Mean	18.04	18.19	27.22	29.70	43.19	42.63
	SD*	7.39	6.74	16.57	31.85	18.85	18.49
P value		.06	.06	.14	.19	<0.001	<0.001

* Standart deviation.

Table 4. Differences between simple snoring and OSA subjects group's frequencies and exact P values.

Groups		Right low frequencies	Left low frequencies	Right high frequencies	Left high frequencies	Right extended high frequencies	Left extended high frequencies
Control (n=21)	Mean	17.39	16.11	21.28	24.61	39.67	39.78
	SD*	8.70	8.12	15.74	15.83	20.41	20.17
OSA subjects	Mean	18.04	18.19	27.22	29.70	43.19	42.63
	SD*	7.39	6.74	16.57	31.85	18.85	18.49
P value		.79	.36	.23	.53	.55	.63

* Standart deviation.

subjects is due to continuous exposure to loud noise, not over-night hypoxemia.

Casale et al. [14] studied 39 OSA patients and 21 simple snoring control subjects. They found that patients with OSAS had a PTA significantly higher than the control group. Analyzing the single frequencies, the OSAS group showed higher thresholds and was statistically significant at 4 kHz compared to the control group. They suggested that the hypoxia in severe OSAS could be a risk factor for auditory pathway damage. In our study, we found that patients with OSA did not differ from the simple snoring group. In addition, our patients did not have hearing loss at 4 kHz and other low and high frequencies. The hearing loss in our subjects occurred at extended high frequencies.

We believe that hearing loss may be associated with continuous noise exposure.

Anatomic and physiologic knowledge of the auditory mechanism reveals that the frequency coding along the basilar membrane progresses from high to low frequencies with the distance from the basal end of the cochlea [15]. Taken together, these findings indicate that long-term listening to recorded music at high volume may result in harm to the high-frequency basal end of the cochlea, evident here as worse extended high-frequency hearing. It may ultimately prove to be the case that some patterns of exposure are more likely to result in slowly progressive changes in the basal cochlea than other patterns of exposure, a finding that would explain the diverse

outcomes regarding the usefulness of extended high-frequency monitoring [16].

While hearing loss in OSA patients at high frequency was based on hypoxia in some studies [13], other studies [12] have shown hearing loss based on continuous noise exposure. In our study, we found a significant correlation between the duration below 90% saturation and mean saturation and high-frequency hearing loss, but we did not find a correlation between AHI and high-frequency hearing loss. We found a significant difference between the 2 groups (control vs. simple snoring and OSA patients); however, we did not find a difference between OSA patients and the simple snoring group in high-frequency hearing loss.

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Conclusions

This study is the first to investigate hearing loss in healthy control, simple snoring, and OSA subjects. We believe that hearing loss may be due to continuous noise exposure rather than hypoxia in patients with OSA. In addition, early auditory screening of OSA patients may provide early diagnosis of hearing loss and may also contribute to their awareness in the fight against OSA. Preventing noise in patients with snoring and OSA can prevent hearing loss that may occur early. Further studies need to be performed to clarify this issue.

Declaration of interest

The authors report no conflicts of interest.