Obstructive Sleep Apnea and Hearing Loss: Is There Any Correlation?

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Abstract

Introduction Obstructive sleep apnea (OSA) is a breathing disorder related to sleep that has a negative effect on the behavior and health of people around the globe. Chronic hypoxemia and variations in the concentration of oxygen in the blood noticed in OSA individuals may have adverse effects on the process of auditory transduction and transmission.

Objective To assess the effect of OSA on hearing and to find out the parameters that have more influence on hearing recorded during polysomnography of patients with OSA.

Methods The present is a hospital-based, observational, analytical, cross-sectional study conducted over a period of one year. After application of the exclusion criteria, the patients were then submitted to the application of the Berlin questionnaire, as well as polysomnography, impedance, and pure tone audiometry (PTA).

Results A total of 58 individuals were studied. Age, gender, and height were comparable among cases and controls, while weight, body mass index (BMI), and neck circumference were significantly higher in cases in comparison to controls. The mean PTA was significantly higher at 10 kHz or higher frequencies in cases in comparison to controls.

Conclusion We observed that there is evidence that the auditory mechanism is affected in OSA patients. We recommend early auditory screening of OSA patients for timely diagnosis and to raise awareness about its prevention.

Keywords
- sleep apnea
- obstructive sleep apnea
- polysomnography
- audiometry

Introduction

Obstructive sleep apnea (OSA) is a breathing disorder related to sleep that involves a reduction or complete cessation of airflow despite ongoing breathing effort, which is a result of the relaxation of pharyngeal muscles.[1] It is the second most common respiratory disorder after asthma. People in any age group may be affected, but OSA is more common among

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middle-aged males, with rates between 2% to 4% of the adult population.[2] With negative effects on the behavior and health of people around the globe, OSA has a direct correlation with many diseases, such as hypertension, heart attack, heart failure, arrhythmias, and various cardiovascular events. It is a long-lasting condition that significantly influences the life of the individual in many aspects.[3] For the diagnosis of OSA, polysomnography (PSG) is the gold standard, and at present there is no satisfactory alternative to it.[4]

From the point of view of an ear, nose, and throat (ENT) surgeon, patients with OSA generally experience considerable hypoxemic situations as a sequela of recurrent apnea events. The chronic hypoxemia and variations in the concentration of oxygen in the blood noted in OSA patients may be harmful to the process of auditory transduction and transmission. The sense of hearing is made possible by peripheral and central auditory processing, both of which are at risk because of the effects of hypoxemia.[5] Obstructive apnea-hypopnea can hamper the generation and transmission of nerve impulses at the level of the auditory system, as the transduction process of the inner ear and transmission of nerve impulses along the auditory pathway are highly dependent on the oxygen supply.[6]

There are only a few studies that identify a link between OSA and hearing; however, their results are inconsistent. The present study was designed to analyze the auditory function in cases of OSA.

Materials and Methods

The present hospital-based, observational, analytical, cross-sectional study was conducted over a period of one year after obtaining approval from the institutional ethical board. Cases and controls between the ages of 20 to 50 years and matched for age and gender were enrolled after providing written informed consent. Patients with any chronic diseases (such as hypertension, diabetes mellitus, hypothyroidism, chronic kidney failure, congestive heart failure, hepatic illness), middle-ear disease, conductive hearing loss, history of previous ear surgery, any neurological disease, psychiatric disorders, acoustic neuroma, brain tumor, head and neck radiation exposure, occupational noise exposure, history of acoustic trauma, autoimmune diseases, and any exposure to ototoxic substances were excluded from the study. After the exclusion criteria were applied, the patients were interviewed using the Berlin questionnaire, and those who with positive results were subjected to polysomnography (PSG). Those with an Apnea-Hypopnea Index (AHI) > 5 were labeled as the OSA patients and formed the case group, while the rest composed the control group. Cases and controls were further evaluated through impedance and pure-tone audiometry (PTA).

The PSG examination was performed by trained technicians using a 16-channel polysomnograph (Alice LE, Philips Respironics, Pittsburgh, PA, Unite States). Various parameters were observed, including electroencephalogram, electrooculogram, and electrocardiogram results, nasal-oral airflow measurement by a pressure transducer, snoring, chin-leg electromyogram result, transcutaneous oxygen saturation, and chest-abdominal wall motion by respiratory inductive plethysmography. The data obtained on sleep were scored manually according to standard criteria.

Impedance audiometry (tympanogram and acoustic reflex) was performed using the GSI 38 AutoTymp (Grason-Stadler, Milford, NH, United States) device in a setup with ambient noise levels < 30 dB. Subjects with type-A tympanograms and present acoustic reflex were included in the study to eliminate those who had pathological conditions in the middle ear. The PTA examination was performed in all the subjects using the AC 40 clinical audiometer (Interacoustic A/S, Middelfart, Denmark) in a fully sound-attenuated chamber with ambient noise < 30 dB. The audiometry variables of each patient were evaluated, such as low-frequency audiometry (LFA) from 0.5 kHz to 2 kHz; high-frequency audiometry (HFA) from 4 kHz to 8 kHz; and extended high-frequency audiometry (EHFA) from 10 kHz to 16 kHz.

The data were entered and cleaned using an Excel (Microsoft Corp., Redmond, WA, United States) spreadsheet. The statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, United States) software, version 17.0. The quantitative variables were expressed as means $\pm$ standard deviations (SDs), while the categorical variables were expressed as proportions (%). The Student *t*-test was applied after checking for the normalcy of the data. Values of $p \leq 0.05$ were considered statistically significant.

Observation and Results

We studied 35 cases and 23 controls (total sample of 58 patients) with mean respective ages of 36.88 ± 4.16 years and 35.35 ± 4.55 years ($p = 0.192$). There was a predominance of male patients in a ratio of 5:1 ($p = 0.289$); the mean body mass index (BMI) of the controls and cases was of 26.94 ± 2.20 Kg/m^2 and 29.30 ± 2.41 Kg/m^2 respectively ($p < 0.0001$);and the mean neck circumference of the controls and cases was of 38.09 ± 2.63 cm and 40.59 ± 2.16 cm respectively ($p < 0.0001$) (►**Table 1**).

The mean PTA thresholds of the controls at 0.5 kHz, 1 kHz, 2 kHz, 4 kHz, and 6 kHz in the right and left ears were of 15.61, 16.30, 19.17, 21.00, and 22.96, and of 15.61, 17.17, 20.30, 21.87, and 23.48 respectively. As for the cases, these same values were of 16.28, 16.71, 20.14, 21.66, and 22.77, and of 16.51, 17.88, 20.88, 22.26, 23.88 respectively. In the comparison between groups, these thresholds were not significantly different. The mean PTA thresholds at 8 kHz in the right and left ears of the controls were of 25.74 and 25.65 respectively, and, for the cases, they were of 26.20 and 26.14 respectively. Therefore, the PTA thresholds in the left ears were significantly higher in the case group compared to the controls. The mean PTA thresholds at 10 kHz, 12 kHz, 14 kHz, and 16 kHz in the right and left ears of the controls were of 27.87, 33.39, 38.83, and 54.96, and of 27.96, 33.74, 39.39, and 55.00 respectively. As for the cases, these same values were of 28.68, 34.46, 41.57, and 59.17, and of 29.08, 34.97, 41.68, and 59.54 respectively. Therefore, the PTA

Table 1 General characteristics of the study participants ($N = 58$)

Variables	Control group ($n = 23$)	Case group ($n = 35$)	p-value
Mean age (in years)	35.35 ± 4.55	36.88 ± 4.16	0.192
Gender (male:female)	18:5	31:4	0.289
Mean weight (in Kg)	77.74 ± 8.75	85.71 ± 9.62	**0.002**
Mean height (in cm)	1.70 ± 0.06	1.71 ± 0.04	0.445
Mean body mass index (Kg/m^2)	26.94 ± 2.20	29.30 ± 2.41	$<$ **0.001**
Mean neck circumference (in cm)	38.09 ± 2.63	40.59 ± 2.16	$<$ **0.001**

thresholds in both ears were significantly higher in cases in comparison to controls at the aforementioned frequencies (►**Table 2**).

Discussion

There has been a little research in OSA patients using hearing tests such as the PTA at higher frequencies. In the present study, we found that, at higher frequencies, the PTA thresholds of the cases were significantly higher than those of the controls. Hwang et al.[7] (2011) conducted a study which showed that OSA had no obvious positive relationship with PTA thresholds at low and high frequencies of all subjects. Conversely, there was an obvious negative relationship between OSA and pitch pattern sequence (PPS) scores for all subjects, even after adjusting for age, gender, and other variables. Thus, the present study is not consistent with this study.

In the study, the mean ages of the controls and cases were of 35.35 ± 4.55 years and of 36.88 ± 4.16 years respectively. We observed that the mean values regarding weight, BMI, and neck circumference of the cases was significantly higher than those of the controls, with $p = 0.002$, $p < 0.0001$, and $p < 0.0001$ respectively. We also found a predominance of OSA among male patients in a ratio of 5.44:1. Sheu et al.[8] (2012) performed a population based case-control study and noticed that male individuals with sudden sensorineural hearing loss (SSNHL) were more likely to have a history of OSA than the controls (odds ratio: 1.48; 95% confidence interval: 1.02–2.16; $p = 0.04$). But no such relationship was present in the female individuals.

In the present study, we observed that the PTA thresholds at frequencies of up to 6 kHz in both ears were not significantly different between cases and controls, whereas the threshold in both ears at frequencies ranging from 8 kHz to 16 kHz were significantly higher in the cases compared to the controls. Mohammadi et al.[9] (2016) evaluated the hearing functions in patients with severe sleep apnea, and they found that the mean hearing thresholds of cases and controls at frequencies ranging from 250 Hz to 8 kHz were of 24.44 ± 6.80 dB and 15.75 ± 5.10 dB respectively ($p < 0.01$). They noticed that there was a significant relationship between hearing loss and the severity of the OSA.

We observed that the PTA thresholds of the whole sample at frequencies of up to 6 kHz in both ears was not significantly different, but, at frequencies ranging from 8 kHz to 16 kHz, they were significantly higher in the cases compared to the controls. Martines et al.[10] (2016) conducted a study with 160 individuals and noticed a rate of hearing loss of 41.66% among individuals with moderate to severe degrees of OSA ($p < 0.0001$). All groups were described by a mean hearing threshold < 25 dBHL for 250 Hz –3 kHz frequencies and a continuous decrease in hearing sensitivity, specifically for 6–16 kHz frequencies ($p < 0.05$). Concluded that development of an early cochlear damage and a more marked high-frequency hearing loss in case of severe OSA ($p < 0.05$).

In the present study, the PTA threshold at higher frequencies was significantly higher in the case group, and hearing loss was observed at higher frequencies in some individuals. Chopra et al.[11] (2016), in a multicenter population-based study, observed a dose–response association between the severity of the AHI index and any hearing impairment, that is, the more severe the sleep apnea, the more severe the hearing impairment. They concluded that sleep apnea is associated with high- and low-frequency hearing impairments, independent of snoring and other confounders. Ekin et al.[6] (2016) conducted a study with 66 participants, and the data showed that snoring may cause hearing loss at extended high frequencies. In the present study, the PTA thresholds in both ears were significantly higher in the case group at extended high frequencies ranging from 10 kHz to 16 kHz. Therefore, the present study is in line with the one by Ekin et al.[6]

Conclusion

There is evidence that the auditory mechanism is affected in OSA patients at extended high frequencies ranging from 10 kHz to 16 Hkz. We recommend early auditory screening of OSA patients for early diagnosis and health education through Behavior Change Communication about its prevention. So, with the early use of the various treatment modalities for OSA, further detrimental effects on the auditory system can be prevented. A further larger study will be helpful to confirm the association of hearing loss and OSA using a battery of audiological tests (such as, otoacoustic emissions, brainstem-evoked response audiometry etc.) to detect early neuronal and vascular dysfunction of the auditory pathway.

Table 2 Pure-tone audiometry at various frequencies

			Mean	Standard deviation	*p*-value
0.5 kHz	Right ear	Controls ($n = 23$)	15.61	± 2.59	0.260
		Cases ($n = 35$)	16.28	± 1.93	
	Left ear	Controls ($n = 23$)	15.61	± 2.48	0.092
		Cases ($n = 35$)	16.51	± 1.54	
1 kHz	Right ear	Controls ($n = 23$)	16.30	± 2.65	0.483
		Cases ($n = 35$)	16.71	± 1.77	
	Left ear	Controls ($n = 23$)	17.17	± 2.40	0.185
		Cases ($n = 35$)	17.88	± 1.64	
2 kHz	Right ear	Controls ($n = 23$)	19.17	± 1.94	0.098
		Cases ($n = 35$)	20.14	± 2.26	
	Left ear	Controls ($n = 23$)	20.30	± 2.10	0.308
		Cases ($n = 35$)	20.88	± 2.11	
4 kHz	Right ear	Controls ($n = 23$)	21.00	± 1.88	0.240
		Cases ($n = 35$)	21.66	± 2.17	
	Left ear	Controls ($n = 23$)	21.87	± 1.71	0.407
		Cases ($n = 35$)	22.26	± 1.74	
6 kHz	Right ear	Controls ($n = 23$)	22.96	± 1.84	0.706
		Cases ($n = 35$)	22.77	± 1.80	
	Left ear	Controls ($n = 23$)	23.48	± 1.38	0.218
		Cases ($n = 35$)	23.88	± 1.10	
8 kHz	Right ear	Controls ($n = 23$)	25.74	± 0.62	0.075
		Cases ($n = 35$)	26.20	± 1.10	
	Left ear	Controls ($n = 23$)	25.65	± 0.77	**0.022**
		Cases ($n = 35$)	26.14	± 0.77	
10 kHz	Right ear	Controls ($n = 23$)	27.87	± 0.76	**0.004**
		Cases ($n = 35$)	28.68	± 1.13	
	Left ear	Controls ($n = 23$)	27.96	± 0.70	**< 0.001**
		Cases ($n = 35$)	29.08	± 1.29	
12 kHz	Right ear	Controls ($n = 23$)	33.39	± 0.89	**0.003**
		Cases ($n = 35$)	34.46	± 1.46	
	Left ear	Controls ($n = 23$)	33.74	± 0.75	**< 0.001**
		Cases ($n = 35$)	34.97	± 1.32	
14 kHz	Right ear	Controls ($n = 23$)	38.83	± 1.03	**< 0.001**
		Cases ($n = 35$)	41.57	± 2.90	
	Left ear	Controls ($n = 23$)	39.39	± 1.53	**0.002**
		Cases ($n = 35$)	41.68	± 3.19	
16 kHz	Right ear	Controls ($n = 23$)	54.96	± 2.80	**0.001**
		Cases ($n = 35$)	59.17	± 5.00	
	Left ear	Controls ($n = 23$)	55.00	± 4.17	**0.001**
		Cases ($n = 35$)	59.54	± 5.12	

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Conflict of Interests
The authors have no conflict of interests to declare.

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