ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA AND HEARING LOSS: A LITERARY REVIEW

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ABSTRACT

Objective: *Obstructive Sleep Apnea Syndrome (OSAS) is a syndrome characterized by obstruction of upper airways with several episodes of apnea and hypopnea. There are many consequences reported in literature, among them, cochlea seems to be damaged by lack of oxygenation with possible auditory loss. The aim of this work is to summarize the effect of OSAS on cochlear function and the factors, which determine it.*

Methods: *We have selected found on PubMed by searching with the keywords "hearing loss" and "obstructive sleep apnea". We found 11 manuscript, but only 4 satisfied inclusion criteria. We reviewed 4 manuscript that enrolled a total of 200 patient. Our review evaluated percentage of tinnitus, pure tone average for the frequencies from 250 to 8000 Hz and from 9000 to 16000 Hz, Transient-evoked otoacoustic emissions (TEOAE) for each ear, Distortion product otoacoustic emissions (DPOAEs), Latency of the V wave in Auditory Brainstem Response (ABR).*

Results: *The 57.14% of moderate to severe OSAS reported tinnitus from tinnitus with respect to the 31.03% of mild OSAS (P=0.024). Higher percentage of hearing loss (41.66%) was found in patient with severe and moderate OSAS (P<0.0001) particular for 6-16 kHz frequencies (P<0.05). Otoacoustic emissions evidenced a difference between patients with simple snoring and patients with severe OSAS for 3 and 4 kHz frequencies (p<0.05).*

Conclusion: *These data show that snoring may cause hearing loss at extended high frequencies. Oxidative stress and ROS have been implicated in the activation of the inflammatory response with cytokines and chemokines, endothelial dysfunction and vascular damage, which can contribute to the development of sensory neural hearing loss.*

Keywords: *OSAS, hearing loss, Obstructive Sleep Apnea, review.*

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Introduction

Obstractive sleep apnea syndrome (OSAS) is a disease characterized by obstruction of the upper airways with frequently episodes of hypopnea and/or apnea which leads sleep alteration, hypoxia and hypercapnia with reduction of peripheral perfusion. This circulatory alteration has been associated with cardiovascular diseases and stroke risk. Some authors highlighted the effects of sleep apnea episodies with an increased incidents of hearing disorders. Hypoxia and reduction of blood flow may damage cochlear cells because of anatomy of the hear with absence of collateral circulation. Many authors have talked about cochlear damage from hypoxemia caused by OSAS.

For example M. Deniz et al.[1-4] studied the impact of hypoxiemia on the auditory function of OSAS patients evidenced that patients who present moderate and severe OSAS should prevent episodes of hypossemia to protect cochlea and avoid hearing loss.

OSAS is a common disease affecting 3-7% of middle aged man and 2-5% middle aged woman[5-7] characterized by obstruction of upper airways with cycles of hypopnea/apnea. Patients refer symptoms such as: heahache, daytime sleepness, reduced concentration and memory, endocrine and metabolic alteration. The most frequent causes of OSAS are: reduction of the tone of the pharyngeal dilatator muscles, obstraction of velopharynx, tongue base and lateral walls of pharynx.

Other predisposing factors are: obstruction of nasal cavities, modification of palate, jow and tongue, tonsillar hypertrophy, obesity, drugs and alcohol abuse.

According to AASM's (American accademy of sleep medicine) 2007 criteria, apnea is a an interruption of breathing during sleep (>90% decrease in airflow), with persistence of toracic and/or abdominal movements, leading to a decrease in the arterial blood oxygen saturation.

Hypopnea is defined by >30% decrease of airflow that leads to a reduction in the blood oxygen saturation > 4% or by >50% reduction in the airflow that leads >3% reduction in blood oxygen saturation. These episodes must last for at least 10 seconds and no longer than 3 minutes[5].

According with international guidelines OSAS can be classificated with AHI index (apnea/hypopnea): mild (5>AHI>15), moderate (15>AHI>30) , severe (AHI>30). While an AHI <5 is considered of simple snoring without OSAS.

Literature data evidenced the prevalence of OSAS in middle aged man and the difference between genders is related to hormonal factors, different distribution of body fat and anatomical differences of the upper airways[8-11]

Also obesity, drugs and and alcohol are factor that increased the situation of airway obstruction and reduce the tone of the faringeal muscles that bring back the tongue base aggravating the consequent obstruction of the upper airways.

Materials and methods

We have selected articles about hearing loss in patients with OSAS. All articles were found on PubMed by searching with the keywords "hearing loss" and "obstructive sleep apnea". We included manuscript with full text access, in English language, with numeric data about audiological profile. We found 11 manuscript, but only 4 satisfied inclusion criteria. We reviewed 4 manuscript that enrolled a total of 200 patient.

Our review evaluated the following evaluation parameters:

- Percentage of tinnitus.
- Audiometric threshold was considered as the pure tone average (PTA) for the frequencies from 250 to 8000 Hz.
- PTA from 9000 to 16000 Hz.
- Transient-evoked otoacoustic emissions (TEOAE) for each ear, expressed as percentage of PASS.
- Distortion product otoacoustic emissions (DPOAEs), expressed as mean of mean of amplitude.
- Latency of the V wave in Auditory Brainstem Responce (ABR).
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Results

Martines et al. examined one hundred and seventy two snoring patients from January 2012 to June 2014 at the Hearing Section of Department of Bio-technology of Palermo University. They were divided in four groups: simple snoring, mild OSAS, moderate OSAS and severe OSAS. The patients underwent micro-otoscopy, audiometric examination, acufenometry, TEOAE recording. The 57.14% of moderate to severe OSAS reported tinnitus from tinnitus with respect to the 31.03% of mild OSAS (P=0.024). Higher percentage of hearing loss (41.66%) was found in patient with severe and moderate OSAS (P<0.0001) particulary for 6-16 kHz frequencies (P<0.05). Otoacoustic emissions evidenced a difference between patients with simple snoring and patients with severe OSAS for 3 and 4 kHz frequencies (p<0.05)[5].

Casale et al. studied audiological exams of 18 individuals with severe OSAS highlight a prolongation of wave latencies of waves V and also a compromission of higher PTA.

In Ekin et al. manuscript, statistically significant differences were found between the patient group and the control concerning the extended high frequency of hearing.

Hwang et al. analyzed only the PTA up to 8 khz, not finding statistically significant differences between cases and controls.

The results are summarized in Table 1.

Author	N° patients	Tinnitus	PTA (0,25-8 kHz) dB HL	PTA (9-16 kHz) dB HL	TEOAEs (PASS percentage)	DPOAEs (amplitude)	ABR (Wave 5 latency)
Martines et al.	Mild OSAS 58	18	23.25	46.38	24%	/	/
	Moderate OSAS 18	9	27.05	64.97		/	/
	Severe OSAS 24	15	26.83	67.43		/	/
Casale et al.	39	/	14.23	/	57%	5.96	5.84
Ekin et al.	27	/	23.27	42.65	/	/	/
Hwang et al.	34	/	26.2	/	/	/	/

Table 1: Data collection e analysis.

Discussion

Audiological Profile

Many report evidenced an association among OSAS and hearing loss.

Muchnik et al. ad Kotterba et al. studying the auditory brainstein response (ABR). The first one collected data from a sample of 79 OSAS patients highlighting increase in the latency between waves I-III and I-V in moderate and severe OSAS subgroup[12-16]. The others instead analyzing 20 patients affected by severe OSAS found a prolonged trasmission time in the wave latency I and interpeak latency I-V in the 60% of the patients[17-21].

Also Casale et al. comparing audiological exams of 18 individuals with severe OSAS and 21 simple snoring patients highlight a prolongation of wave latencies of waves I,III e V and also a compromission of higher pure tone audiometry (PTA) in OSAS group [22-26]. But Chopra et al. evidenced hearing loss not only in the high frequencies but also in the low ones[27-32].

Matsumura et al. investigated the association between OSAS and middle ear acoustic transference and cochlea function. All subjets were submitted to polysomnography, evaluation of wideband acoustic immitance by energy of absorbance (EA), and distortion product otoacoustic emission (DPOAE). They observed that acoustic transference function of middle ear is similar in adults with and without OSAS. Instead severe OSAS impair cochear function, these patients showned lower DPOAE amplitutude values in all frequencies analyzed but mostly in high frequencies because hair cells located on cochea basal region seem to be more suscettible to episodes of hypoxia[33-37].

In a Sardesai et all. study, another aspect is considered, hypoacusis due to the noise of snoring partners. They evidenced an unilateral high frequency pattern of hearing loss associated with nose induced hearing loss but the affected ear was the one cronically exposed to snoring noise[38-41].

In Ekin et al. manuscript, polysomnography and audiometric examination were performed in all participants. 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.

Other studies instead have shown that OSAS does not predispose to cochlear damage and hearing loss

Spinosi et al. evaluated audiological performance of patients with simple snoring and mild OSAS to investigate the impact of this conditions on auditory function, considering the significant levels of chronic noise. They analized standard tonal audiometric exams (250-8000 Hz) in the Otorinolaringologist Clinic of Policlinico Santa Maria alle Scotte in Siena between September 2015 and April 2016 excluding patients with acute inflammation of the ear ,patients with other patologies of the middle and internal ear and patients previously exposed to acoustic trauma or chronic noise. They excluded also patients older than 65 years old. The study included 80 subjects: thirty non snoring patients as control group, 15 simple snorers and 35 patients with mild OSAS. In this study they evidenced an indipendence between hearing loss in simple snoring and mild OSAS patients when compared to non snoring patients in terms of hearing performance highlighting that chronic exposure to noise does not seem to be a predisposing or causating factor of hearing loss in these patients[42-44]. This idea has been confirmed Huwang et al. in his study of 34 OSAS patients which reported no significant positive association with PTA low or PTA high for all subjects.

Another aspect analized is the correlation between snoring and presbycusis with audiometry test. Hoffestein et al. have not dimostrated a significant impact on hearing loss showning that snoring is not a factor that leads hearing loss[45-49].

Ros and cochlear damage

OSAS is a condition caracterized by intermittent obstruction of upper airways with episodies of apnea and or hypopnea that produced desaturations and altered sleep pattern. Cicles of hypoxia and normoxia influenced the production of reactive oxygen species (ROS) and the promotion of endotelial damage, activation of inflammatory cascades with vascular dysfunction and impaired peripheral perfusion.

Therefore OSAS may lead vascular insufficiency with blood flow alteration during episodies of apnoea and also ischemic injury to the cochlea.

Inner ear and acoustic nerve are sensitive to oxygen level change and specifically the cochlea depend on this alteration because of its terminal circulation with insufficient collateral circulation.

Martines et al. have measured reactive oxygen metabolites (ROM's) levels in serum samples by Diacron reactive oxygen metabolites (d-ROM) test. In this work they showed higher level of ROS in patients with severe OSAS with respect to simple snoring patients. This data evidenced the significant role of hypoxia caused by apnoea in the development of cochlear damage and high frequency hearing loss and tinnitus in case of severe OSAS[8].

Oxidative stress and ROS have been implicated in the transcriptional activation of nuclear factor kB (NF-kB) wich leads to the activation of the inflammatory

response with cytokines and chemokines (because of presence of their receptor in cochlear tissue) , endothelial disfunction and vascular damage wich can contribute to the development of sudden sensory neural hearing loss (SSNHL)[50].

Osas and sudden hearing loss

Sudden sensoryneural hearing loss (SSNHL) commonly known as sudden deafness is a rapid loss of hearing develop within a few hours. It is still unclear what exactly causes sudden deafness. Some possible causes include: viral infection or reactivation, arteriosclerosis and circulatory disorders, metabolic disorders such as diabetes or hypercholesterolemia, thrombosis of the inner ear vessel's, autoimmune diseases or previosus acute otitis media.

Since OSAS causes alterations in circulation and therefore tissue oxygenation, some authors deduce that sudden hearing loss may be related to this syndrome.As reported by Jau-Juan Sheu et al. there is a statistically significant higher proportion of OSAS in patient with SSNHL[50].

This association is more evident in male subjects but not in women. The mechanism is unclear, but it is hypothesizes that it is related to an underdiagnosis of OSAS in women. Also Fisher at al. highlights the correlation between the development OSAS and sudden hearing loss. Patients which develop sudden hearing loss tend to have OSAS more frequently than those in the control group owing to the similar risk factors for cerebral infarction and sudden hearing loss[51].

Conclusion

The present REVIEW, examining the inner ear function of patients with OSAS, underlines the key role of chronic nocturnal intermittent hypoxia in the development of auditory dysfunction and demonstrates a more marked high-frequency hearing loss in case of severe OSAS. Oxidative stress and ROS have been implicated in the activation of the inflammatory response with cytokines and chemokines, endothelial dysfunction and vascular damage, which can contribute to the development of sensory neural hearing loss.

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