

A Case of a Sudden Hearing Loss in a Patient with Undiagnosed Severe Sleep Apnea: More than Just a Coincidence

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Abstract

Obstructive sleep apnea (OSA) is a common disorder established as an independent risk factor of cardiovascular morbidity and mortality. Cases of hearing impairment associated with sleep apnea have been described and multiple studies have tried to find a significant relationship between both disorders. Many pathophysiological pathway mechanisms have subsequently been suggested. We present a case of a 32-year-old male Tunisian patient with no relevant pathological history apart from smoking and obesity, who presented with a sudden sensorineural hearing loss (SSHL). He had a two-year-history of asthenia and daytime sleepiness. Physical examination was normal and so were the laboratory findings. Further investigations concluded that it was idiopathic sensorineural hearing loss and severe obstructive sleep apnea. The patient was treated with a continuous positive airway pressure device (CPAP). The asthenia and sleepiness notably improved. The right hearing impairment remained stable with a normal left hearing function.

Keywords Sleep Apnea; Hearing Loss

Introduction

Obstructive sleep apnea (OSA) is a common condition affecting 2 to 4% of middle-aged population [1]. Numerous serious complications have been associated with OSA including arterial hypertension, heart failure, cardiac arrhythmias, stroke and sudden death [1,2]. It is now considered as an independent risk factor of cardiovascular and cerebrovascular morbidity and mortality [1]. Besides, cases of hearing impairment associated with OSA have been described. Studies of the relationship between both disorders are not unanimous.

Sudden sensorineural hearing loss SSHL is commonly defined as an impairment greater than 30 dB occurring within a three-day period [3]. Identified causes of SSHL can be bacterial (syphilis, meningitis), viral (cytomegalovirus, mumps, varicella/zoster), inflammatory (sarcoidosis, Cogan syndrome, granulomatous with polyangiitis), vascular (hypercoagulability states, emboli, post radiation therapy), tumoral, traumatic or toxic/iatrogenic [3].

We hereby present the case of a 32-year-old male patient with a 2-years-history of untreated severe sleep apnea who suddenly developed a unilateral hearing loss.

Case Report

A 32-year-old Tunisian male patient presented to our clinic with a sudden right sensorineural hearing loss (confirmed by audiometric tests) that had occurred 3 weeks before. He had no medical history apart from smoking. There was no relevant history of trauma or of recent drug intake. Clinical examination didn't reveal any abnormality. The patient weighed 94 kg. His body mass index was 32.14 kg/m². Initial laboratory tests showed a normal complete blood cell count, ESR level at 35 mm and normal hepatic and renal functions. Magnetic resonance imaging (MRI) with gadolinium enhancement along with a

computed tomography (CT) scan excluded any responsible tumor or mass. Further laboratory findings including fasting blood glucose level, thyroid-stimulating hormone, fluorescent treponemal antibody-absorption (FTA-Abs), Cytomegalovirus serology, antinuclear antibodies (ANA), anticardiolipin antibodies and antiB2 glycoprotein1 antibody were all normal. On the second consultation, the patient was notably sleepy. He complained of asthenia and daytime sleepiness that had started 2 years before. Polysomnographic findings confirmed the diagnosis of severe obstructive sleep apnea with an apnea-hypopnea index of 49.1. The patient was treated with a continuous positive airway pressure device (CPAP). The asthenia and the sleepiness notably improved. The right hearing impairment remained stable with a normal left hearing function.

Discussion

We present a case of undiagnosed severe obstructive sleep apnea OSA in a patient who develops a sudden hearing loss. The association of OSA with hearing impairment is nowadays believed to be more than just a coincidence. Even if the early German series in 2003 didn't find any statistical difference when comparing the prevalence of OSA in people with hearing loss and control group [4], more recent cohorts were affirmative of a putative association between the two disorders. In fact, a Taiwanese case-control study in 2012 assessed data of 3192 patients with SSHL and 15960 controls and found that men with SSHL were more likely to have prior OSA [5]. Assessment of female patients in this study didn't find a similar result. Another American multicenter population-based study in 2016 assessing 13967 participants, concluded that OSA was associated with hearing impairment at all frequencies [6]. Furthermore, studies assessing patients with mild, moderate and severe OSA concluded that severe OSA was associated with sudden hearing loss, independently of other confounders [7,8].

Upon these conclusions, many hypotheses have been suggested to identify the underlying mechanisms. SSHL definition englobes hearing impairment of defined and undefined causes [3]. More than half of the

cases remain idiopathic, described as a “frustrating endpoint” for these patients [3] and so was our case. Multiple risk factors for idiopathic SSHL were however identified in 2015 in a study assessing 118 patients with a first occurrence of idiopathic SSHL and 415 healthy controls [9]. It concluded that, along with deficiencies of antithrombin or protein C or S, the presence of cardiovascular risk factors (arterial hypertension, diabetes mellitus, hyperlipidemia, smoking) were likely to increase the risk for idiopathic SSHL. Following this scheme, OSA is already established as an independent cardiovascular risk factor confirmed by epidemiological, clinical and pathological studies [1]. Therefore, in our case, the patient was at risk for SSHL not only because of smoking and obesity but also due to his untreated severe OSA. On a pathophysiological level, studies on idiopathic SSHL have identified 4 theoretical pathways that can account, mutually but not individually, for the hearing impairment: viral infection, vascular compromise, intrachochlear membrane rupture and immune-mediated inner ear disease [3]. Three of these 4 mechanisms were suggested to be enhanced by OSA (mechanisms 2,3 and 4).

In fact, OSA is characterized by numerous episodes of apnea and/or hypopnea causing intermittent hypoxia and repetitive changes of pressure in the upper airways. Both mechanisms can be related to SSHL. On the one hand, intermittent hypoxia causes fluctuations in blood flow, including cochlear. As cochlear function is extremely sensitive to oxygen supply [3], hearing can be compromised by intermittent hypoxia (mechanism 2). It is also established that intermittent hypoxia followed by phases of reperfusion is responsible for vascular inflammation and oxidative stress [1]. We can presume that if untreated, severe OSA causes chronic vascular inflammation and therefore immune-mediated disorders can occur (mechanism 4). Knowing that chronic OSA has already been suggested as a risk factor for auto-immune diseases [10], further studies in this matter are needed to confirm this hypothesis. On the other hand, it has been assessed that fluctuation of pressure in the upper airways causes imbalance of pressure in the Eustachian tube, leading chronically to irreversible alterations in the middle ear and compromising the acoustic transference [7]. Although this mechanism would not lead to membrane rupture, it can alter the endocochlear potential and therefore endangers the hearing function (mechanism 3).

Another hypothesis on the relationship between OSA and SSHL was suggested by a Turkish study assessing 66 participants; 27 in the OSA group, 18 in the simple snoring group and 21 in the control group, and concluded that snoring may cause hearing impairment at extremely high frequencies [11].

Finally, treatment of OSA in these cases is crucial, not only to prevent classic complications of OSA but also to preserve the contralateral ear function and so was the case for our patient.

Conclusion

Our case emphasizes on the importance of early diagnosis of OSA, since it is established that severe and untreated OSA is related to hearing impairment. Many plausible hypotheses have been suggested in this matter. The best therapeutic strategy of SSHL in these cases is therefore the early treatment of OSA.

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