Otic Barotrauma Resulting from Continuous Positive Airway Pressure: Case Report and Literature Review

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Abstract
Obstructive sleep apnea (OSA) is an ever-increasing problem affecting millions of people in the United States. The prevalence of OSA has risen drastically over the past few decades concurrently with the increasing prevalence of obesity. Subsequently, there has been an ever-increasing rise in the use of continuous positive airway pressure (CPAP) devices. While there are many adverse effects to the use of CPAP, the majority are described as being relatively benign. Here we discuss a case of significant sudden sensorineural hearing loss in relation to a suspected perilymphatic fistula, caused by traumatic barotrauma resulting from excessive self-titration of CPAP in an in-home setting.

Introduction
Obstructive sleep apnea (OSA) is a common, yet under recognized condition. It is estimated that some form of OSA affects approximately 17% of the adult population. Over the years, this number has consistently increased possibly secondary to the rising prevalence of obesity in the population [1].

The traditional treatment for patients with OSA is continuous positive airway pressure (CPAP) [2]. CPAP prevents the collapse of the upper airway by providing continuous positive pressure to the oro- and naso-pharynx. The pressure required for individual patients is variable, and is determined by careful manual titration of pressures during a CPAP titration study. The goal of the titration study is to establish the minimal pressure at which snoring is cessation of snoring and the absence of apneic or hypopneic events [2].

Adverse effects from CPAP use are numerous, but most commonly include congestion, dryness of the oral and nasal cavities, aerophagia, epistaxis, and sinusitis [3]. In this paper we present a case of otic barotrauma resulting from excessive self-titration of CPAP in an in-home setting.

Case Report
A 50-year-old obese male presented initially in 2007 with a complaint of snoring. He reported that in 2005 he was diagnosed with obstructive sleep apnea. Polysomnography at that time of diagnosis revealed an apnea-hypopnea index of 57.5 with oxygen desaturations to 84%. He had never been titrated for CPAP therapy and returned to the clinic at this time because of worsening symptoms. The patient subsequently underwent a CPAP titration study. Snoring and apneic events were eliminated at a pressure of 15cm H2O.

The patient was seen intermittently over the next four years, until 2011 when the patient presented to the clinic with a one week history of sudden hearing loss with the sensation of aural fullness, pain, and “pop” sound in the right ear that began suddenly after excessive self-titration of CPAP. He also elicited sudden onset tinnitus, but denied any symptoms of vertigo. On exam there was no perforation of the tympanic membrane, however, there was evidence of a significant clear serous effusion. Further questioning revealed a history of weight gain and subjective improvement in his OSA symptoms as he systematically increased the pressure on his CPAP machine.

On the day that his hearing loss began, he recalled turning the CPAP up to the maximum allowed setting. Audiograms were performed and compared with baseline audiograms (Fig. 1A), performed in 2007. Audiometry revealed significant mixed hearing loss in the right ear (Fig. 1B). A CT scan of the temporal bones showed complete opacification of the right middle ear cavity and right mastoid air cells (Fig. 2). The ossicles were not visualized in either the middle ear or in the mastoid. The left ear appeared to be normal. The patient was started on a prednisone taper and ciprofloxacin, but had no improvement in hearing or tinnitus with follow up 3 days later. A repeat audiogram performed 1 week after initial presentation did, however, reveal modest improvement in hearing (Fig. 1C). At follow up 2 weeks after presentation, a repeat audiogram demonstrated more significant improvement in hearing (Fig. 1D). The patient was followed on an as needed basis thereafter. A perilymphatic fistula was suspected, however, the patient elected not to undergo exploratory tympanotomy because of the improvement in his hearing loss.

Discussion
Over the last few decades the prevalence of OSA has been steadily increasing, with the increased prevalence of obesity thought to be the main contributor [1]. There are numerous health risks associated with OSA, increasing the importance of diagnosis and treatment of this disease. The main treatment of OSA is the use of CPAP, typically titrated during polysomnography to determine the minimum pressure at which apneic and/or hypopneic events are effectively eliminated. The first set of clinical guidelines created in effort to standardize the manual titration of CPAP was established in 2008 [2].

The risks of CPAP therapy should also be considered when deciding on treatment options for patients with OSA. The main side effects of CPAP therapy are typically mild, and include dryness of the nasal and oral mucosa, sneezing, congestion, aerophagia, sinusitis, and epistaxis. However, more serious adverse effects, including pneumonia, pulmonary barotrauma, intraocular hypertension, and subcutaneous emphysema, may occur [3]. The effects of positive airway pressure on the ear should not be overlooked as a potential consequence of CPAP therapy.

Otic barotrauma has been well described in air travel and deep sea diving, however, barotrauma as a result of CPAP has not been widely documented. Otic barotrauma results from a pressure differential between the middle ear and external environment [4]. While middle ear barotrauma can cause enough inflammation within the middle ear to cause a conductive hearing loss, it may also be transmitted to the inner ear causing a sensorineural hearing loss. This inner ear barotrauma may result from opening of the eustachian tube, resulting in a brisk inward movement of the round window, which may cause disruption of the cochlear membranes or lead to formation of a perilymph fistula [4]. A similar mechanism may occur with the use of CPAP, whereby a sudden increase in pressure in the middle ear, by way of the eustachian tube, disrupts the integrity of the oval or round window.

Yung demonstrated that there is indeed an increase in middle ear pressure during use of CPAP [5]. Lin et al. found that after swallowing, a maneuver which effectively opens the eustachian tube, during the use of CPAP increases middle ear pressures, and the increase in pressure was directly proportional to the pressure delivered via CPAP [6]. Furthermore, a study designed by Slivn, et al. determined that there was a significant increase in middle ear pressure after 6 months of CPAP therapy [7]. Thus, it has been determined that the use of CPAP increases pressure within the middle ear cavity.

Weaver, et al. described a case of bilateral tympanic membrane perforations as a result of CPAP [8]. Segal, et al. reported three cases of sudden sensorineural hearing loss in post-operative patients postulated to be secondary to the use of nitrous oxide (NO), which causes an increase in middle ear pressure, similar to what would be expected with CPAP, albeit via a different mechanism. On exploratory tympanotomy rupture of the round window and a perilymph fistula at the oval window were found in each of these patients [9]. Friedman and Sasaki described two cases of hearing loss occurring after resuscitative efforts in which positive pressure ventilation was used. The first patient developed hearing loss without tinnitus or vertigo. Audiogram revealed conductive hearing loss and exploratory tympanometry revealed incudomalleal discontinuity. The second patient developed severe unilateral hearing loss with tinnitus and dizziness. Audiogram confirmed sensorineural hearing loss and exploratory tympanometry revealed a leak of perilymph from the anterior margin of the stapes footplate [10].

References

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FIG 1A-B. Audiometric Analysis of Hearing Loss
Baseline audiogram (A) reveals normal hearing. Audiogram at initial visit (B) demonstrates moderate to severe mixed hearing loss in the right ear. Audiogram at the 1-week follow-up visit (C) shows some improvement in hearing, but still moderate to severe hearing loss at higher frequencies. Audiogram at the 2-week follow-up visit (D) with continued improvement in hearing.