KBB ve BBC Dergisi 24 (3):83-5, 2016

DOI: 10.24179/kbbbbc.2016-52879

Hyperbaric Oxygen in the Treatment of Streptomycin-Induced Ototoxicity: Case Report

Streptomisin Nedenli Ototoksisite Tedavisinde Hiperbarik Oksijen

Sinan ULUYOL, MD

Van Training and Research Hospital, Clinic of Otolaryngology Head & Neck Surgery, Van

ABSTRACT

Aminoglycosides can cause toxic side effects to the inner ear and kidneys, and aminoglycoside-induced hearing loss is usually considered irreversible. We report a case of ototoxicity coexisting with nephrotoxicity following streptomycin administration that was associated with improvements in patient's hearing loss after hyperbaric oxygen therapy. Clinical and treatment characteristics of this condition was discussed in light of the current literature.

Keywords

Hearing loss; hyperbaric oxygenation; streptomycin

ÖZET

Aminoglikozidler iç kulak ve böbreklerde toksik yan etkilere neden olabilir ve aminoglikozid nedenli işitme kaybı genellikle geri dönüşümsüz olarak kabul edilir. Bu çalışmada, hiperbarik oksijen tedavisinden sonra işitme kaybında düzelme olan, streptomisin uygulamasını takiben nefrotoksisitenin eşlik ettiği ototoksisite olgusu literatür eşliğinde tartışıldı.

Anahtar Sözcükler

Hiperbarik oksijenasyon; işitme kaybı; streptomisin

Çalışmanın Dergiye Ulaştığı Tarih: **13.08.2016** Çalışmanın Basıma Kabul Edildiği Tarih: **05.12.2016**

≈

Correspondence

Sinan ULUYOL

Van Training and Research Hospital,

Clinic of Otolaryngology Head & Neck Surgery,

Van, Turkey

e-mail: sinanuluyol@hotmail.com

INTRODUCTION

Streptomycin is effective for Gram-negative bacteria and Mycobacterium tuberculosis. In addition to its potent antimicrobial effect, it can cause toxic side effects to the inner ear and kidneys. Streptomycin ototoxicity is usually bilateral, and it affects high-frequency hearing (>8 kHz) first, followed by low-frequency hearing. Although nephrotoxicity is reversible, ototoxicity is usually permanent.[1,2] It has been known that aminoglycoside ototoxicity is related to alterations in the antioxidant mechanisms in the hair cells. Several studies showed that aminoglycosides appear to generate free radicals in the inner ear.[3-5] Thus, recent studies focused on developing strategies, and to finding out ways to prevent aminoglycoside-induced ototoxicity. Among them, anti-free radical agents are becoming increasingly important. Hyperbaric oxygen therapy (HBOT) has been shown to have a strong antioxidant activity. It has been considered as a valuable therapeutic tool due to its antioxidant characteristics.[6-8]

In this study, we report a case with simultaneous ototoxicity and nephrotoxicity following streptomycin administration. The hearing loss improved after HBOT. Clinical and treatment characteristics of this condition was discussed in light of the current literature.

CASE REPORT

A 44-year-old male patient was admitted to our clinic with the complaints of hearing loss, ear fullness, and tinnitus in his both ears.His symptoms started two days prior to his visit to our clinic, and he did not have any hearing problems previously. He had been prescribed streptomycin (1 g/day, intramuscular) due to brucellosis, and was on day 11 of this regimen. He did not have symptoms of imbalance, dizziness, vertigo, nausea, or vomiting. Except for streptomycin administration, there were no other significant points (e.g. acoustic/barotrauma, upper airway infection, other drug use, surgery) in his medical history. On physical examination, bilateral tympanic membranes and the ear canals were normal, Rinne test was positive for both ears, and the Weber test showed no lateralization. Nystagmus was not observed in any directions. Pure tone audiometry (PTA) revealed bilateral sensorineural hearing loss (SNHL) beginning at 4 kHz, and reaching to 70 dB at 8 kHz (Figure 1a). Blood chemistry showed urea and creatinine levels of 74 mg/dL and 2.17 mg/dL, respectively. The patient was consulted with a nephrologist, and he was subsequently diagnosed with streptomycin-induced nephrotoxicity. Streptomycin was stopped. HBOT was prescribed for treatment of hearing los,s because nephrotoxicity limited the use of corticosteroids or other medical treatment options. HBOT consisted of 2.5 atm 100% oxygen for 120 min every day (the standard protocol in our institution), and continued for 18 days. At the end of HBOT, PTA was performed. PTA results indicated bilateral SNHL starting at 6 kHz, and reaching to 40-45 dB at 8 kHz (Figure 1b). PTA performed three months after the onset of ototoxicity was similar to the PTA results just after HBOT. High frequency thresholds could not be obtained due to technical issues. Written informed consent was obtained from the patient prior to the presentation of this case report.

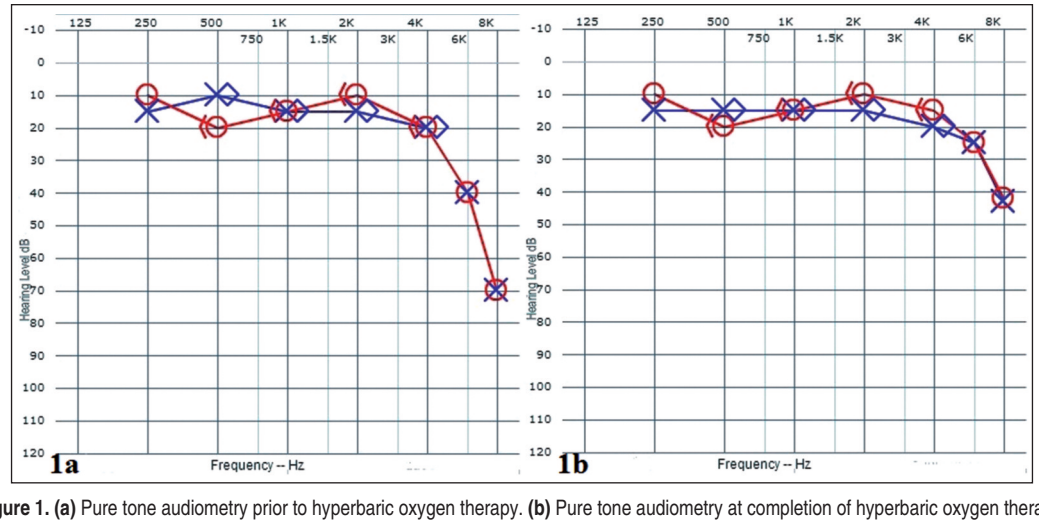

Figure 1. (a) Pure tone audiometry prior to hyperbaric oxygen therapy. **(b)** Pure tone audiometry at completion of hyperbaric oxygen therapy.

DISCUSSION

The biochemical and molecular mechanisms underlying aminoglycoside-induced ototoxicity remain poorly understood. This type of ototoxicity can be described on a cellular level as the destruction of cochlear hair cells. Histopathologic studies have shown that the outer hair cells are more sensitive to ototoxicity than the inner hair cells. Aminoglycosides appear to generate free radicals in the inner ear, with subsequent permanent damage to sensory cells and neurons, resulting in permanent hearing loss in the highest frequencies (>8000 Hz).[3-5]

HBOT is based on inhalation of 100% oxygen under a pressure >1 atm (range: 1.5-3.0 atm) in a pressure chamber. HBOT is usually the preferred treatment option for sudden SNHL and acute acoustic trauma. The cochlear effect of HBOT is still unclear and its mechanism of action is controversial. It has been shown that HBOT has an antioxidant activity, which consists of a positive contribution to the vascular supply of the inner ear and an increased oxygen delivery to the damaged cells.[9,10] In addition, HBOT enhances axonal regeneration and cellular proliferation.[11] To date, only one animal study reported treatment of streptomycin-induced ototoxicity with HBOT. However, in this model, the authors were unable to demonstrate any therapeutic effect of HBOT on ototoxicity.[12] In a similar study, the effects of HBOT on amikacin ototoxicity was investigated in guinea pigs in terms of morphology (scanning electron microscopy) and function (otoacoustic emissions and brainstem auditory evoked potentials). The authors reported that HBOT did not change the cochlear hair cell morphology or the electro-physiological thresholds of inner ear in the guinea pigs given amikacin.[7] Previously, only Yassuda et al. reported that HBOT had an otoprotector effect against cisplatin-induced ototoxicity in an experimental study.[8]

Aminoglycoside ototoxicity is possibly multifactorial, and further investigation goes on. Systemic corticosteroids, N-acetyl-cysteine, vasodilators, and vitamin E may be used as the treatment options. Currently, no optimal treatment is available; therefore, prevention is of paramount importance.[1,2,5] In the present report, the presentation of nephrotoxicity limited use of corticosteroids or other medical treatment options for hearing loss. Although aminoglycoside-induced hearing loss is considered irreversible, and the effectiveness of HBOT on this indication is controversial, we treated the patient with HBOT, and had a positive hearing outcome. There might be an association between HBOT and hearing recovery in aminoglycoside ototoxicity. This phenomenon may be explained by the antioxidant, neuroregenerative, and cellular proliferative effects of HBOT, as well as early initiation of treatment. To the best of our knowledge, this is the first study that reports HBOT as a successful treatment option in streptomycin-induced ototoxicity. The results of this report emphasize the need for more extensive research on aminoglycoside-induced SNHL, and its treatment with HBOT.

REFERENCES

1. Xie J, Talaska AE, Schacht J. New developments in aminoglycoside therapy and ototoxicity. Hear Res 2011;281(1-2):28-37.
2. Rizzi MD, Hirose K. Aminoglycoside ototoxicity. Curr Opin Otolaryngol Head Neck Surg 2007;15(5):352-7.
3. Roland PS. New developments in our understanding of ototoxicity. Ear Nose Throat J 2004;83(9 Suppl 4):15-6.
4. Rybak LP, Kelly T. Ototoxicity: bioprotective mechanisms. Curr Opin Otolaryngol Head Neck Surg 2003;11(5):328-33.
5. Selimoglu E. Aminoglycoside-induced ototoxicity. Curr Pharm Des 2007;13(1):119-26.
6. Wu WJ, Sha SH, McLaren JD, Kawamoto K, Raphael Y, Schacht J. Aminoglycoside ototoxicity in adult CBA, C57BL and BALB mice and the Sprague-Dawley rat. Hear Res 2001;158(1-2):165-78.
7. Amora Lde A, Murashima Ade A, Rossato M, Moreira MB, Hyppolito MÂ, Fagundes DJ. The effects of hyperbaric oxygen therapy upon ototoxic injuries produced by amikacin in guinea pigs. Braz J Otorhinolaryngol 2013;79(3):342-8.
8. Yassuda CC, Righetti AE, Cury MC, Hyppolito MA, Oliveira JA, Féres O. The role of hyperbaric oxygen therapy (hot) as an otoprotection agent against cisplatin ototoxicity. Acta Cir Bras 2008;23 Suppl 1:72-6; discussion 76.
9. Gaitanou K, Fildissis G, Vavasis P, Kalentzos V, Baltopoulos G. Management of sudden hearing loss with hyperbaric oxygen therapy. Undersea Hyperb Med 2014;41(5):363-70.
10. Gasier HG, Fothergill DM. Oxidative stress, antioxidant defenses and nitric oxide production following hyperoxic exposures. Undersea Hyperb Med 2013;40(2):125-34.
11. Bajrović FF, Sketelj J, Jug M, Gril I, Mekjavić IB. The effect of hyperbaric oxygen treatment on early regeneration of sensory axons after nerve crush in the rat. J Peripher Nerv Syst 2002;7(3):141-8.
12. Bakır S, Özbay M, Kınış V, Yorgancılar E, Gün R, Gül A, et al. The effects of hyperbaric oxygen therapy against streptomycin ototoxicity. J Clin Anal Med 2014;5(2):97-102.