



Case report

Sudden bilateral hearing loss after organophosphate inhalation

Mehmet Akif Dundar ^a, Serhan Derin ^{b,*}, Mitat Aricigil ^a, Mehmet Akif Eryilmaz ^a^a Necmettin Erbakan University, Meram Medical Faculty, Department of Otorhinolaryngology, Turkey^b Mugla Sitki Kocman University, School of Medicine, Department of Otorhinolaryngology, Mugla, Turkey

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ABSTRACT

Sudden bilateral hearing loss are seen rarely and the toxic substance exposure constitutes a small part of etiology. A Fifty-eight-year-old woman admitted to our clinic with sudden bilateral hearing loss shortly after chlorpyrifos-ethyl exposure. Otolaryngologic examination findings were normal. The patient had 40 dB sensorineural hearing loss (SNHL) on the right ear and 48 dB SNHL on the left ear. Additional diagnostic tests were normal. The conventional treatment for sudden hearing loss was performed. On the second week following organophosphate (OP) exposure the patient's hearing loss almost completely resolved. OP's are heavily used in agriculture and should be taken into consideration as an etiologic factor in sudden hearing loss.

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1. Introduction

Chlorpyrifos-ethyl is a broad spectrum OP insecticide. Insecticides kill insects by impairing their nervous system and at the same time have neurotoxic effects in humans.¹ Neurotransmission in neurons is affected by these chemicals which are neurotoxic and this can sometimes be very fatal.^{1,2} Sudden bilateral SNHL quite rare and it constitute less than 5% of entire sudden SNHLs.³ In this article, a rare case of bilateral sudden hearing loss after chlorpyrifos ethyl exposure is presented.

2. Case presentation

A Fifty-eight-year-old woman admitted to our clinic with bilateral hearing loss. It was learned that the patient was heavily exposed to OP derivatives (Chlorpyrifos-ethyl) while gardening the previous day. A few hours after the exposure, tinnitus had occurred first in the left ear followed by the right ear, then bilateral hearing loss had developed. She did not undergo vertigo. Bilateral ear canal, auricles and tympanic membrane were seen to be normal on physical examination and no spontaneous nystagmus was detected. Other

otolaryngologic examination findings were normal. It was learned from the patient's history that the patient had been administered tenofovir for the treatment of chronic hepatitis B and she suffers from asthma and diabetes mellitus. The patient had no history of hearing loss, vertigo or any other otologic disease. The patient had 40 dB SNHL on the right ear and 48 dB SNHL on the left ear on the pure tone audiogram (Fig. 1). Speech discrimination was %76 on right ear, %82 on left ear. Tympanometry was bilateral Type A thus, it was ruled out any middle ear pathology. Blood count and blood biochemistry tests, were normal except for blood sugar level and LDH. Blood sugar levels were detected in the range of 97–150 mg/dl in repeated blood sugar measurements in the hospitalization process and LDH was elevated (300 u/L). On contrast-enhanced MR imaging, both internal auditory canal, cochlea and semicircular canals were normal. This condition was diagnosed as an OP ototoxicity and 1 mg/kg prednisolone intravenously (IV), 2 × 4 g piracetam (IV), 2 × 250 ml dextran 40 (IV) and 1 × 1 vitamin B complex (250 mg vitamin B1, 250 mg vitamin B6, 1 mg vitamin B12) (IV) were started. By reducing the dose of prednisolon every three days the treatment was completed in 15 days. On the second week following OP exposure the patient's hearing loss almost completely resolved (Fig. 2) and no recurrences of hearing loss, tinnitus or vertigo were reported during the one-year follow-up period.

3. Discussion

Bilateral sudden SNHL is a very rare entity. It is known to constitute less than 5 per cent of sudden SNHL cases, and a review of the literature proves this with only a few number of case series.³

* Corresponding author. Mugla Sitki Kocman University, School of Medicine, Department of Otolaryngology, Mugla Sitki Kocman Universitesi Tip Fakultesi, Orhaniye Mah. Haluk Ozsoy Cad, 48000 Mugla, Turkey. Tel.: +90 505 7784530; fax: +90 252 211 1345.

E-mail address: serhanderin@yahoo.com.tr (S. Derin).

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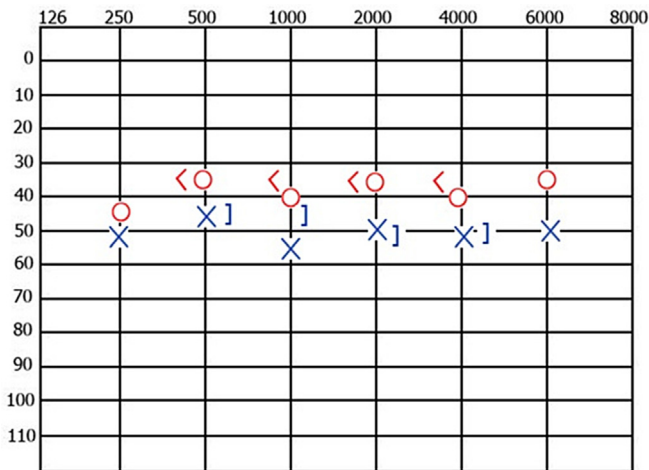


Fig. 1. Audiometric analysis of the patient after chlorpyrifos-ethyl exposure. <= right bone conduction with masking; J= left bone conduction with masking; O= right air conduction x= left air conduction.

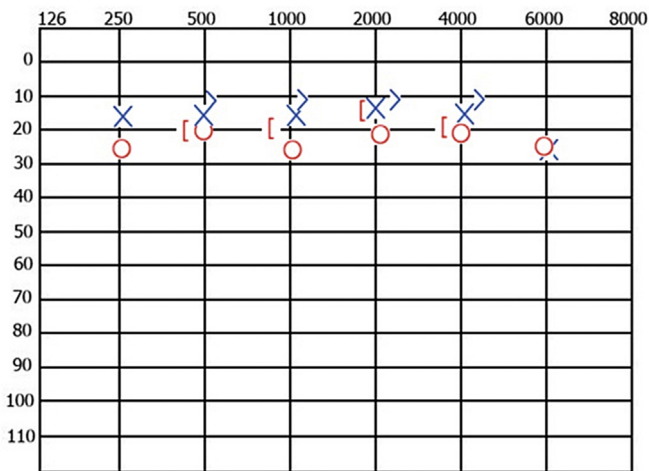


Fig. 2. Audiometric analysis of the patient after treatment (Second week). <=right bone conduction with masking; J= left bone conduction with masking; O= right air conduction x= left air conduction.

Bilateral disease is a medical emergency because it is almost associated with serious illness and results in worse hearing prognosis and results. The most common etiologic factors of bilateral sudden SNHL are toxic substances, neoplasm, and vascular diseases. Autoimmune diseases are also highly pronounced and idiopathic etiology is surprisingly quite low.³ OP insecticides are responsible for a very small part of these etiologic factors.

OP poisoning is seen frequently in the developing world as its use is widespread and its accessibility easy. OP poisoning commonly occurs following ingestion, inhalation, and absorption of OP compounds^{4,5} The most common mechanism of ototoxicity associated with OP poisoning is production of reactive oxygen species (ROS).⁶ OPs also affect acetylcholinesterase, whose function is to hydrolyze acetylcholine that most important neurotransmitter in the central and peripheral nervous systems.^{1,2,5}

Various OPs are neurotoxic and could potentially affect hearing.⁷ However the mechanism behind hearing loss following OP toxicity is not very clear yet. There is a rarity of literature on insecticide exposure and hearing loss in both animals and humans.

Jayasinghe et al stated that there were no significant changes in brain stem auditory-evoked potentials in patients with acute

suicidal exposure of OP.⁶ They believed that even though this condition is associated with the generation of ROS within the perilymphatic space following the ingestion of OP, the auditory pathway is not sufficiently affected to result in lesions.

Crawford et al presented that risk of hearing loss increased in OP exposure. They also showed the existence of a relationship between hearing loss and solvent and metal exposures. Many insecticide formulations include solvents, metals, and other inert ingredients, therefore there is a possibility that these exposures play a role in the associations between hearing loss and pesticide exposure.⁷

Acute poisoning with OP insecticide (malathion) as a potential cause of permanent sudden bilateral SNHL is reported in only one case study.⁸ However chlorpyrifos-ethyl is not yet associated with any hearing loss.

The exact pathogenesis of sudden SNHL associated with toxic agents exposure is not yet clear therefore, medication of the situation is controversial. Systemic steroids, betahistine and cochlear vasodilators are used for medication. In our case, bilateral sudden SNHL developed after the patient inhaled chlorpyrifos-ethyl the previous day and we investigated the etiology of the clinical situation. We didn't find any systemic or local etiologic factor other than chlorpyrifos-ethyl exposure. There were no symptoms associated with OP poisoning and we therefore diagnosed the condition as bilateral sudden SNHL. On treatment 1 mg/kg prednisolone, piracetam, dextran and vitamin B complex were administered. The patient's hearing had returned to normal on the second week following exposure.

High LDH levels may be seen because of OP induced oxidative tissue damage or muscle injury in these cases.⁵ Our patients LDH level was 300 u/L (normal range 125–220 u/L). It decreased to normal levels in the second week.

Acute exposure to OPs may cause adverse effect on central nerve system (CNS), in some cases, in a long time.¹ There were no CNS finding in our patient for one year.

In conclusion OPs are heavily used in agriculture as insecticides and should be taken into consideration as an etiologic factor in sudden SNHL. Whenever there is poisoning from these agents the patient's hearing should be evaluated and especially with the development of bilateral hearing loss early treatment should be started to avoid the occurrence of serious morbidities in the future.

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Conflicts of interest

None declared.

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