Unilateral Hearing Loss Following Carbon Monoxide Poisoning

Peer review status:
No

Corresponding Author:
Dr. Nuno D Costa,
Nuno Ribeiro-Costa, Hospital Pedro Hispano, Rua Dr. Eduardo Torres. Senhora da Hora., 4464-513 - Portugal

Submitting Author:
Dr. Nuno D Costa,
Nuno Ribeiro-Costa, Hospital Pedro Hispano, Rua Dr. Eduardo Torres. Senhora da Hora., 4464-513 - Portugal

Other Authors:
Dr. Diogo Abreu Pereira,
Diogo Abreu Pereira, Hospital Pedro Hispano, Rua Dr. Eduardo Torres. Senhora da Hora., 4464-513 - Portugal
Dr. Inês Gambôa,
Inês Gambôa, Hospital Pedro Hispano, Rua Dr. Eduardo Torres. Senhora da Hora., 4464-513 - Portugal
Dr. Paula Azevedo,
Paula Azevedo, Hospital Pedro Hispano, Rua Dr. Eduardo Torres. Senhora da Hora., 4464-513 - Portugal
Dr. Delfim Duarte,
Delfim Duarte, Hospital Pedro Hispano, Rua Dr. Eduardo Torres. Senhora da Hora., 4464-513 - Portugal

Article ID: WMC005250
Article Type: Case Report
Submitted on: 30-Dec-2016, 03:09:35 PM GMT Published on: 04-Jan-2017, 05:24:33 AM GMT
Article URL: http://www.webmedcentral.com/article_view/5250
Subject Categories: OTORHINOLARYNGOLOGY
Keywords: Hearing Loss, Carbon Monoxide Poisoning, Otorhinolaryngology, Neurotology, Unilateral Hearing Loss, Inner ear

How to cite the article: Costa ND, Abreu Pereira D, Gambôa I, Azevedo P, Duarte D. Unilateral Hearing Loss Following Carbon Monoxide Poisoning. WebmedCentral OTORHINOLARYNGOLOGY 2017;8(1):WMC005250

Copyright: This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC-BY), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Source(s) of Funding:
The authors declared that no grants were involved in supporting this work

Competing Interests:
No Competing interests were disclosed
Unilateral Hearing Loss Following Carbon Monoxide Poisoning

Author(s): Costa ND, Abreu Pereira D, Gambôa I, Azevedo P, Duarte D

Abstract

The ototoxicity associated with carbon monoxide is rare and is usually associated with chronic exposure. The pathophysiological mechanisms include hypoxia due to decreased import of oxygen and free radical damage to the cochlea, auditory nerve, and central nervous system.

We present a case of a 49-year-old man admitted to our emergency department after acute voluntary intoxication by carbon monoxide. Due to psychomotor restlessness, the patient was admitted in intensive care for surveillance and early treatment with hyperbaric oxygen therapy. On the 7th day, sedoanalgesia was suspended. At this point refers decreased hearing acuity of the right ear associated with tinnitus. The audiogram showed a severe right-sided sensorineural hearing loss from 500 to 8000 Hz. Systemic corticosteroid therapy was initiated associated with hyperbaric oxygen therapy. After three months the audiogram shows a partial recovery of hearing loss in his right ear.

Case Report

A 49-year-old man admitted to our emergency department after acute voluntary intoxication by carbon monoxide. He had a medical history of alcoholism, cirrhosis (Class A in Child-Pugh score) and chronic gastritis. He was chronically medicated with propranolol, baclofen and pantoprazole.

At presentation, due to psychomotor restlessness, the patient was admitted in intensive care for surveillance and early treatment with hyperbaric oxygen therapy. On the 7th day, the sedoanalgesia was suspended. There are no records of ototoxic agents administrated to the patient during his 7 days sedation. At this point, the patient complained of hearing impairment of the right ear associated with tinnitus. There was no complaint of dizziness, vertigo or ear pain. The otoscopic examination was normal with the tuning fork test compatible with a right-sided sensorineural hearing loss. There was no nystagmus (spontaneous or induced). There was no evidence of other cranial nerve or focal neurological deficit. The rest of the systemic examination was within the normal limits.

The audiometric examination showed a severe right-sided sensorineural hearing loss from 500 to 8000 Hz (Illustration 1). The speech discrimination score was normal bilaterally. The tympanometry revealed a type A curve bilaterally. MRI examination was performed, which showed evidence of fine areas of hypersignal around the white matter of the basal ganglia (Illustration 2).

The treatment consisted of intravenous corticosteroid therapy with methylprednisolone (1 mg/kg/day) for 10 days. The patient was submitted to hyperbaric oxygen therapy for a total of 30 sessions with one-hour duration each.

The patient reported minimal subjective improvement in hearing loss after pharmacotherapy and hyperbaric oxygen therapy. At 3-months follow-up, the audiogram showed a partial recovery of hearing loss in his right ear (Illustration 3). The patient kept his tinnitus complaints in the right ear, but without significant impact in his daily routines. No other neurologic sequelae were identified.

Discussion

Carbon monoxide poisoning is an uncommon cause of hearing loss, especially after acute poisoning. The mechanism responsible for hearing loss in CO poisoning has not been definitively established but is likely due to hypoxia (which results from the conversion of oxyhemoglobin to carboxyhemoglobin) and free radical damage.[1,3,6] The central auditory pathways, including the auditory cortex and the inferior colliculus are more sensitive to hypoxia than the cochlea[7]. Other brain regions are frequently affected such as basal ganglia (especially globus pallidus), cerebral cortex, hippocampus and substantia nigra.[4,5]

The CO-induced hearing loss is usually bilateral, with variable severity and affecting mostly high frequencies, but further exposure to CO may affect low frequencies as well.[6] Although rare, unilateral hearing loss due to CO poisoning has also been previously reported.[1,8] Dizziness and vestibular impairment have been previously documented in CO poisoning.[9] but such symptoms were denied by our patient.
In our case, the presence of unilateral hearing loss combined with normal speech discrimination suggests a cochlear damage rather than retrocochlear. Therefore, the findings in the brain MRI aren’t probably related to the hearing loss observed in this case. These mismatch between the literature and our case demonstrates that there are other possible unknown mechanisms through which CO induces damage to the cochleae. Further electrophysiological tests could help to understand the underlying damage mechanism.

Though beneficial in the treatment of idiopathic sudden hearing loss, there is no clear evidence of the effectiveness of steroid therapy in deafness associated with CO poisoning. Some reports suggest that the hearing loss recovery occurs spontaneously, without any specific treatment.[1] Since there is no standard treatment for hearing loss and taking into account the physiopathology of cochlear damage in CO poisoning, we implemented corticotherapy and continued the hyperbaric oxygen therapy that is already used in cases of acute CO poisoning and idiopathic sudden hearing loss.

References

Illustrations

Illustration 1

Initial tonal and vocal audiogram

Illustration 2

Brain MRI presents fine areas of hypersignal around the white matter of the basal ganglia
Illustration 3

3rd month follow-up tonal and vocal audiogram