The Journal of International Advanced Otology

J Int Adv Otol 2023; 19(5): 435–439 • DOI: 10.5152/iao.2023.231117

Case Report

Sensorineural Hearing Loss due to Acute Carbon Monoxide Poisoning

Takuya Endo[1], Jun Suzuki[1], Ryoukichi Ikeda[2], Yukio Katori[1]

[1]Department of Otolaryngology-Head and Neck Surgery, Tohoku University Graduate School of Medicine, Sendai, Miyagi, Japan
[2]Department of Otolaryngology-Head and Neck Surgery, Iwate Medical University, Yahaba, Iwate, Japan

ORCID iDs of the authors: T.E. 0009-0008-1600-9233, J.S. 0000-0002-9164-6389, R.I. 0000-0003-0076-6092, Y.K. 0000-0003-1963-5551.

Cite this article as: Endo T, Suzuki J, Ikeda R, Katori Y. Sensorineural hearing loss due to acute carbon monoxide poisoning. *J Int Adv Otol.* 2023;19(5):435-439.

Carbon monoxide (CO) can cause "irreversible" severe-to-profound sensorineural hearing loss. However, there are few reports of detailed hearing test results. Here, we report a case of acute sensorineural hearing loss caused by acute CO poisoning with partial hearing recovery, evaluated by a detailed hearing examination. A 25-year-old woman was brought to the emergency department for attempted suicide. On admission, her consciousness was impaired, and she was treated for severe CO poisoning, including using hyperbaric-oxygen therapy. After regaining consciousness, symptoms of hearing loss and tinnitus were discovered, and a detailed audiological examination revealed bilateral hearing loss, suggesting cochlear damage. Steroids were systemically administered, and her hearing impairment was partially resolved. Sensorineural hearing loss caused by acute CO poisoning includes cochlear pathology and may be partially treatable. The early evaluation of hearing in patients with severe CO poisoning is advisable for early treatment.

KEYWORDS: Carbon monoxide, sensorineural hearing loss, hearing tests, poisoning, treatments

INTRODUCTION

Carbon monoxide (CO) is a colorless, tasteless, and odorless gas that binds to ferrous heme-containing proteins, such as hemoglobin (Hb) with high affinity, reduces oxygen-carrying capacity, and interrupts critical cellular functions.[1] Although several cases of sensorineural hearing loss due to acute or chronic CO poisoning have been reported,[2-8] most reports have shown only the results of pure-tone audiometry (PTA) for audiological examination. Therefore, whether the primary pathology of sensorineural hearing loss due to CO poisoning is a cochlear disorder or a central nervous system disease is still unclear. In this report, we describe a case of sensorineural hearing loss caused by acute CO poisoning, in which we performed detailed audiological examinations and treated the patient with hyperbaric-oxygen treatment (HBOT) and systemic steroids.

CASE PRESENTATION

A 25-year-old woman was referred to our hospital for the management of acute CO poisoning. More than 24 hours before admission, she had attempted suicide by burning charcoal briquettes in her car. A neighbor noticed a suspicious vehicle and called for emergency medical assistance because she was unconscious. Vital signs on admission included a body temperature of 36.3°, blood pressure of 94/67 mmHg, and heart rate of 109/min. Her Glasgow Coma Score was 12: eye (4) verbal, (3); and motor (5). Her laboratory data showed the following: hemoglobin, 15.6 g/dL; white blood cell count, 23 800/mL; platelet count, 426 000 /mL; serum sodium, 137 mmol/L; potassium, 5.3 mmol/L; chloride 104 mmol/L; lactate dehydrogenase, 693 U/L; creatine phosphokinase, 34 005 U/L; blood urea nitrogen, 39.0 mg/dL; creatinine, 0.91 mg/dL; and estimated glomerular filtration rate, 60.0 mL/min/1.73 m^2. Although these data did not meet the criteria of acute kidney injury according to the Kidney Disease Improving Global Outcomes (KDIGO) guidelines, they did suggest the rapid deterioration of renal function and rhabdomyolysis. Arterial blood gas analysis revealed a carboxyhemoglobin level of 19.8%. Brain computed tomography (CT) and magnetic resonance imaging (MRI) revealed an abnormal signal bilaterally in the globus pallidus (Figure 1). The patient was treated for severe CO poisoning with impaired consciousness by the emergency department, including HBOT. After regaining consciousness, she complained of bilateral hearing

Corresponding author: Jun Suzuki, e-mail: j_suzuki1212@orl.med.tohoku.ac.jp

Received: March 02, 2023 • **Accepted:** May 14, 2023 • **Publication Date:** September 29, 2023
Available online at www.advancedotology.org

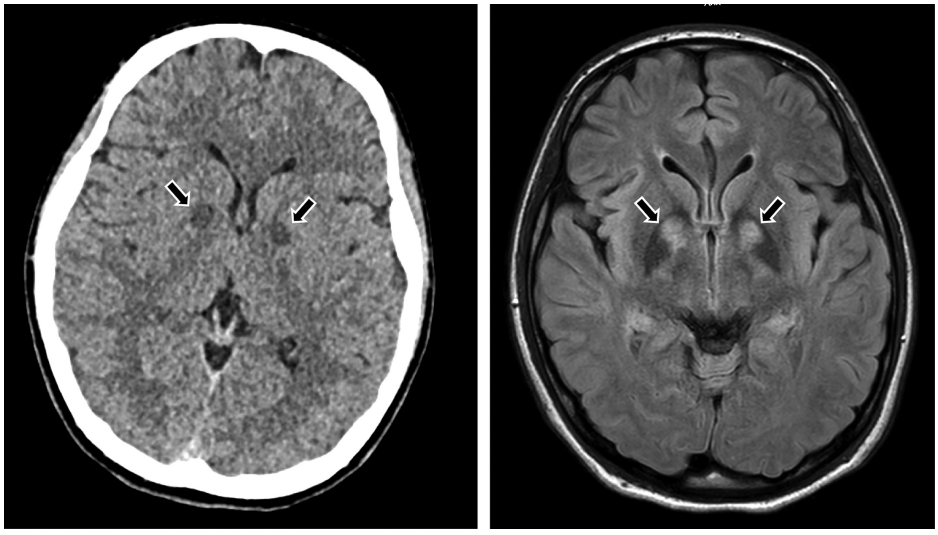

Figure 1. Images at the basal ganglia level. Computed tomography (left) shows a low-density area in the globus pallidus bilaterally (arrows). Fluid-attenuated inversion recovery-weighted magnetic resonance imaging (right) shows bilaterally increased signals in the globus pallidus (arrows).

loss and tinnitus on day 3 of hospitalization. She was referred to our department 7 days after admission after her general condition improved.

Pure-tone audiometry measured by a pure-tone audiometer (AA-H1, RION, Tokyo, Japan) showed mild right hearing loss and moderate left hearing loss (right: 35.0 dB hearing level (HL), left: 41.25 dB HL; arithmetic mean of pure-tone thresholds at 500, 1000, 2000, and 4000 Hz) (Figure 2A). Distortion product otoacoustic emissions (DPOAEs) were measured using an OAE Screener (ER-60, RION) in both the ears, which revealed reduced/absent emissions at 1.5, 2.0, and 3.0 kHz in the right ear and at 1.5, 2.0, 3.0, and 4.0 kHz in the left ear (Figure 3). Speech discrimination testing was performed using a pure-tone audiometer (AA-H1) with a standardized protocol, the Japanese Monosyllable Word List 67, which uses 20 monosyllables in a semi-closed form. The word discrimination score for the normal 20 monosyllables in this test generally reaches almost 100% in the range of 30- and 40-dB HLs. Our patient's maximum discrimination scores were 85% and 95% in the right and left ears, respectively, with a rollover phenomenon (Figure 4). Self-recording audiometry measured by a pure-tone audiometer (AA-H1) showed a type II pattern (Jeger classification) in both the ears (Figure 5). The short increment sensitivity index test results were 95% at 1000 Hz in the right ear and 75% at 1000 Hz in the left ear (normal < 65%; Figure 6). These results of detailed audiological examinations suggested the presence of loudness recruitment which reflected a cochlear disorder. There were no findings suggesting vestibular dysfunction such as vertigo or instability in the patient.

Under the diagnosis of acute sensorineural hearing loss due to CO poisoning, she was administered 60 mg/day of oral prednisolone for 8 days, with the dose tapering every 2 days thereafter. PTA showed an apparent threshold recovery in both the ears (right: 22.5 dB HL, left: 32.5 dB HL) on hospital day 15 (Figure 2B). Auditory brainstem responses were recorded on hospital day 14, and the threshold was 35 dB HL without extension of latency in both the ears. She was transferred to a long-term care hospital 18 days after hospitalization because her general condition improved. However, she gradually developed delayed neurological sequelae (DNS) approximately 1 month after discharge from the hospital, which made it difficult for her to follow-up at our hospital; thus, her follow-up was terminated. Written informed consent was obtained from the patient.

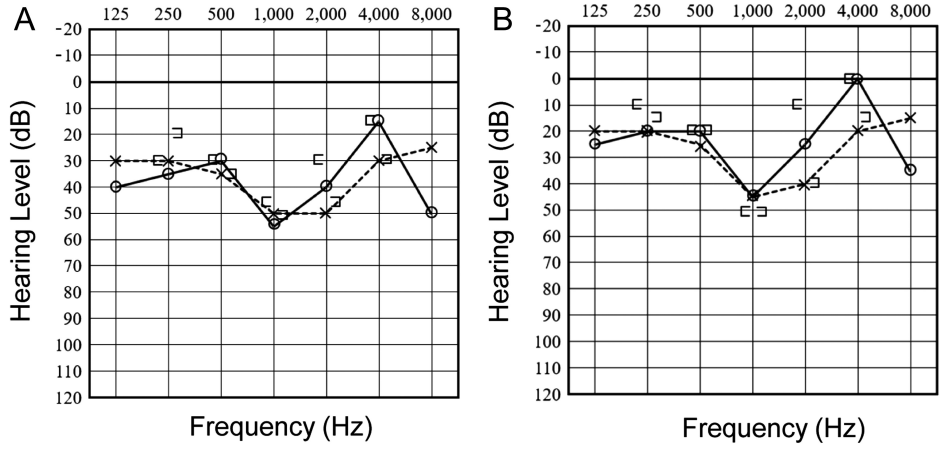

Figure 2. (A) Upon the first presentation, the pure-tone audiometry shows mild-to-moderate bilateral hearing loss. (B) The pure-tone audiometry performed 7 days after steroid treatment shows partial hearing improvement in both the ears.

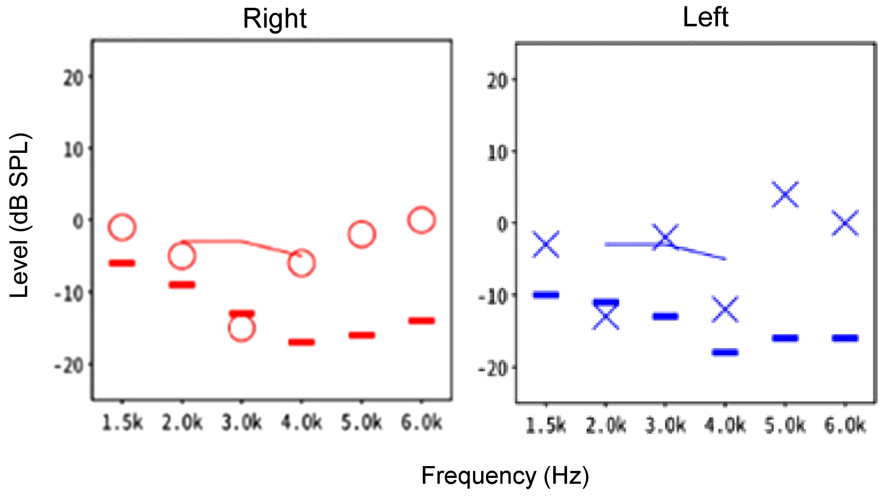

Figure 3. Distortion product otoacoustic emissions reveal reduced/absent emissions in middle frequencies. Circles: distortion product amplitude of right ear. Crosses: distortion product amplitude of left ear. Horizontal lines: noise floor.

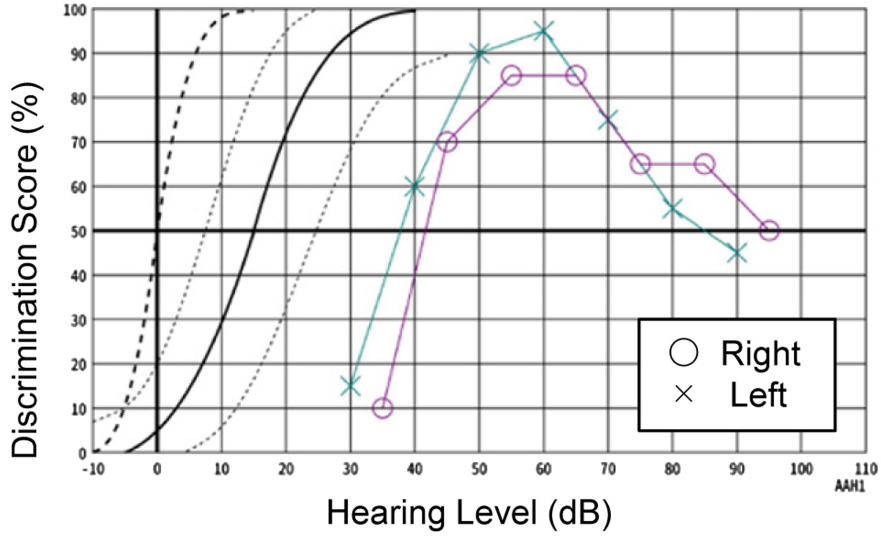

Figure 4. Speech discrimination testing shows deterioration of speech intelligibility in both the ears.

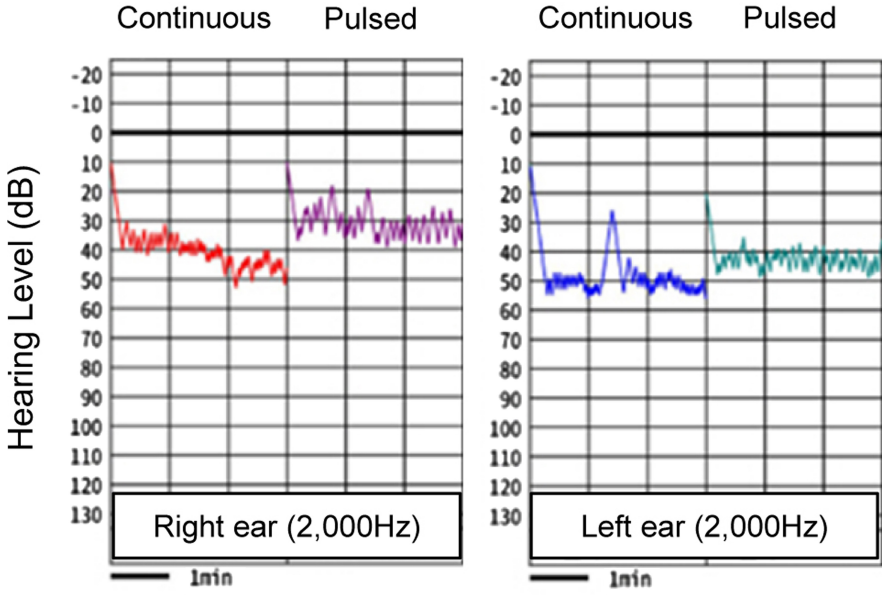

Figure 5. The self-recording audiometry (fixed-frequency tracing) shows a Jeger type II abnormality.

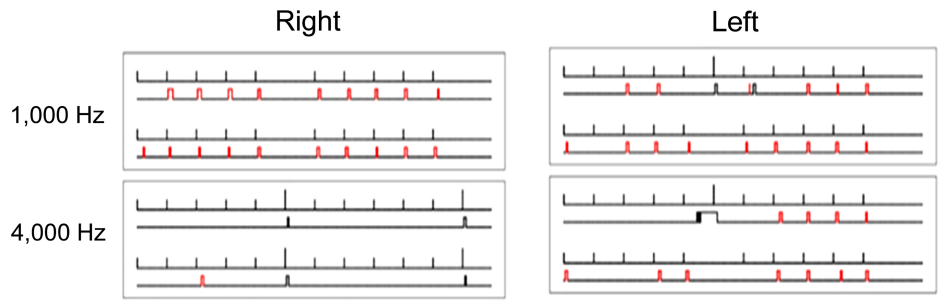

Figure 6. The short increment sensitivity index test suggests the presence of loudness recruitment. Right ear (1000 Hz); level: 75 dB, increment: 1.0 dB, score: 95% (19/20). Right ear (4000 Hz); level: 35 dB, increment: 1.0 dB, score: 5% (1/20). Left ear (1000 Hz); level: 70 dB, increment: 1.0 dB, score: 75% (15/20). Left ear (4000 Hz); level: 50 dB, increment: 1.0 dB, score: 55% (11/20).

DISCUSSION

There are various reports on the pathology of sensorineural hearing loss due to CO poisoning, such as cochlear or central hearing loss. However, there is no consensus on the primary pathological lesion for hearing loss.[7,8] Postmortem examination of victims of acute CO poisoning has shown hemorrhage and cellular degeneration in the cochlear and vestibular nuclei, spiral ganglion, and in various parts of the cochlea itself.[9] From an animal experiment, Fechter et al[10] suggested that the primary target of CO damage is the inner hair cells and/or type I spiral ganglion cells, with the outer hair cells being less affected. Regarding the audiograms, although hypoxia to the cochlea associated with CO poisoning may cause mid-tone damage similar to our case, resulting in a "U-shaped curve" in pure-tone audiograms,[7] there have been several reports of a high-frequency threshold deterioration in CO-induced hearing loss.[2] These results suggest that the audiograms of CO-induced hearing loss vary and are difficult to use in detecting the pathological location of sensorineural hearing loss due to CO poisoning. Note that Makisima et al. reported that the central pathways, such as the auditory cortex and inferior colliculus are the primary site of lesion with damage in CO intoxication.[11] Therefore, the primary pathology of sensorineural hearing loss due to CO poisoning has not been clear for a long time.

From the results of the DPOAE in this study, we suspect that the main lesion of hearing loss was in the cochlea, especially the outer hair cells. Although the rollover phenomenon in speech audiometry implies the possibility of a mixture of retrocochlear pathologies, the results of other audiological examinations also support cochlear hearing loss. The patient's hearing threshold improved partially after steroid treatment. These results suggest that sensorineural hearing loss caused by CO poisoning may be reversible, at least in part, and is consistent with previous reports.[6,8] In other reports, the timing of treatment initiation varied due to differences in pathogenesis, general condition, and other factors. Further studies are required to confirm the results. Collectively, the cochlear pathology of hearing loss due to CO poisoning was suggested in our patient. However, the detailed mechanism of cochlear damage remains unclear and further studies are required.

Severe CO poisoning is often accompanied by impaired consciousness in the acute phase,[12] making it difficult to detect hearing loss and tinnitus symptoms and perform subjective tests, such as PTA. Considering the simplicity of the examination, the DPOAE test, an objective test with a short examination time, may be helpful for screening hearing loss associated with CO poisoning. Further research on the effect of CO intoxication on outer hair cells is also necessary to examine the appropriateness of evaluating CO poisoning–induced hearing loss using the DPOAE test.

Oxygen supplementation is key for treating CO intoxication. In particular, HBOT has been proven to be effective for acute CO poisoning and reducing the risk of DNS compared to normobaric-oxygen treatment.[12] Furthermore, in cases where administration of HBOT is limited owing to unstable vital signs and intubation, or absence of an available HBOT, induced hypothermia has been suggested to be effective for severe CO poisoning.[13] However, there is no established treatment for the hearing loss itself that is caused by CO poisoning.

Although controversial, steroids are supposed to be beneficial and widely used to treat idiopathic sudden sensorineural hearing loss.[14] However, there is no clear evidence of its efficacy in sensorineural hearing loss associated with CO poisoning. The anti-inflammatory and anti-edema effects of steroids may contribute to recovery from neurological dysfunction caused by CO poisoning,[15] suggesting that steroids are useful for the treatment of CO poisoning itself. The partial hearing recovery in our patient supports the use of steroids for sensorineural hearing loss due to CO poisoning, but the effectiveness of steroids should be researched further.

Acute CO poisoning can cause sensorineural hearing loss owing to a cochlear pathology. CO poisoning–induced hearing loss may be partially treatable, and oral steroid therapy may effectively treat this disease. For early treatment, evaluation of hearing in patients with severe CO poisoning is advisable.

Informed Consent: Written informed consent was obtained from the patient who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – J.S.; Design – T.E., J.S.; Supervision – Y.K.; Resources – N/A; Materials – N/A; Data Collectiong – E.T., J.S., R.I.; Analysis and Interpretation – T.E., J.S., R.I.; Literature Search – T.E., J.S.; Writing – T.E., J.S.; Critical Review – R.I., Y.K.

Declaration of Interests: The authors have no conflict of interest to declare.

Funding: The authors declared that this study has received no financial support.

REFERENCES

1. Rose JJ, Wang L, Xu Q, et al. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. *Am J Respir Crit Care Med*. 2017;195(5):596-606. [CrossRef]
2. Li C-Z, Li C-C, Lin M-C, Tseng W-S. Sudden hearing loss following acute carbon monoxide poisoning: a case report and literature review. *HK J Emerg Med*. 2023;30(1_suppl):9S-13S. [CrossRef]
3. Mehrparvar AH, Davari MH, Mollasadeghi A, et al. Hearing loss due to carbon monoxide poisoning. *Case Rep Otolaryngol*. 2013;2013:940187. [CrossRef]
4. Morris TMO. Deafness following acute carbon monoxide poisoning. *J Laryngol Otol*. 1969;83(12):1219-1225. [CrossRef]
5. Pillion JP. Sensorineural HEARing Loss following carbon monoxide Poisoning. *Case Rep Pediatr*. 2012;2012:231230. [CrossRef]
6. Shahbaz Hassan M, Ray J, Wilson F. Carbon monoxide poisoning and sensorineural hearing loss. *J Laryngol Otol*. 2003;117(2):134-137. [CrossRef]
7. Baker SR, Lilly DJ. Hearing loss from acute carbon monoxide intoxication. *Ann Otol Rhinol Laryngol*. 1977;86(3 Pt 1):323-328. [CrossRef]
8. Razzaq M, Dumbala S, Moudgil SS. Neurological picture. Sudden deafness due to carbon monoxide poisoning. *J Neurol Neurosurg Psychiatry*. 2010;81(6):658. [CrossRef]
9. Schullenberg W. Uber die Schadigungen des Innenohres durch akute Kohlenoxydgasvergiftung [Injuries of inner ear caused by acute carbon monoxide poisoning]. *Z Laryngol Rhinol Otol*. 1953;32(6):339-353.
10. Liu Y, Fechter LD. MK-801 protects against carbon monoxide-induced hearing loss. *Toxicol Appl Pharmacol*. 1995;132(2):196-202. [CrossRef]
11. Makishima K, Keane WM, Vernose GV, Snow JB Jr. Hearing loss of a central type secondary to carbon monoxide poisoning. *Trans Sect Otolaryngol Am Acad Ophthalmol Otolaryngol*. 1977;84(2):452-457.
12. Weaver LK, Hopkins RO, Chan KJ, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. *N Engl J Med*. 2002;347(14):1057-1067. [CrossRef]
13. Oh BJ, Im YG, Park E, Min YG, Choi SC. Treatment of acute carbon monoxide poisoning with induced hypothermia. *Clin Exp Emerg Med*. 2016;3(2):100-104. [CrossRef]
14. Chandrasekhar SS, Tsai Do BS, Schwartz SR, et al. Clinical practice guideline: sudden hearing loss (update). *Otolaryngol Head Neck Surg*. 2019;161(1_suppl):S1-S45. [CrossRef]
15. Iwamoto K, Ikeda K, Mizumura S, Tachiki K, Yanagihashi M, Iwasaki Y. Combined treatment of methylprednisolone pulse and memantine hydrochloride prompts recovery from neurological dysfunction and cerebral hypoperfusion in carbon monoxide poisoning: a case report. *J Stroke Cerebrovasc Dis*. 2014;23(3):592-595. [CrossRef]