

Early Resolution Of Bilateral Sudden Sensorineural Hearing Loss Due To Acute Carbon Monoxide Intoxication With Normobaric Oxygen, Systemic Steroid, Dextran And Piracetam Treatment

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Citation

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Abstract

Exposure to carbon monoxide (CO) is a well-recognized cause of morbidity and mortality. Acute accidental poisoning is associated with a range of adverse health related effects. CO affects multiple organ systems but it cause permanent damage especially in neurologic and cardiovascular systems. Association of acute CO poisoning with sudden sensorineural hearing loss (SSHL) is a rare entity. In this paper, we present a case of bilateral SSHL due to acute CO intoxication in a 25-year-old female patient who refused to have hyperbaric oxygen treatment. A favourable result was achieved with normobaric oxygen, systemic steroid, dextran and piracetam treatments.

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INTRODUCTION

CO is a common indoor pollutant and an important cause of adverse health effects. These effects consist a wide spectrum from minor symptoms to death⁽¹⁾. CO is a colourless, odourless, tasteless gas and is produced by the incomplete combustion of carbon-containing materials⁽²⁾. CO poisoning is a frequently seen form of poisoning, with common mechanisms including faulty heating appliances of deliberate self harm and attempted suicide⁽³⁾. In survivors of acute poisoning CO intoxication, it may also be associated with disabling neurological symptoms. It is a known but unusual cause of SSHL⁽⁴⁾.

The symptoms of CO poisoning are thought to be due to tissue hypoxia. CO has a 240 times greater affinity for haemoglobin than oxygen and so displaces oxygen from erythrocytes⁽⁵⁾. The standard treatment for CO poisoning is oxygen to reverse hypoxia, compete with CO for haemoglobin binding, and promote carboxyhaemoglobin dissociation. Effects are increased at high pressure, shortening carboxyhaemoglobin half-life⁽⁴⁾.

In this paper we report a case of bilateral SSHL due to acute

CO intoxication who refused to have hyperbaric oxygen treatment and a favourable result was achieved with systemic steroid and piracetam treatment in adjunctive to normobaric oxygen.

CASE REPORT

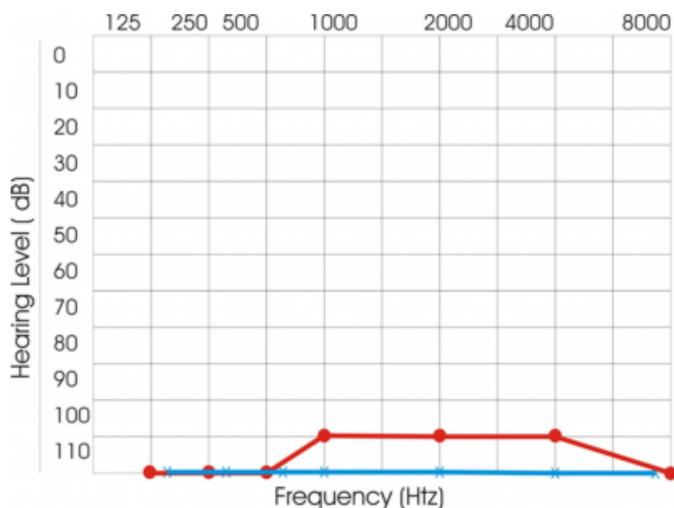
A 25-year-old female patient with no history of previous neurological or other systemic disease was brought unconscious by her family to emergency room (ER) of our hospital because of poisoning from stove while sleeping. At this accident her elder sister who was sleeping at the same room was died and her new born baby was cured in neonatal unit at reanimation service. On presentation at hospital, she was deeply comatose with no clinical signs of cyanosis. Arterial blood gas analysis showed 26.7% carboxyhaemoglobin, 62.9% oxyhaemoglobin and 0.4% methaemoglobin. She was treated with continuous 100% oxygen by mask. Her arterial carboxyhaemoglobin content reduced to 6.6% after four hours, and 1.8% after seven hours, while her oxyhaemoglobin content increased to 97%. Eight hours following admission, she regained consciousness but was suffering with amnesia, blurred vision, deafness and tinnitus. Her vision rapidly returned to normal. The patient was consulted to our clinic as she told that she had bilateral sudden hearing loss and tinnitus. The

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patient had given birth 4 days before this event and after parturition her hearing had been normal. We learned that she had nor troubled pregnancy or hard parturition. On examination after acceptance to our clinic, she was awake, alert and oriented to person, place, time and situation. She followed commands easily without hesitation. Her speech was clear with no disartria. The neurological routine examination including cranial nerve function, motor system examination, reflexes, sensory and cerebellar evaluations were within normal limits. On inspection of her gait; her posture was normal but she had difficulty in walking due to nausea and vertigo. Examination of her ears was normal as were the peak patterns of the tympanograms. However, a pure tone audiogram showed a bilateral total sensorineural hearing lost (Figure 1). Remaining otorhinolaryngological examinations and laboratory tests were completely normal. Hyperbaric oxygen (HBO) treatment had planned but rejected by the patient.

Figure 1

Figure 1: Audiogram on the first day following admission for acute CO poisoning showing bilateral severe SSHL.



Oxygen inhalation at atmospheric pressure was continued intermittently for two more days. Systemic steroid treatment was started combined with dextran and piracetam. The treatment regimen was explained in details in table 1. In control odigrams of the day after first dosage of systemic steroids applied to the patient, air-bone thresholds of right ear was 57 dB, and left ear was 50 dB (figure 2). Hearing gain at the end of therapy was 66 dB for right ear and 60 dB for left ear (Figure 3).

Figure 2

Figure 2: Pure tone audiogram showing hearing thresholds after the first day treatment began.

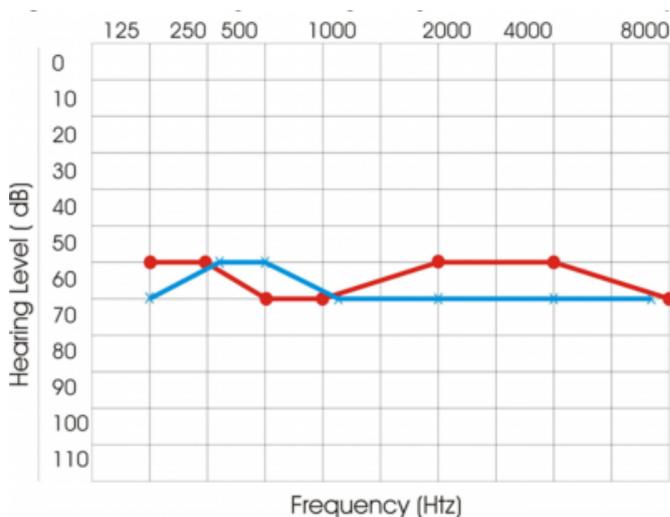


Figure 3

Figure 3: Pure tone audiogram showing hearing thresholds after the eleventh day treatment began.

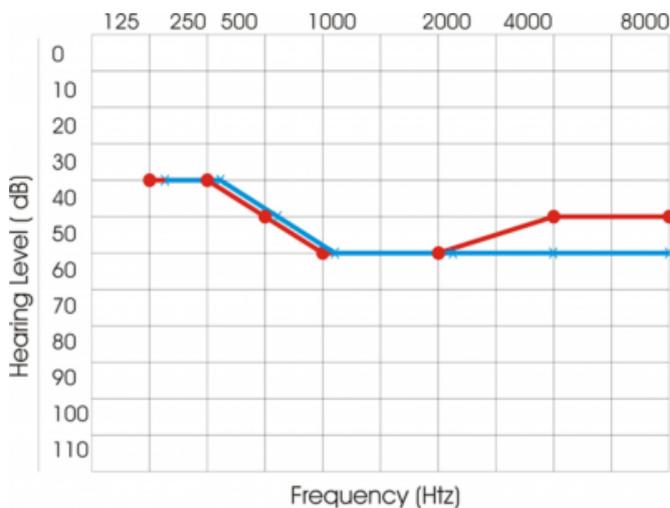


Figure 4

Table 1: Dose schedule of the treatment regimen adjunctive to normobaric oxygen

Medication	Days	Dose scheme
1. Prednisone	First 3 days	1st day: 4 mg/kg IV infusion
		2nd day: 2 mg/kg IV infusion
3rd day: 1 mg/kg IV infusion		
	4th day and so on	Started orally with 1 mg/kg/day divided into 3 doses with a reduction of 20 mg every 3 days
2. Dextran	First 5 days	5 cc/kg/day IV infusion
3. Piracetam	First 5 days	2 mg IV infusion every 8 h
	6-21 days	1.6 mg given orally every 8 h

DISCUSSION

CO poisoning is known to cause a sensorineural hearing loss although it has been a rare aetiology. Other symptoms include headache, nausea, lethargy, pulmonary oedema, arrhythmias, neuropsychiatric problems. The sensorineural hearing loss may be associated with other otological symptoms of tinnitus, nystagmus and ataxia. It may be symmetrical or asymmetrical. In acute poisoning there is typically a U shaped audiogram and the hearing may improve over time (4). SSHL with tinnitus, nausea and vertigo was observed in our patient. Our case is unique with total bilateral hearing loss without any other neurological and circulatory sequel.

Reports about sudden hearing loss in association with acute CO poisoning have been limited in the literature. Furthermore these reports are about mild to moderate hearing loss (1). Actually our case is the first one being total and bilateral SSHL.

The mechanisms by which CO causes cellular damage are complex and multifactorial. Although the neuropathologic changes associated with CO poisoning are related to CO-induced hypoxia since CO binds to haemoglobin, other biochemical mechanisms appear to be involved in cellular damage. These mechanisms include binding of CO to various intracellular proteins, neuro-excitotoxicity, ischemia/reperfusion injury and apoptosis(7). These facts

may explain the possible adjunctive curative effect of systemic steroid, dextran and piracetam in our case.

The auditory dysfunction produced by CO appears to be frequency specific and although the mechanism of this local cochlear effect remains unclear, one possibility is that the basal (high-frequency) region of the cochlea is selectively vulnerable to the effects of CO(1,6). The auditory cortex, inferior colliculus and cochlea are all sensitive to oxygen deprivation (4). Bilateral total hearing loss in our patient may be due to multi-level neurological adverse effects of CO.

Treatment of CO intoxication involves removing the patient from the source CO and administering high partial pressures of oxygen. Practically this involves administering 100% oxygen via a tight-fitting face mask. However, higher partial pressures of oxygen can only be achieved by using oxygen at hyperbaric pressures. Higher partial pressure of oxygen shortens the elimination half-life of carboxyhaemoglobin and increases dissolved oxygen in the blood stream. Hyperbaric oxygen may also reverse the effects of lipid peroxidation and so decrease delayed neurological damage (5). Hyperbaric oxygen is now generally accepted as the treatment of choice in cases of severe CO poisoning.

In our case we planned an immediate HBO treatment but it was rejected by the patient. Favourable hearing results with systemic steroid, dextran and piracetam treatment in adjunctive to normobaric oxygen may encourage us to apply this alternative treatment whenever HBO is not achievable and adjunctive to HBO in severe sensorineural hearing losses due to CO poisoning.

CONCLUSION

Although, the accepted treatment for SSHL due to CO poisoning is 100% oxygen delivered at hyperbaric pressure immediate systemic steroid, dextran and piracetam treatment may be used when HBO treatment is impossible. It may also be used as an adjunctive to HBO especially in severe SSHL.

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