Case Report

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Carbon Monoxide-Induced Cortical Visual Loss: Treatment with Hyperbaric Oxygen Four Years Later

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Key Words

Hyperbaric oxygenation · Delayed neurological sequelae · Visual loss · Carbon monoxide poisoning · Positron emission tomography

Abstract

Objective: We present a patient who developed visual loss after carbon monoxide (CO) poisoning and was treated with hyperbaric oxygen. ***Clinical Presentation and Intervention:*** A 21-year-old woman poisoned with CO (with coma lasting 4 h and carboxyhemoglobin level 46%) developed seizures and cortical blindness 3 days after poisoning. Four years later, her visual acuity was 0.2 in both eyes. An ^{18}F-fluorodeoxyglucose positron emission tomography (PET) scan showed reduced metabolism in the bilateral posterior temporal and occipital lobes. The patient received a total of 50 hyperbaric oxygen sessions over 3 months for visual loss and the visual acuity improved to 0.5 in both eyes. In addition, increased metabolism was detected in the brain in post-treatment PET scans. ***Conclusion:*** PET documented brain hypoperfusion 4 years after CO poisoning and hyperbaric oxygen therapy improved visual acuity. However, we cannot endorse routine use of hyperbaric oxygen for such patients, until results of further clinical trials demonstrate efficacy of hyperbaric oxygen in CO-induced chronic brain injury.

Introduction

Carbon monoxide (CO) poisoning is a main cause of mortality. After a variable period of apparent recovery, delayed neuropsychological symptoms (DNS) may develop [1]. The incidence of DNS has been estimated to range from 1 to 47% [1]. There is no specific therapy for DNS, although a number of beneficial treatments have been reported that include hyperbaric oxygen (HBO_2) therapy [2].

HBO_2 therapy is recommended for the treatment of acute CO poisoning ideally commencing within the first 6 h [3]. HBO_2 reduces 6-week cognitive sequelae following acute CO poisoning as well as prevents DNS [4, 5]. HBO_2 therapy has also been used in the treatment of DNS [2, 6, 7]. In this report, we describe a case with cortical visual loss due to CO poisoning that was successfully treated with HBO_2 therapy 4 years after poisoning.

Case Report

A 21-year-old woman with no history of previous disease was found unconscious 25 min after entering the bathroom shower. There was a water heater operating with liquid petroleum gas in the bathroom. She was immediately taken to the nearest hospital. Her Glasgow Coma Score was 8 on arrival at the Department of Emergency. Her CO hemoglobin level was 46%, pH 7.2, pCO_2

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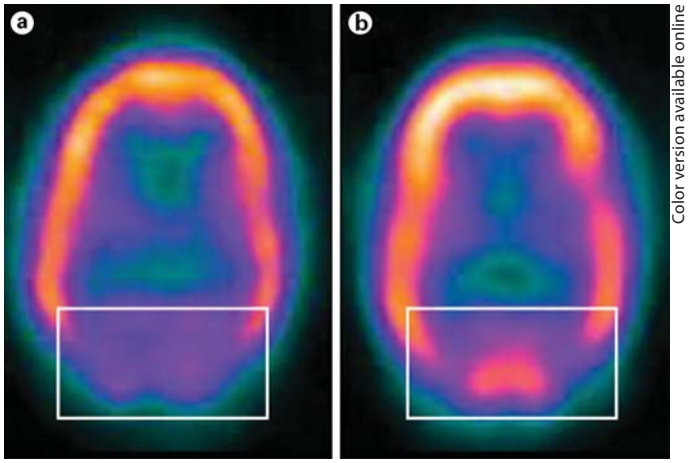

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Fig. 1. PET scans of the patient before HBO_2 therapy (**a**) and after 50 HBO_2 sessions (**b**). For these PET images, ^{18}F-fluorodeoxyglucose was used to demonstrate brain metabolism of glucose. Brain areas of high radioactivity are associated with brain activity. The brightest areas indicate high radioactivity.

46 mm Hg and pO_2 36 mm Hg. The patient was treated with pure oxygen via a nasal mask (10–12 l/min) for approximately 4 h and with 1,000 ml of isotonic sodium chloride solution. The patient regained consciousness after 4 h. She was kept in the hospital for 24 h for observation. Her physical examination, laboratory tests and cranial computerized tomography were normal at the time of discharge.

Three days later, the patient suffered a tonic-clonic (grand mal) epileptic seizure and complained of blurring of vision. In a resting and activation electroencephalography examination, frequent spine-wave-like paroxysms originating from the right hemisphere were observed. The patient received valproic acid (1,000 mg/day p.o.) for 4 years, during which period she suffered no epileptic seizures.

Four years later, the patient again suffered an epileptic seizure and was admitted to the Department of Neurology. The medical treatment for epilepsy was changed to valproic acid (1,000 mg/day p.o.), levetiracetam (500 mg/day p.o.), and piracetam (7,200 mg/day p.o.). Ophthalmological examinations revealed that adnexa and refractive media (i.e. the lens and cornea), and ocular eye movements in all directions were normal. Visual acuity was 0.2 in both eyes according to the Snellen chart. The visual fields in both eyes were normal. Brain magnetic resonance imaging (1.5 Tesla, Magnetom Vision, Siemens, Germany) revealed prominent bilateral cortical hemispheric sulci. Coincidence ^{18}F-fluorodeoxyglucose positron emission tomography (PET) assessment of the patient showed decreased metabolism in the bilateral occipital and posterior temporal lobes (fig. 1a).

Since HBO_2 had been shown to be beneficial in CO-induced visual loss and brain injury in previous reports [6, 7], the patient was referred to the Department of Hyperbaric Medicine. After the epileptic seizures had been brought under control with antiepileptic treatment, the patient was accepted for HBO_2. The sessions were carried out in a multiplace hyperbaric chamber at 2.4 absolute atmospheres for 120 min daily. After the initial 20 sessions, her visual acuity improved, so additional HBO_2 sessions were planned. The patient received a total of 50 HBO_2 treatments in 3 months, rendered daily, Monday through Friday. No epileptic seizure or other complications of HBO_2 were observed during HBO_2 sessions.

After the cessation of HBO_2, visual acuity was 0.5 in both eyes using the Snellen chart. She was not able to read prior to HBO_2 therapy, but with the improvement in visual acuity she began to read again. In addition, PET scanning showed increased metabolism in the bilateral occipital and posterior temporal lobes (fig. 1b).

Discussion

The effects of CO poisoning are not confined to the period immediately after exposure, as shown by the occurrence of DNS [1]. DNS usually appear after a latency period of 2–40 days [1]. Our patient presented with epilepsy and visual loss as a late consequence of CO poisoning 3 days later and she was not able to read. Anterior visual pathways were normal by ophthalmologic examination. Her parents were told that her visual complaints were a component of epilepsy, hence they did not seek any special treatment for her visual loss except magnetic resonance imaging which did not reveal any lesion that might have accounted for the patient's loss of vision. However, PET imaging showed hypometabolism in the posterotemporal and occipital lobes which could account for cortical visual loss.

Our patient's visual acuity improved three lines on the Snellen eye chart (from 0.2 to 0.5) after 50 HBO_2 sessions, in both eyes. In addition, PET scans showed increased metabolism after HBO_2 treatment.

Hon et al. [8] reported visual loss related to CO poisoning in 3 adolescent girls with spontaneous recovery after a few days and near-normal levels after 3 weeks in all 3 patients. Similar to our case, the visual symptoms developed after a latent period between days 5 and 7 after exposure, but did not recover spontaneously in our case. However, our patient showed significant improvement after the initiation of HBO_2 therapy most probably due to the HBO_2 therapy.

In case of acute CO poisoning, HBO_2 therapy improves outcomes by several mechanisms including acceleration of CO elimination from hemoglobin and other heme-containing molecules, improved mitochondrial oxidative metabolism, inhibition of lipid peroxidation, inhibition of leukocyte adherence to injured microvessels and attenuation of immune-mediated delayed neurological dysfunction [3, 9]. However, none of those beneficial

effects may have improved the vision of our patient. In her case, it is likely that HBO_2 led to improvement in visual acuity through mechanisms that are different from the acute period of CO poisoning. After an ischemic and inflammatory insult to the brain, some neurons are damaged irreversibly and these cells proceed to necrosis. In addition, CO can induce premature apoptosis [10]. Nearby cells, which are less affected by hypoxia and inflammation, so-called 'idling neurons', form the ischemic penumbra [11]. The idling neurons are still viable but are dysfunctional, potentially unable to generate action potentials [11]. Neubauer et al. [12] suggested that idling neurons can be reactivated by supplying sufficient oxygen with HBO_2 therapy. They reported that HBO_2 therapy improves both clinical function and brain perfusion documented by single photon emission computed tomography scans in a patient with chronic brain injury even though HBO_2 therapy was initiated 12 years after the incident [12]. If HBO_2 helps recover from late brain injury, other additional mechanisms, which have yet to be elucidated, may be operative.

Conclusion

PET documented brain hypoperfusion 4 years after CO poisoning and HBO_2 therapy improved visual acuity. However, we cannot endorse routine use of HBO_2 for these patients, until results of further clinical trials demonstrate efficacy of HBO_2 in CO-induced chronic brain injury.

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