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

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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 (HBO2) therapy [2]. HBO2 therapy is recommended for the treatment of acute CO poisoning ideally commencing within the first 6 h [3]. HBO2 reduces 6-week cognitive sequelae following acute CO poisoning as well as prevents DNS [4, 5]. HBO2 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 HBO2 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, pCO2...
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 epileptic 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 HBO2 sessions, in both eyes. In addition, PET scans showed increased metabolism after HBO2 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 HBO2 therapy most probably due to the HBO2 therapy.

In case of acute CO poisoning, HBO2 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 neurologic dysfunction [3, 9]. However, none of those beneficial
effects may have improved the vision of our patient. In her case, it is likely that HBO₂ 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 HBO₂ therapy. They reported that HBO₂ 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 HBO₂ therapy was initiated 12 years after the incident [12]. If HBO₂ 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 HBO₂ therapy improved visual acuity. However, we cannot endorse routine use of HBO₂ for these patients, until results of further clinical trials demonstrate efficacy of HBO₂ in CO-induced chronic brain injury.

References